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A CONVENIENT TYPE OF ELECTRO-EJACULATOR FOR BULLS AND RAMS

S. W. J. van RENSBURG and W. H. de VOS

Onderstepoort

The practical application of the many types of electro-ejaculators which have been described within recent years has to a large extent been confined to use in laboratories and in areas in which electric current is available.

The rapidly increasing demand for the examination of semen of both bulls and rams in the field, and the difficulty frequently experienced under such conditions in the collection of semen from animals not trained to serve into an artificial vagina emphasise the need for an ejaculator which is not dependent on electrical current or batteries, and which can easily be transported as part of the equipment normally carried by a practising veterinarian.

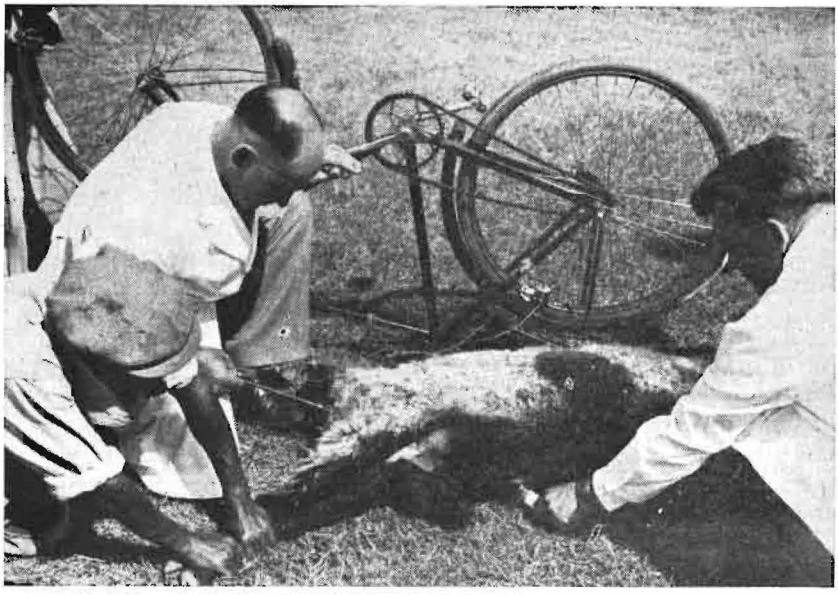

Fig. 1
Collecting semen from a ram. The generator is fixed in its normal position on the back wheel of a bicycle.

In an effort to devise a suitable ejaculator for a valuable bull on a bushveld ranch, that had been incapacitated for natural service by a broken penis, one of us (W.H.d.V.) conceived the idea of obtaining current from the small 6 volt generator which is ordinarily fixed to the hind wheel of a bicycle for supplying light. One of the two leads was attached to the generator, and

the other was earthed on any part of the metal frame of the cycle (Fig. 1).

The electrodes for the bull consist of two copper rings, as described by Rowson and Murdoch (1954) and shown on the right hand of the operator in Figure 2.

For the ram the ends of the two leads are attached one to a copper cap at the anterior end of a 10 inch ebonite tube of ¼ inch diameter, and the other to the inside of a copper ring embedded in the wall of the ebonite tube about an inch behind the cap (Figs. 2 and 3).

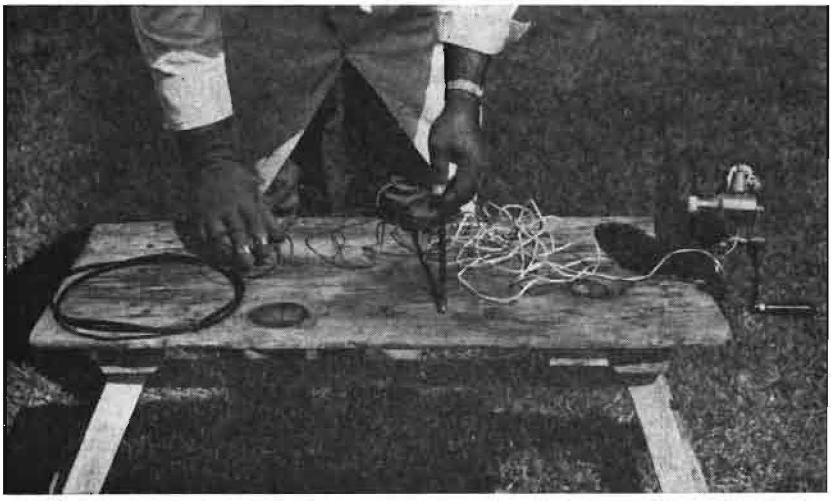

Fig. 2
The generator is applied to a rubber-tyred wheel. The two copper ring electrodes for the bull are shown on the operator's right hand, while that for the ram is held in his left hand.

For the collection of semen from the bull the two copper rings on the gloved fingers are held down on the floor of the rectum directly over the ampullae, while in the ram the ebonite tube with the electrodes is passed into the rectrum for about half its length (i.e. 5 inches) and is also pressed down over the vesiculae seminales and the ampullae.

Current is supplied intermittently by turning the wheel of the cycle very slowly for about 3 seconds and stopping for a second or two. This is repeated until the animal ejaculates.

As with other methods of electro-ejaculation in the bull the first secretion is watery and is derived mainly from the accessory glands. This can be discarded. It is generally followed immediately by normal semen. The ram on the other hand very often yields the full ejaculate straight away.

Tests with the bicycle having proved very successful the generator was next fixed to a small rubber tyred wheel of 6 inches diameter (Figs. 2 and 3) and this yielded equally good results.

Fig. 3
Method of using the ejaculator for the collection of semen from the ram.

The use of this method on a fairly extensive scale on bulls and rams justifies the conclusion that in addition to its simplicity it is also quicker and causes less physical discomfort to the animal than any other method used previously.

A warning note must, however, be sounded. Though this method will greatly facilitate the collection and examination of semen in the field, and will be useful in the case of bulls and rams that are incapable of serving naturally or into an artificial vagina on account of some physical non-hereditary defect, it or any other type of electro-ejaculation should not replace collection by the artificial vagina for routine insemination purposes. As yet there are no data available on the effect which electrical stimulation applied through the rectal wall to the spermatozoa in the ampullae may have on them. There is just a possibility that it may influence the chromosomes and thus give rise to mutations, many of which may be of an undesirable type. Until progeny tests have shown these fears to be groundless it will be well to limit the use of electro-ejaculation in artificial insemination to those cases in which semen cannot be obtained with the artificial vagina.

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A NEW POISONOUS PLANT

by

P. L. UYS and T. F. ADELAAR

Pretoria

During 1954 it was definitely proved that the plant, Pavetta harborii is toxic to cattle. The toxicity is similar to that of the better known plant Pachystigma pygmaeum which is responsible for a disease in South Africa called "gousiekte". The latter is characterised mainly by its chronic nature resulting in a productive myocarditis, fatty degeneration and replacement of the cardiac muscle fibres by scar tissue. Death is, however, usually of a sudden nature (heart failure) and occurs 30 to 50 days or more after the ingestion of the plant.

Distribution and Description of Plant

Pavetta harborii is a reduced shrub with underground stems and erect, woody branches 20-40 cm. high and up to 6 mm. thick. The bark is smooth and greyish to pale yellow. *Leaves* are sessile, oblanceolate, entire 3-4.5 cm. long and 0.8-1 cm. broad, with scattered bacterial nodules, dark grey green and puberulous above, densely canescent below. *Inflorescence* corymbose, subsessile, producing dense clusters of flowers at the nodes. *Flowers,* white, scented, tetramerous; style well exerted. *Fruit* a small, pealike drupe, 1-2 seeded, black when ripe.

Distribution : On deep sandy soil in South Eastern Bechuanaland (Mochudi area) and North Western Transvaal in the districts of Rustenburg (near Rooibokkraal), Waterberg (Ellisras area) and Soutpansberg (Soutpan).

History : The species was described by Spencer Moore in Jour. Bot. (1919) 57, p. 89 based on a specimen collected by C. E. Harbor in 1914, of which an isotype is present in the National Herbarium, Pretoria. C. E. Harbor was the son of William A. H. Harbor, who in the early days of the Mafeking-Bulawayo railway ran a hotel and store at Mochudi Station (now Molotoana Siding) about nine miles north of Mochudi Villiage.

History of Mortality

The earliest reference to possible "Gousiekte" in the North Western Transvaal is contained in letters written by farmers to D.V.S. Onderstepoort during the years 1931, 1935 and 1936 and later during 1946, 1948, 1950 and after as a result of an unknown mortality amongst cattle. The following conclusions were arrived at from descriptions :

(1) The mortality occurs during the period April 1 to August (i.e. during autumn and winter) and especially during periods of drought.

(2) Cattle die as a rule very suddenly without showing any symptoms.

(3) Mainly cows with calves at foot and pregnant cows and heifers are affected.

(4) The mortality is restricted to certain areas only. These later proved to be those infested with P. harborii.

It was, however, only during July 1936 that the mortality was first investigated by Dr. T. F. Adelaar following severe losses amongst the cattle of a certain Mr. Potgieter, residing in the North-western area of the Transvaal. According to a letter written by Mr. Potgieter, 56 head of cattle died within a month without showing any definite symptoms of disease except for the fact that whilst some animals were found dead others were seen to just die suddenly.

The investigations of Dr. Adelaar led him to the conclusion that the cause of the mortality was poisoning by the plant Pachystigma pygmaeum. Histological results of specimens examined, further strengthened his diagnosis. The causative plant, however, could not be found on the farm or adjacent farms, but another known poisonous plant called Dichapetalum cymosum was found in abundance. (This plant is known as one of the most toxic plants for stock in South Africa, containing a potent cardiac and nerve poison. On ingestion of a sufficient quantity, death may follow within a few to about 24 hours afterwards, depending on whether the animals drink water or not. The toxic principle is highly soluble in water).

The presence of Dichapetalum cymosum and the complete absence of P. pygmaeum during a further investigation of the losses in April 1937 by Drs. D. G. Steyn and T. F. Adelaar, led them to believe that the cause of the mortality was the former plant.

Apparently no losses occurred since this last investigation until the winter months of 1946, 1947 and 1948 during which farmers again complained of a severe mortality of unknown origin amongst cattle, characterised by sudden deaths. No investigations, however, were carried out.

During April 1950, Mr. Potgieter and his neighbour suffered heavy losses again amongst their cattle. The mortality was investigated by one of us (P. L. Uys) and a tentative diagnosis of poisoning by Dichapetalum cymosum was made, taking into consideration that the whole history and course of the disease was that of "gousiekte" caused by P. pygmaeum. The interesting part, however, was that he, like Drs. Adelaar and Steyn, could not find this plant on the farm or adjoining farms, with the result that he became suspicious of the possibility that another plant might be responsible for these periodic losses.

During the investigation his attention was drawn to a certain plant, which proved to be in a reasonably green state for that time of the year. It was also noticed that this particular plant was fairly well grazed off by the cattle and for some or other reason he suspected it as a possible cause. Samples were taken for identification and simultaneously reports were received to the effect that histological examination of specimens proved to be "positive" for "gousiekte" and that the plant submitted was identified as Pavetta harborii, belonging to the same family (Rubiaceae) as the common "gousiekte" plant P. pygmaeum. This family relationship could be of significance or not and it was decided to test P. harborii for toxicity. Plant material was then fed to two sheep, each receiving 200 Grams over a period of six days. A severe enteritis followed and the sheep eventually died with symptoms of a cardiac poison. These experiments, however, did not prove that P. harborii was the causative plant.

During the period June/July 1952 another farmer suffered heavy losses amongst his cattle and upon investigation Dr. P. L. Uys came to the conclusion that the mortality was similar to that on Mr. Potgieter's farm during 1936 and 1950. No P. pygmaeum or Dichapetalum cymosum could be found in the camps in which the cattle were kept but varying amounts of Pavetta harborii, were present. With the assistance and help of the farmer, plant material was collected and forwarded for feeding experiments with cattle and sheep.

In the 1st experiment 7.7 lbs. of a composite sample (leaves, twigs and stems) of the plant was fed to a sheep over 17 days and 43 lbs. to a bovine over 30 days. The sheep died 44 days after commencement of the experiment with symptoms of diarrhoea and the bovine suddenly dropped down dead 54 days afterwards, without any previous symptoms.

Towards the end of 1954 the experiment was continued on cattle alone, with similar results. In all cases histological examination of heart muscle showed a progressive myocarditis with replacement of muscle fibres by scar-tissue. Other microscopical changes were of minor importance.

Summary

(1) The history and cause of serious periodic losses in cattle in the North-Western part of the Transvaal is described.
(2) A plant Pavetta harborii similar in toxicity to Pachystigma pygmaeum, is responsible for these losses.
(3) The toxic principle of the plant affects the heart muscle causing replacement of the cardiac muscle fibres by scar-tissue, resulting in acute or sudden death, in spite of its chronic nature.
(4) The mortality is restricted to certain regions coinciding with the distribution of the plant.

(5) Losses are sustained mostly during the autumn and winter following a drought.
(6) Pregnant animals and cows with calves seem to be the most susceptible.
(7) Small stock (sheep and goats) are apparently not affected, although one farmer mentioned the loss of 30 sheep during the 1950 epidemic. Experimentally sheep may be affected, showing diarrhoea as a definite symptom before death.

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PURULENT PNEUMONIA IN FOALS CAUSED BY CORYNEBACTERIUM EQUI (MAGNUSSON)

J. F. W. GROSSKOPF
Onderstepoort

R. C. TUSTIN
Onderstepoort

R. W. MUIR
Kimberley

Primary pneumonia in foals caused by *Corynebacterium equi* (*Magnusson*) is to the best of our knowledge recorded for the first time in South Africa. The disease was recently encountered in a thoroughbred stud near Kimberley and soon afterwards positive specimens were received from Colesberg and Middelburg (C.P.).

Magnusson first described this condition in 1923. All his cases occurred on one farm in Southern Sweden. Ten foals were affected in one year and from each one he isolated a bacterium which he named *Corynebacterium equi*. This organism was also isolated from purulent pneumonia in foals in Germany by Miessner and Wetzel (1923) and Lütje (1923). It was referred to by them as *Corynebakterium pyogenes* (*equi*) and *Corynebakterium* (*pyogenes*) *equi roseum* respectively. In 1931 the bacterium was recovered from foals suffering from primary pneumonia in the U.S.A. by Dimock and Edwards.

Although the name *Corynebacterium equi* is in common use this organism is not specific for horses. It was isolated from cervical lymph glands and pulmonary lesions in swine by Bendixen and Jepsen (1939) in Denmark and subsequently by Karlson, Moses and Feldman (1940) in the United States and McDonald (1942) in Australia. In 1945 Holtman described a chronic pneumonia in calves caused by the same organism.

Recently Roberts (1957) isolated C. equi from a sheep affected with chronic pneumonia and pleurisy

Symptoms

The disease usually occurs in foals of two to four months of age. In most cases no previous illness has been noticed and there seems to be no relation between this disease and joint-ill which is usually met with in younger foals.

In this outbreak the main symptoms noticed were coughing and rattling sounds from the throat, especially after exercise or excitement. On the slightest exertion marked dyspnoea appeared. Notwithstanding daily high doses of different antibiotics the temperature remained at about 105°F. The pulse rate was increased and slight lachrymation was present. On percussion and auscultation numerous dull areas where found bilaterally over the lung area.

Crepitant and moist râles as well as friction sounds could be heard on auscultation of the lungs. No abnormal nasal discharges were seen throughout the course of the disease. Dimock and Edwards (1931), however, described a purulent nasal discharge in their outbreak in Kentucky.

Notwithstanding all these changes the foals apeared alert throughout and stayed in excellent bodily condition. They sucked and remained interested in food until a few hours before death.

The mortality is high, with any treatment, once lung abscesses are established. Magnusson (1923) reported a death rate of 80%. The course of the disease is usually from 6 to 14 days although antibiotic treatment seems to postpone death for up to two weeks.

Pathology and Post Mortem

The disease has in our cases been a primary respiratory infection. The infection apparently becomes established in the lung tissue, later spreading through the body causing a septicaemia and often metastatic foci in other organs.

The most marked lesions are broncho-pneumonia with multiple abscesses scattered throughout the lungs. These abscesses may vary in size from a few millimetres to about 8 cms. in diameter. They contain a dull yellowish pus which is fluid in the smaller foci but more solid in the larger ones. Different lymph glands may be enlarged and may also contain metastatic abscesses.

Characteristics of the Organism

These were described by Magnusson (1938) and by Bruner, Dimock and Edwards (1939). The organisms isolated from the liver of one foal and lung abscesses of the three others resembled the organism described by these workers.

Pathogenicity

Although *Corynebacterium equi* was first isolated from foals and thus named after the horse it is also pathogenic to calves and young pigs. Dimock and Edwards (1931) successfully infected adult horses. Their temperatures soon rose to about 106°F and they lost their appetites. Both pulse and respiration were accelerated. Pyrexia only lasted for 48 hours, and recovery was uncomplicated.

According to Dimock and Edwards (1939) *Corynebacterium equi* is apparently non-pathogenic for laboratory animals. Cultures injected into guinea-pigs, rabbits and rats did not result in infection.

Treatment

All affected foals with their dams should be isolated immediately and the temperatures of all other foals on the premises taken daily. Treatment should be instituted after the first signs of fever. Chloramphenicol at the rate of ½ to 1 Gm. intramuscularly twice

daily, seems to be the drug of choice. It may be given alone or in combination with sulphadimidine. Once abscesses become established in the lungs treatment with antibiotics seems to be ineffective. It is very important, therefore, that treatment should be started with the first rise of temperature and not after the symptoms have appeared.

Measures such as the provisions of warm well ventilated stables, restriction of exercise, steaming with Eucalyptus oil and covering with blankets during cold weather should assist the patient in its recovery.

As calves and piglets have already been proved to be susceptible to this disease it is advisable to keep affected animals away, not only from the other foals, but from all young animals.

SUMMARY

Three outbreaks of purulent pneumonia, caused by *Corynebacterium equi*, in foals in South Africa are reported. Treatment with chloramphenicol and sulphadimidine seems to be successful only in the early stages before symptoms of lung abscesses appear.

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IMPORTANT STATEMENT

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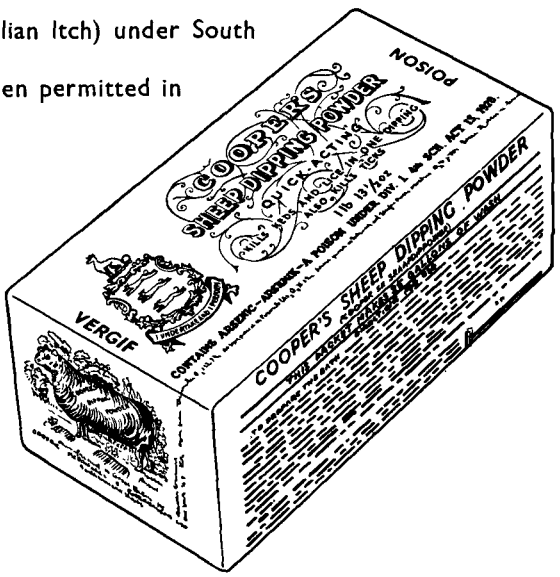
Furthermore, the Authorities had previously accepted and registered C.D.P. (Q.A.) as a dip for the eradication of Keds and the control of Sheep Lice at single dippings.



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FROM A SURGEON'S CASE BOOK

A FEW CASES OF POST-TRAUMATIC DYSPNOEA IN THE DOG

C. F. B. HOFMEYR
Pretoria

Dyspnoea in dogs after street accidents is quite common. It has many causes, which should not be confused with those producing hyperpnoea, like internal haemorrhage. It is not intended to present an extensive differential diagnosis or a lengthy discussion, but to write brief notes on a few cases, using diaphragmatic hernia as the main condition.

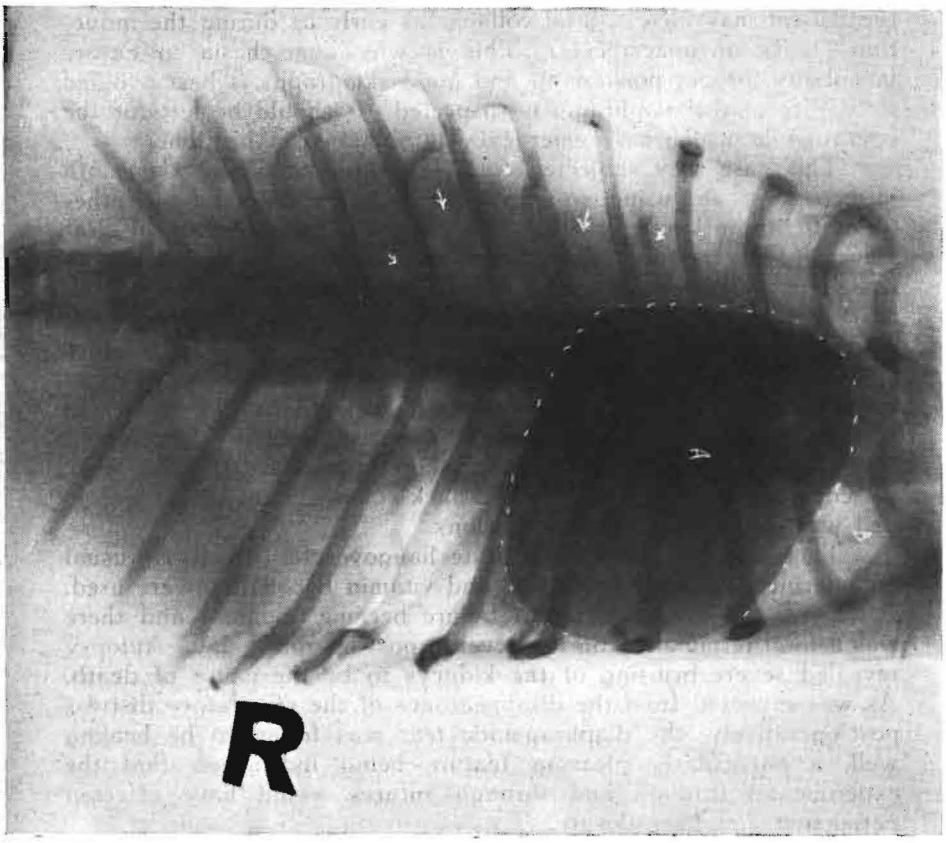

Fig. 1.
A shows the heart shadow transposed to the right chest wall.
B indicates normal lung.
The arrows point to uneven opacities caused by parts of the alimentary tract and, possibly, spleen.
An outline of the normal diaphragm can be seen just below the lower "B". On the left hand no diaphragm is visible.

CASE 1 (Fig. 1):

A Dachshund was presented for examination soon after an accident. Besides the clinical findings of extensive bruising, pallor of the visible mucous membranes and dyspnoea were present. The heart beat could more clearly be felt and heard on the right chest wall than on the left. Similarly, at auscultation, the respiratory sounds were clearer on the right than on the left. Percussion confirmed the suspected transposition of the heart and showed patches of decreased resonance or dullness on the left.

Diagnosis : Left diaphragmatic hernia and internal haemorrhage.

Röntgenographic examination confirmed the diagnosis. Owing to the absence of dense shadows, it was thought that the left chest contained parts of the alimentary tract and spleen, but not liver. The presence or absence of the liver in the chest may have an important bearing on the prognosis. If one or more lobes of the liver are half strangulated, hydrothorax is likely in which case the patient may have a fatal collapse as early as during the induction stages of anaesthesia. This is why anaesthesia to ensure immobility, proper positioning and good skiagraphs is best avoided if rupture of the diaphragm is suspected. It should be left for the operation in which case emergency intubation can be done.

This case was subjected to a left thoracotomy at the ninth intercostal space (using a pump and a Magill's endotracheal catheter with an inflatable cuff). The diagnosis was confirmed. Replacement of the herniated organs (spleen and small intestines) was as easy as was suturing of the dorsal three-quarters of the hernial wound. The ventral quarter represented a tearing of the diaphragm from its costo-chondral attachment and was at right angles to the direction of the surgical wound. The usual method in such a case is to join the tear to the incision. This was impossible here as the lower part of the tear was inaccessible. Interrupted sutures were thus passed from the skin through the thoracic wall, the edge of the unsutured tear and back again. Stout catgut tightly tied was used to encourage adhesions.

This case had some barbiturate hangover for which the usual methedrine, injectable vitamin C and vitamin B complex were used. After five days signs of renal failure became apparent and there was a fatal termination on the seventh post-operative day. Autopsy revealed severe bruising of the kidneys to be the cause of death. As was expected from the disappearance of the respiratory distress post-operatively, the diaphragmatic tear was found to be healing well, a particularly pleasing feature being indications that the experimental through and through sutures would have affected permanent and firm closure.

CASE 2 (Fig. 2):

A Dobermann crossbred dog was seen by Dr. C. L. Craig shortly after being knocked down by a motor car. It had pronounced dyspnoea. The usual treatment was administered. The case was then admitted to hospital and an X ray photo taken.

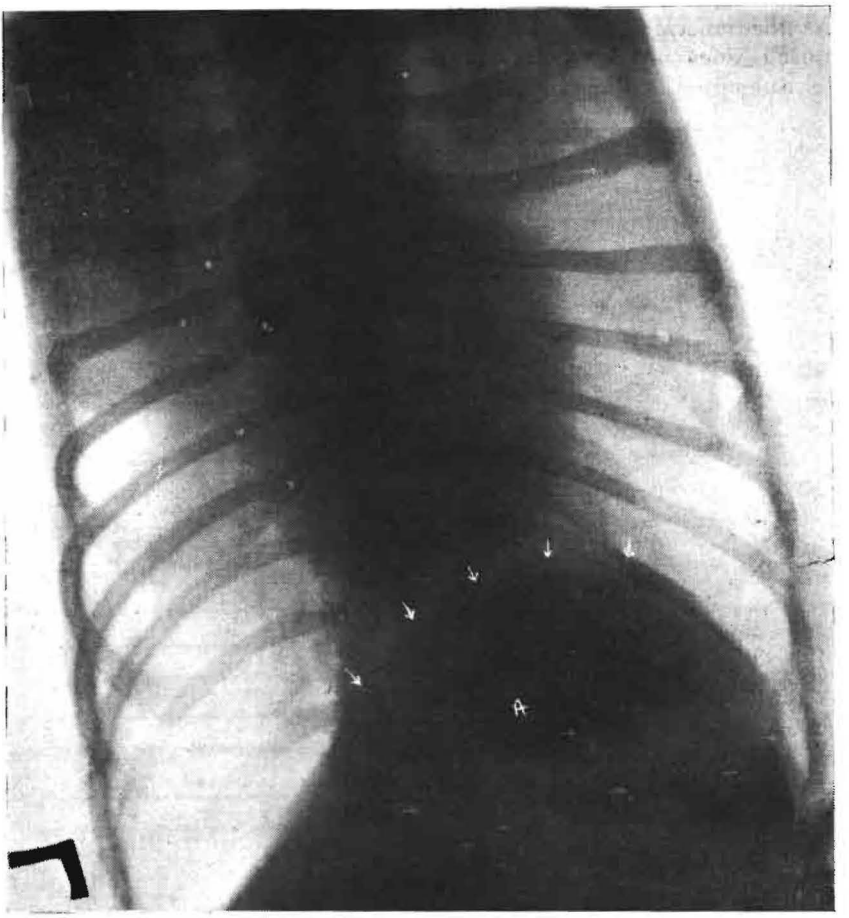

Fig. 2.

A indicates the dense shadow of the liver.
The arrows point to the marked, abnormal bulge of the liver into the thoracic cavity, leading to the presumption that the liver was partly herniated through the diaphragm.
The heart shadow is normal.

The author was then consulted. The only important clinical finding was some decreased resonance over the diaphragmatic lobe of the right lung.

Diagnosis : Right diaphragmatic hernia containing liver was suspected. This appeared to be confirmed by the skiagraph, but was subsequently proved wrong.

A right thoracotomy was performed. The thoracic cavity contained no abdominal organs. There was a tear in the post-cardiac mediastinum. The diaphragm bulged deeply into the thorax. On palpation the bulge was found to contain liver. The fleshy portion of the diaphragm had a number of rents of the muscle fibres, but the pleura and, as far as could be judged, the peritoneum were

intact. The part of the diaphragm in view appeared paralysed. As the muscle separations were multiple and spread out and as muscle does not hold sutures, no repair of the diaphragm could be attempted. The operation wound was closed and the owner

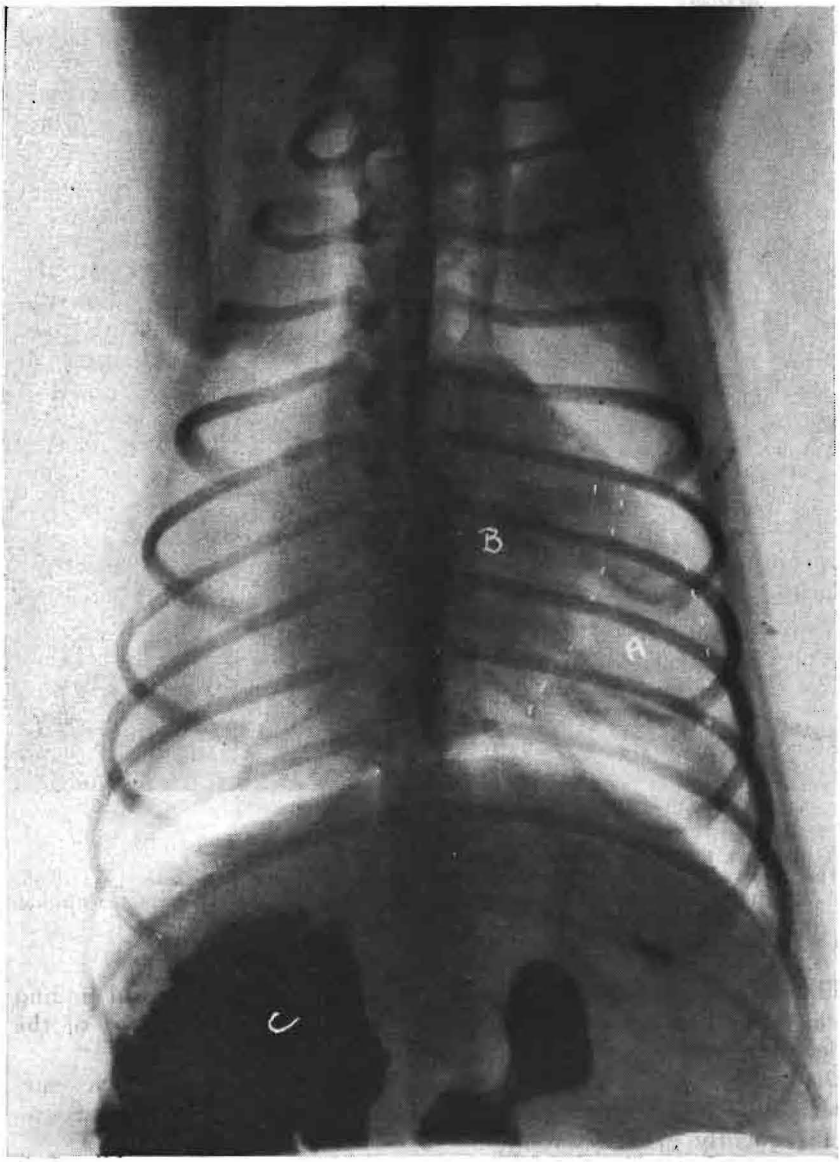

Fig. 3.

A shows an area of lung consolidation as a result of haemorrhage. The rest of the lung fields are clear.
B is the normal heart shadow.
C represents barium sulphate (shadoform) administered to act as a radio-opaque agent for contrasting the stomach or small intestine in case of herniation into the chest.

informed that paralysis of part of the diaphragm would cause persistance of the dyspnoea. It was felt that, even if the paralysis of the diaphragm did not improve, the dyspnoea would become less. as the chest wall would adapt itself by increasing the costovertebral angle. When the case was discharged a week after operation, it showed appreciable improvement.

This case illustrates the difficulty sometimes associated with diagnosis, even in the presence of diagnostic aids like X ray apparatus. As the liver bulging through a single large tear would give the same picture, operation is indicated in every case showing these radiographic features.

CASE 3 (Fig. 3):

A small Fox terrior was examined soon after having been run over in the street. It had severe dyspnoea and, besides pallor and cyanosis of the mucous membranes, there were râles in both lungs. some crepitation and a small area of dullness in the right diaphragmatic lobe. There was no haemoptysis. Treatment was given as in Case 1.

Diagnosis: A diagnosis of pulmonary bruising and haemorrhage was made. An X-ray taken showed a consolidated area in the right diaphragmatic lobe i.e. haemorrhage. If there had been a subpleural haematoma, draining through a needle after a few days or, if unsuccessful, thoracotomy could have been contemplated. This haemorrhage. being in the substance of the lung, required, above all, rest. After some days in hospital the case was discharged, clinically normal.

FOOTROT IN SHEEP

(A review)

R. C. TUSTIN

Onderstepoort

Until about 1941 it was generally considered that the aetiological factor in foot rot in sheep was Fusiformis necrophorus. However, Professor Beveridge working in Australia found that the disease was caused by Fusiformis nodosus in association with a spirochaete, S. penortha. F. necrophorus is the causal agent of footrot in cattle and a condition in sheep called foot abscess. It is suggested here that we adhere to the terminology used overseas.

Footrot in sheep is a contagious disease of the foot characterised by the separation of horn from the underlying soft tissue, due to a spreading infection under the horn. It usually runs a chronic course.

Economic Importance

Mortality occurs mainly among lambs which affected ewes are unable to suckle. These cases are, however, infrequent. The main economic loss is sustained through the loss of body weight, the decrease in quantity and quality of the wool and the cost of treatment.

Aetiology

It is sufficient to mention here that F. nodosus is considered to be the primary causal agent, and it is very probable that S. penortha is a specific accessory causal agent.

Both these organisms are obligatory parasites and only the diseased foot of the sheep or goat appears to be their natural habitat.

A motile fusiform organism is also usually mentioned in connection with this disease. This organism is a constant secondary invader and probably does play some part in the pathogenesis of the condition. Micrococci, corynebacteria and other organisms are also usually found in the lesions. F. Necrophorus is sometimes present as a secondary invader and may be responsible for some of the complications such as joint infections, etc.

Survival of Infective Material in Nature

The maximum period of survival outside the host under the most favourable conditions is not more than two weeks. In areas where the disease breaks out periodically, the infection is either reintroduced or survives the inter-epizootic periods on the feet of some sheep. In other words carriers occur.

These facts are important as the control and eradication of footrot are based upon them.

Natural Transmission

Infective material is deposited on the soil from diseased feet and is picked up from there by healthy feet.

Incidence and Susceptibility

The disease is most prevalent during wet warm periods. During dry spells the disease may not be seen for years. On the other hand in wet years the incidence may be as high as 75%.

On irrigated pastures the disease may be expected to occur throughout the year. In South Africa this type of pasture is becoming more common, and we may expect the incidence of the disease to increase accordingly. The higher the carrying capacity of a pasture is, the wetter it is and with more sheep to the acre the chances of infection are greater.

Sheep of all ages are susceptible. Some claim that young sheep are more resistant than older animals. Rams are perhaps more susceptible than ewes, presumably because of their greater weight.

The merino is considered to be the most susceptible breed. British breeds are probably less susceptible than merinos, and crosses between merinos and British breeds come in between the two. This is resistance to initial infection, however, and once the infection is established the lesions are as severe as in the merino. Goats are susceptible to foot rot both in the field and experimentally. Bovines are not susceptible to a natural attack. Foot rot in bovines, as we know it, more closely resembles foot abscess.

Predisposing Factors

Since foot rot has a fairly well defined seasonal incidence it seems that something more than a mere contact with infective material is necessary to bring about transmission of the disease. This fact also holds experimentally, where the application of infective material to the healthy foot does not set up the infection. The resistance of the foot must be decreased in some way beforehand e.g. by scarification. Under natural conditions it is believed that chapping or chafing by continued wetness is the main factor, although this point has not yet been fully clarified.

An outbreak may follow after sheep have been driven through long wet grass or over stony ground. The wounds caused by the larvae of worms such as Strongyloides papillosus and Bunostomum spp. which can penetrate through the skin have also been incriminated as predisposing factors.

Symptoms

The initial lesion is usually a mild inflammation in the interdigital space. A break in the skin horn junction then occurs and the infection spreads between the horn and the underlying soft tissue of the foot. The accumulation of necrotic material causes a separation of the horn. During the next few days the separation progresses to the edge of the sole and then under it or around the heels. Eventually the hoof may only be held onto the foot by

the skin of the coronet. Pus is in evidence although there is never any copious production of it. Usually both digits of a foot are involved.

Due to lack of wear the hooves become overgrown and misshapen. Without treatment the lesion may persist for months, and then heal or partially heal spontaneously. Fever may occur and this may result in a break in the wool.

The infection may spread up the leg, and joints, tendons, ligaments and bones may become involved, rendering the animal permanently lame. These secondary lesions are probably caused more by the secondary invaders.

At the commencement of an outbreak the first indication is the appearance of lame sheep. On examination the affected digit may feel hot, and if pressure is applied, the animal may flinch. Lameness is progressive and becomes more severe as the infection spreads in the foot, until the leg is carried. If both forefeet are affected the animal will graze on its knees, if both hind feet, the animal will attempt to move along on its belly. Affected animals lie down frequently and lose weight rapidly.

The duration of the disease is usually several months and one case reported by Beveridge persisted for three and a half years.

A pocket infection may persist and these carrier sheep can be detected by trained observers by the misshapen horn of the hoof. The infection may also be carried for several months in the interdigital space, the lesion being a more moist appearance than normal of this area.

Diagnosis

This is based on the symptoms, lesions, incidence and the examination of smears made from an active part of the lesion. More than one smear should be made from a lesion and smears should be made from several cases. Failure to detect F. nodosus in a smear does not justify a negative diagnosis.

Differential Diagnosis

(1) *Foot abscess*: This disease is caused by Fusiformis necrophorus (also known as Bacterium necrophorus, Actinomyces necrophorus, Spherophorus necrophorus and Bacteriodes funduliformis or Bacteriodes necrophorus). The organism is considered to be a normal inhabitant of the gut of animals and man and is responsible for a variety of diseases. This fact makes any control measures of the organism rather difficult.

The main points of difference between foot abscesses and foot rot are :-

(a) In foot abscesses the first sign is usually an acute lameness. No obvious lesions may be present but careful paring may reveal imprisoned pus. In older cases quite a large area of horn may be underrun, with an abscess finally breaking out at the coronet or between the digits.

(b) Usually only one foot of a sheep is affected and only one digit of a foot. In foot rot frequently both digits and more than one foot are affected.

(c) Isolated cases of the disease occur especially in stud rams, but when outbreaks do occur in wet seasons usually only about 10% of the flock may be affected, whereas foot rot spreads rapidly through the flock.

(d) Foot abscess is characterised by pus formation. In foot rot excessive pus formation is not common, and when it does occur, is usually due to secondary infection.

(e) Complications are more common in foot abscess as the infection more frequently spreads up the leg.

(f) Some claim that foot abscess responds to sulphonamide and penicillin treatment. This is not the case in foot rot.

(g) Foot abscess arises spontaneously under wet conditions.

(2) *Strawberry foot rot* which is presumably caused by Actinomyces dermatonomus.

(3) *Post dipping lameness* due to Erysipelothrix rhusiopathiae infection.

(4) *Scald.* This appears to be a distinct condition and is characterised by an inflamed appearance of the skin between the claws and accompanied by some exudation of fluid. It may occur on properties free of foot rot and is associated with frost and wet pastures.

(5) *Laminitis* and any other condition affecting the foot.

Treatment

The surgical preparation of the foot is the most important part of the treatment. All underrun horn should be pared away, thus exposing infected areas and allowing for drainage.

A 30% solution of copper sulphate or a 10% solution of formalin is used as a routine dressing in flocks. After paring, the foot is either immersed in a chosen solution for several minutes, or the sheep are passed through a footbath containing the solution. They should be held in the footbath for several minutes.

If copper sulphate is used, care must be taken to avoid staining the wool. In order to reduce splashing in footbaths straw or wool is placed in the bottom before filling. One must also ensure that for instance a sheep with one affected foot does place this foot in the solution and does not go through the bath on the three sound feet.

A 10% tincture of chloramphenicol seems to be the drug of choice. This is applied with a brush to the affected areas. Healing is evidently more rapid, a higher percentage of recoveries after initial treatment is obtained and less relapses occur. Sheep treated with chloramphenicol should not be passed through the footbath afterwards.

An ointment containing oxytetracycline also seems to be effective.

Treatment should, if possible, be treated at two to three day intervals until the animal is considered cured. The affected feet of valuable animals, may after treatment, be bandaged or placed in boots.

Obstinate cases which resist treatment should be slaughtered.

Immunity.

One attack of foot rot does not confer an immunity to subsequent attacks. A sheep affected in one foot may become infected in one or more of the other feet.

Control and Eradication.

Until fairly recently no attempt was made to control foot rot as it was believed that the causal organism was F. necrophorus. Now that all the facts are known it is possible to control the disease, and, what is more important, to eradicate it.

The campaign should be planned to begin during the dry season when the incidence of the disease is at its lowest. All fences and gates must be made stock proof and goats should be removed. A footbath must be constructed where the work is to be done and must be so situated that access routes do not interlace. A closed flock must be maintained, and if new members are introduced they should be isolated for at least a month after closely examining and paring the feet and passing through a footbath. If sheep have to cross a public thoroughfare they should pass through a footbath on the other side before entering the new camp.

During the weeks preceding the start of the campaign any lame sheep must be isolated. At least three camps must be kept free of all stock for fourteen days before commencing. One of these camps is to be used for clean sheep, one for a hospital camp and one for a convalescent camp.

The sheep are collected and every foot of every sheep is pared. A person who is experienced in detecting the disease examines the feet after paring. Affected sheep are placed aside to be treated as described. After treatment they are placed in the hospital camp after first passing through the footbath. Suspicious cases should be regarded as infective. Non-infected sheep are passed through the bath and are put into the camp which has been kept for them.

When a sheep in the hospital camp is considered cured it is placed in the convalescent camp after passing through a footbath. It is kept under observation for a period of at least two weeks and is put through the bath while in this camp at weekly intervals.

The clean sheep are inspected weekly and if foot rot appears in them the whole process is repeated. This camp must be kept free of stock for fourteen days before being restocked.

Footbaths for prophylactic use should contain a 2 to 5% solution of formalin or a 10% copper sulphate solution. Sheep should be held in the bath for several minutes and should not be allowed to walk straight through. Footbaths are usually twenty inches wide, ten inches deep and with a length according to the

size of the flock. This is usually fifteen to twenty feet. The formalin or bluestone solutions are maintained at a depth of three to four inches in the bath.

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I would also like to thank the director of Veterinary Services for permission to publish this article.

FROM A PRIVATE PRACTITIONER,S NOTE BOOK.

D. C. L. Wacher.

Matatiele.

(1) A NEW METHOD OF PREDOSING 10% COPPER SULPHATE SOLUTION IN SHEEP

The present method of predosing sheep with 10% Copper Sulphate solution to initiate the reflex closing of the oesophageal groove, is by means of the well known Copper spoon (X spoon). This is supplied together with a number of other different sized dosing spoons, by Veterinary Research Laboratories, Onderstepoort.

In view of the fact that the efficacy of certain Anthelminthics (e.g. Tetrachlorethylene) depends entirely on the dose arriving in undiluted form in the abomasum, the closing of the oesophageal groove must be considered of prime importance, therefore any method that will make this reflex more certain should be adopted.

In using the copper spoon the difficulty of transferring the solution from the copper sulphate container to the sheep's mouth without spillage is a feat, further any sudden movement of the sheep's head while being held for dosing may cause spilling of part or all of the solution. Where care is not being exercised the sheep is dosed without receiving the adequate predose.

The writer has during the past five years used a 5 c.c. plastic or nylon syringe with a 2 inch piece of plastic tubing (Polythene canula size 4) attached to the nozzle. An ordinary syringe with metal parts is not suitable, due to the corrosive effect of the copper sulphate, and glass syringes break too easily.

This method, using a plastic syringe, has proved extremely successful and has been adopted by the majority of sheep farmers in the Matatiele area, who have all commented on the superiority of it as compared with that using the copper spoon.

With good handling facilities it has been found possible to dose at least 500 sheep per hour. A spraying effect of the copper sulphate solution can be achieved by compressing the end of the plastic nozzle and the writer believes that a 2 cc. predose gives better results than the 1 cc. recommended.

(2) REVIVING SHEEP THAT BECOME UNCONSCIOUS FOLLOWING TETRACHLORETHYLENE DOSING

Following dosing of sheep with tetrachlorethylene emulsion severe reactions may be expected occasionally, and in many cases the affected sheep become unconcious. The usual method of dealing with these sheep is to put them in the shade and prop them against a wall or tree in a natural recumbent position to prevent bloating. The majority of these sheep will recover and stagger off within 1 to 8 hours, but there may be an odd death.

The writer has on many occasions experimented with reviving these sheep quickly and has had excellent results with the intravenous injection of *"Methedrine"* (Burroughs Wellcome), a solution of Methylamphetamine hydrochloride 30 mgm. in 1.5 c.c. The injection is given in the cephalic vein of the foreleg, a vein which is very easy to find.

Within half a minute of injecting the drug twitching of the lips and eyelids is noticeable, and a marked increase in the depth of respiration is apparent. Generally within 2 minutes the sheep will rise and stagger off to join the flock and though in a rather excitable state for a short period, will settle down and return to normal quickly.

The rapid results that can be expected and consequent raising of the farmers morale are well worth the small expense of the drug.

(3) A NOTE ON ILLUMINATION WHEN USING A SPECULUM IN THE EXAMINATION OF COWS FOR STERILITY

The satisfactory illumination of the anterior vagina and cervix when examining cows with a speculum is of great importance in conducting sterility examinations and the writer has had such excellent results by using a small mirror to reflect sunlight into the vagina, that it has prompted the writing of this note.

The idea is certainly not original and no doubt has been used by many practitioners but I believe that few South African veterinarians use this method despite our ample sunshine.

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THE ELECTROCARDIOGRAPH IN VETERINARY PRACTICE

C. D. MEREDITH

Davis, California, U.S.A.

That the muscular activity of the beating heart generated minute electrical currents demonstrable by applying electrodes, connected to a sensitive recording apparatus to its surface was established as a physiological phenomenon many years ago. It remained for Enthoven to show that these changes of electrical potential were transmitted through the body's electrolytes and were thus capable of detection in the intact body by using electrodes and an electrolytic paste or fluid to overcome skin resistance. Much of his work on the electrical axis of the heart and the classical limb leads I, II and III is as valid today as when first published.

The first electrocardiographs were large, bulky, very sensitive but very delicate instruments employing the string galvanometer and photographic principles, and by nature were hardly classifiable as portable or reliable under adverse conditions. The tremendous impetus given electronic research by war time exigencies has now produced the electromagnetic direct-wiring electrocardiograph with its advantages of portability, rugged construction and immunity to a host of conditions that would render a delicate string galvanometer type of machine inoperative or inaccurate. This type of electrocardiograph has gained wide acceptance in human medicine because of these advantages, the ease of operation and convenience of direct writing and, above all, because of the lower initial cost.

The medical man today draws on a vast store of knowledge, practical experience and research for the interpretation of electrocardiograph recordings and it is this background that makes possible the accurate diagnosis of often obscure and complex cardiac ailments. Unfortunately in animals this background is absent and electrocardiograph interpretation is still largely a matter of empirical application of the criteria used in the human field.

What then is the value of this complex expensive apparatus to the practising veterinarian?

I. *The Human Application*: Electrocardiograph tracings yield information falling mainly into four groups:—

1. Regularity of heartbeat.
2. Site of origin of arrhythmias (i.e., defective conduction system or ectopic pacemakers).
3. Position of cardiac lesions (e.g., infarctions) and their progress.

4. The alignment of the cardiac axis and its deflection by alteration of the heart's position occasioned by conditions such as hypertrophy.

II. *In Domestic Animals*: If we consider the above four groups as applied to animals then we must modify them as follows:—

1. Regularity of the heartbeat: Especially in the canine, sinus arrhythmia is normal in varying degrees as also "dropped beats" (sino auricular block) in the resting condition of animals adapted to regular exercise. Both of these are of vagal origin and not to be differentiated from similar conditions of a pathological nature.
2. Practically the same information as to the origin of arrhythmias is obtainable as in the case of man, though high pulse rates and difficulties in eliminating interfering electrical currents produced by other muscular activity (somatic tremor) complicate interpretation. It is in this group that most information is gained and the electrocardiograph proves of maximum value in animals.
3. Cardiac infarction is almost unknown in our domestic animals both because of low incidence and lack of diagnostic criteria. However, when present it would presumably yield certain electrocardiograph characteristics especially when explored with the different pre-cordial leads as in the human being.
4. With our many breeds within each species information on the electrical axis in normal and pathological states is largely lacking and when present confused. Especially in dogs, the variety of thoracic conformations and wide differences in athletic adaption lead to a multiplicity of patterns, hence no standards have as yet been evolved.

In addition to the above groups, the morphological pattern of the electrocardiograph in animals differs considerably from that of man. Lead I is generally poor for evaluating the complexes as their amplitude is very small. This may be due to the relatively more posterior placing of the canine and equine hearts. Lead II, and to a lesser extent Lead III, are the leads of choice and a standard technique should be adopted to minimise variable factors, e.g., positioning of animal.

For those not familiar with the normal electrocardiograph pattern, the nomenclature and tracing correlation, a brief description follows (see Figure 1):—

The first electrocardiograph deflection, or wave, produced in a cardiac cycle is designated the P wave and corresponds to the change in potential produced by atrial contraction. Following this the stylus returns to the isoelectric line for a brief period — the PQ interval — which is considered to indicate the time during which the impulse traverses the AV node and enters the bundle of His. The QRS complex designates the spread of the depolarisation wave through the myocardium of the ventricles and therefore corresponds

to the onset of ventricular systole and the first heart sound. When the heart is fully activated there is no further change in electric potential and thus maximal contraction is reached during the ST interval which is therefore normally isoelectric. The recession of cardiac activity, onset of diastole and repolarisation of the myocardium is shown by the T wave which is usually synchronous with the second heart sound. This marks the commencement of the diastolic phase and the TP interval is again isoelectric in the absence of interference or somatic tremor.

Comment on the duration times of the various complexes is omitted since both time and amplitude values show considerable variation from animal to animal, though each animal has a constant pattern. Were it thus possible to obtain "pre" and "post" illness tracings very much more information could be obtained than from the single "post" recording. One of the most striking differences seen when comparing the canine electrocardiograph with that of man is the QRS complex amplitude which may be as much as 6 Mv or more, when the total deflection (i.e., both negative and positive components) is measured. The QRS amplitude is also influenced considerably by respiration, often rising and falling with each breath.

The number of negative components in canine tracings is much more than in man, and the complex most frequently affected is the T wave. Its inversion is often associated with the presence of a Q wave and in Lannek's series of cases it was found that when Q measured 4 mm. or more then 90% of T waves were inverted.

Increase in heart rate usually increases P amplitude. Since the main value of the electrocardiograph in our animals is in determining the origin and extent of cardiac irregularities, these may be considered in more detail. They may conveniently be classified as follows (after Best and Taylor):—

A. Affections due to impaired conduction through A.V. bundle and its ramifications:—

- I. Stem of Bundle:
  - (1) Delayed conduction.
  - (2) Partial Heart Block (missed beats).
  - (3) Complete Heart Block.
- II. Bundle Branch Defects:

B. Affections due to abnormal impulse formation:

- I. Extrasystoles.
  - (1) Ventricular.
  - (2) Nodal.
  - (3) Auricular.
- II. Paroxysmal tachycardia. (Auricular, Nodal, Ventricular).
- III. Auricular flutter.
- IV. Auricular fibrillation.
- V. Ventricular fibrillation.

C. Affections due to Vagal influences.

I. Sinus arrhythmia.

II. Phasic irregularity.

III. Sinus Bradycardia.

IV. Sino-auricular block.

Briefly these may be elaborated as follows:—

(1) Delayed conduction: The intervals between auricular and ventricular systoles are lengthened, i.e., PQ interval. This can only be diagnosed by electro-cardiograph.

(2) Partial Heart Block (Missed Beats). When impaired conduction reaches a certain degree impulses from time to time fail to reach the ventricle and a beat is missed, i.e., P is present but QRS and T are absent. May be regular (2:1, 3:1, or 4:1) or irregular. The PR intervals may progressively increase in complexes preceding the missing beat, returning to normal immediateley after.

(3) Complete Heart Block: Complete dissociation of P and ventricular waves.

II. *Bundle Branch Defects*: Here impaired bundle branch conduction and muscular transmission lead to asynchronous ventricle contraction. Generally appears as QRS prolongation and inversion. Mainly associated with coronary disease in man, however, since strands of conducting tissue also cross the right cavity of the canine heart the application of human criteria is empirical. Bundle Branch disease is of gravest significance, the subject rarely surviving many months after its discovery.

B. I. *Extrasystoles*: An extrasystole can be produced by stimulating the cardiac muscle at any time other than its phase of absolute refractoriness. Usually due to some abnormal impulse formation. Though extrasystoles may be associated with organic heart disease they may more frequently occur in its absence. They may originate reflexly from the viscera or from intoxication, e.g., digitalis, chloroform.

(1) *Ventricular extrasystoles*: These are followed by a compensatory pause. However, when the heartbeat is very slow this may not occur — the so called "interpolated beats". When compensatory pause does occur then term "extrasystole" is incorrect since the succeeding normal beat is displaced.

(2) *Auricular extrasystoles*: Arises from an ectopic centre of stimulus. An auricular extrasystole, therefore, causes a premature contraction of the whole heart. There is rarely a long compensatory pause.

(3) Nodal extrasystoles are uncommon and exhibit simultaneous or nearly simultaneous responses from auricle and ventricle.

Extrasystoles: *Prognosis*: Doubtful if they are indicative of an impaired heart or that they add to the gravity of an existing condition (not necessarily cardiac, e.g., pneumonia) — this refers to ordinary cases. Multiple extrasystoles are, however, serious.

Heartb'ock: 1. The occurrence of partial heartblock during the course of an acute infective disease is a sign, and may be the only sign, of myocardial involvement.

2. Persistent heartblock of a mild degree even is also indicative, of myocardial damage but not necessarily of interference with function.

3. Persistent HB of a severe degree. When indications of cardiac failure are present and especially if these are progressive — life is in danger.

II. *Paroxysmal tachycardia*: This may be defined as a condition in which the rate of the heart is greatly accelerated for a longer or shorter period without obvious cause. There is sudden onset and cessation. The paroxysm, it is believed, consists of a series of rapidly recurring extrasystoles which completely submerge the physiological rhythm.

III. *Auricular flutter*: This may be pure or impure depending on regularity. Differs from paroxysmal tachycardia as follows:—

(1) Rate is usually greater.
(2) Disorder of much longer duration up to months or years.
(3) Ventricle fails to keep pace with the racing auricle due to relatively longer refractory period, thus a varying fixed AV block develops.
(4) Circus movement (i.e., the propagation of the impulse over one or more circular pathways) exists in the disordered auricles.

IV. *Auricular fibrillation*: "The auricular muscle is the seat of incomplete contractions occurring at a rate of 400-600 per minute. So incomplete are the contractions and so rapidly are they produced that the individual beats are scarcely distinguishable from one another. The auricular cavity is never emptied of blood and its wall is a quivering sheet of muscle. This is the most common of serious cardiac irregularities and is usually due to myocardial disease." Ventricular rate slower, but nevertheless faster than normal, irregular and often showing a "pulse deficit". With treatment and improvement in the condition of the cardiac muscle the pulse rate may therefore increase.

V. *Ventricular fibrillation*: "The ventricular muscle may pass into a state of rapid, tremulous and ineffectual contractions closely similar in nature to the condition just described as occurring in the auricle. This may arise from certain chemicals or drugs in excess, e.g., digitalis, calcium chloride. Chloroform anaesthesia renders the hearts of experimental animals highly susceptible to fibrillation. A mere touch of a finger or instrument, the stimulation of a sensory nerve, section of the vagi or their paralysis by atropine may set the

ventricle fibrillating. Adrenalin was found to greatly enhance the effect of chloroform a fact which indicates the danger of adrenalin stimulation under this anaesthetic. "Cyclopropane has the same effect as chloroform but ether is relatively safe".

"The effects of ventricular fibrillation on the circulation are incomparably more serious than those of the corresponding auricular condition. In fibrillation of the lower chamber the propulsive circulation comes to an end."

"In man it may result from:—

(1) Electric shocks.
(2) $CHCl_3$ or Cyclopropane anaesthesia.
(3) Coronary occlusion and other causes of acute anoxia.
(4) Trauma to heart or thorax.
(5) Sequel to paraxysmal ventricular tachycardia.
(6) Toxic doses of digitalis or quinidine."

"The three conditions namely, pure and impure flutter and auricular fibrillation are believed due to a disturbance of impulse initiation and transmission which is of fundamentally the same nature in all three but varies in degree in each. These clinical conditions are due to a circus movement of the excitation wave, i.e., the wave starting at one point takes a devious course through the cardiac musculature to reach the point from which it started and re-enter the path which it had previously traversed."

C. 1. *Sinus arrhythmia*: Rhythmical variations in the rate of the whole heart occur synchronously with respiration. It is due to alterations in the strength of the vagal influence upon the pacemaker (S.A. node) as a result of respiratory excursions.

2. *Phasic Irregularity*: Here periodic slowing of the heart occurs for a few seconds independently of the respirations. May be abolished by atropine thus showing it to be of vagal origin.

3. *Sinus Bradycardia*: Persistent slowing of the heart due to increased vagal tone. Common in athletes and animals conditioned to regular exercise.

4. *Sino auricular Block*: The entire heart (auricles and ventricles) misses a beat at regular or irregular intervals. Differs thus from A-V block where only the ventricle misses. One whole complex P-T is thus omitted. May be abolished by atropine, induced by vagal stimulation and sometimes digitalis administration.

The above "theoretical" grouping may for practical purposes be simplified as follows:—

The Horse:

The thoroughbred racehorse depends on a sound heart for his stamina and speed, hence the importance of accurate diagnosis of heart ailments. Since many irregularities are not detectable by auscultation or pulse palpation the electrocardiograph is particularly valuable in this field.

An electrocardiograph is indicated in all cases where although a horse appears perfectly fit and well; at exercise or during a race the animal slows up without showing any obvious signs of distress and soon recovers.

The commonest cause of this is partial heart block which may be suspected on auscultation and confirmed by an electrocardiograph.

Arrhythmia and auricular fibrillation also occur but less often. Some cases have been ascribed to overwork, spontaneous origin or sequelae to infectious conditions, e.g., strangles, biliary fever.

The Dog:

Apart from the irregularities detailed above some general principles have been observed in the dog. For example an abnormally high T amplitude is common in uraemia as there is also an increase in QRS duration.

Pericarditis with effusion has been associated with low QRS and T waves. Myocarditis associated with thallium poisoning, acute gastroenteritis, uraemia, pneumonia and certain infectious processes has produced:—

(1) Depression of the T wave and ST Segment.

(2) Increase in QRS interval with notching.

(3) Auricular, fibrillation, ventricular extrasystoles, atrioventricular block.

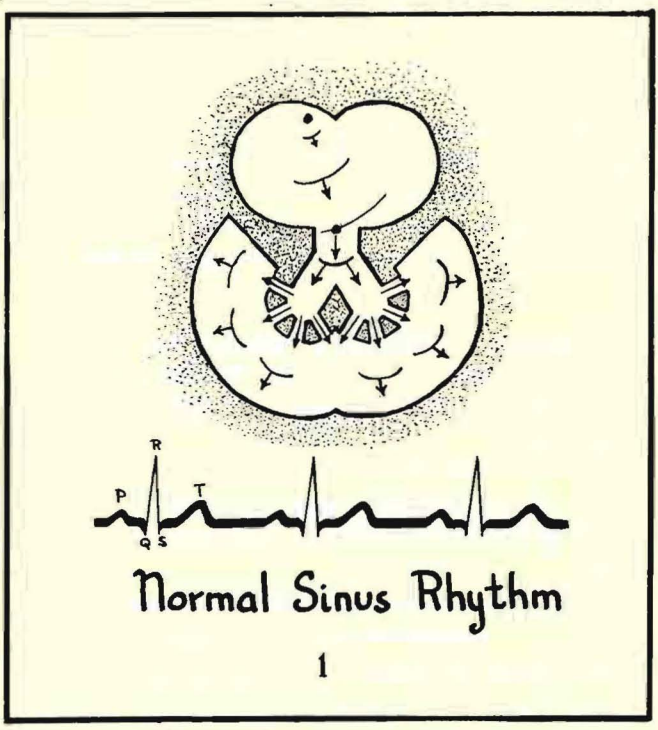
The Bovine:

Very little information exists on electrocardiograph application in cattle. Presumably traumatic pericarditis would inevitably produce marked changes in the normal pattern.

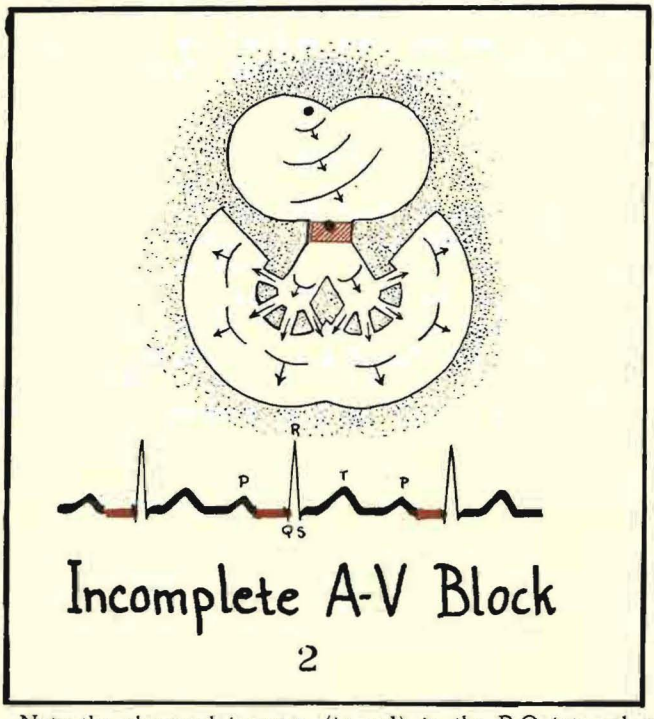
Conclusion

In conclusion it can be suggested that although there is much valuable information obtainable from electrocardiograph tracings in diagnosis and prognosis of disease conditions of heart and other systems the extent of our knowledge hardly justifies the expense involved with the single exception of the well established lucrative equine practice. The practitioner who claims to have the interests of his clients and profession at heart can hardly omit to utilise this useful diagnostic asset where valuable racehorses are involved.

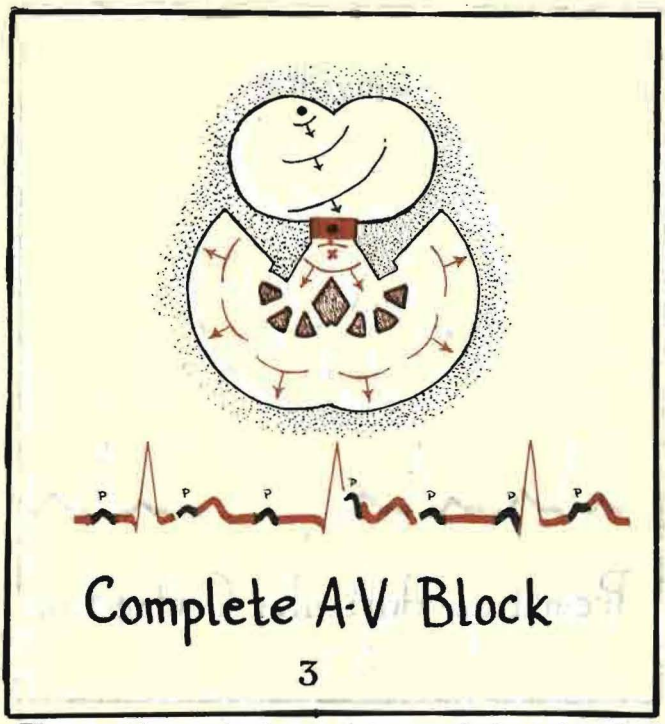
DIAGRAMMATIC PRESENTATION OF ELECTROCARDIOGRAPHS
IN VARIOUS CARDIAC ARRHYTHMIAS



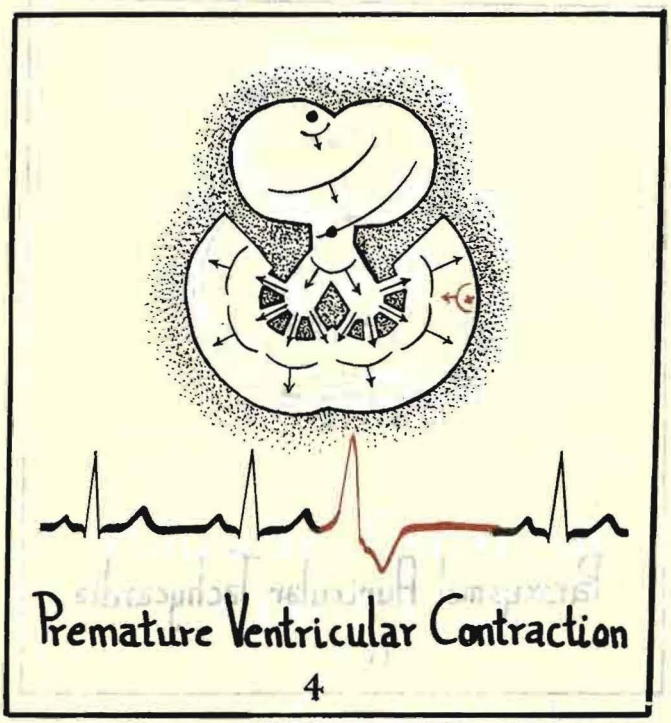
No. 1. — For description see text.



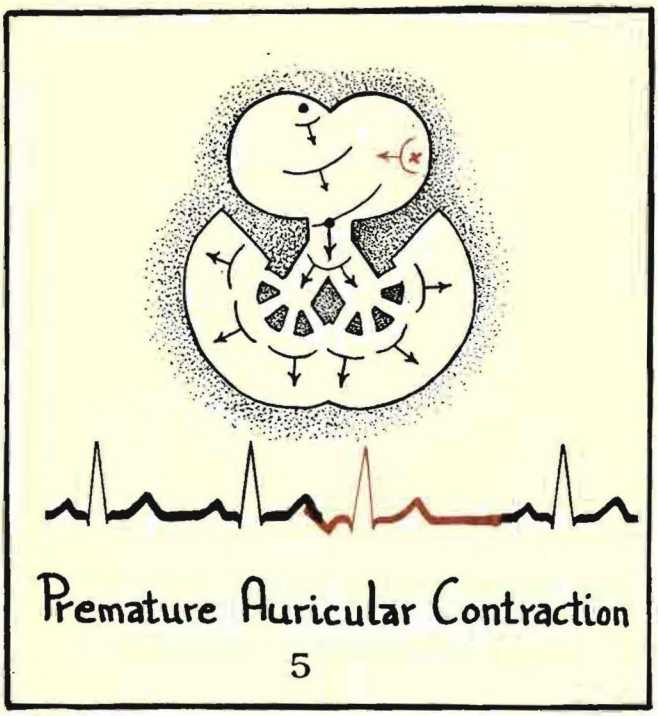
No. 2. — Note the abnormal increase (in red) in the P-Q interval caused by the delay in transmission of the impulse through the A-V node.



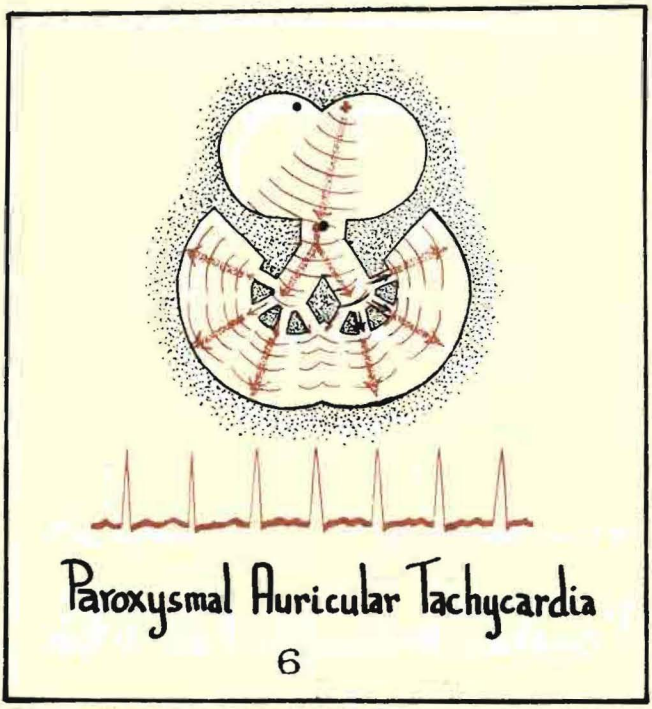
No. 3. — The ventricles are beating to their own rhythm independently of the auricles.



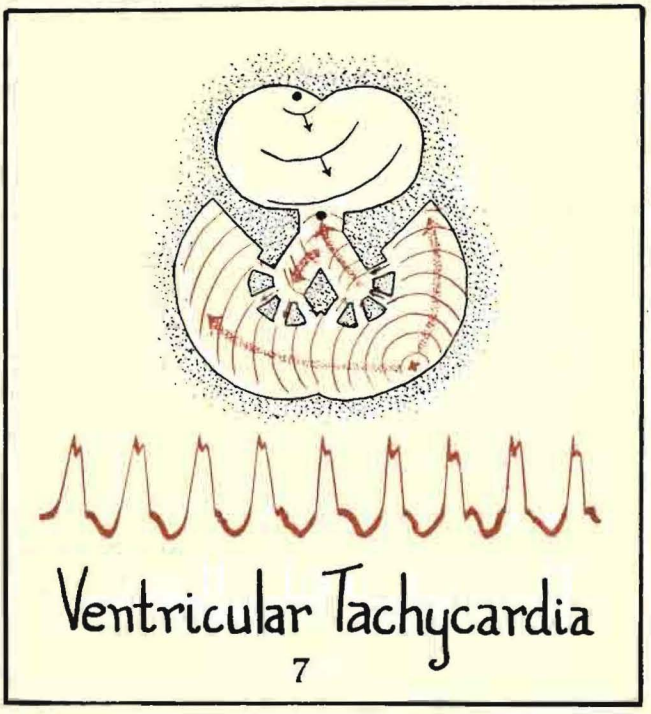
No. 4. — Note the extra systole followed by the typical compensatory pause.



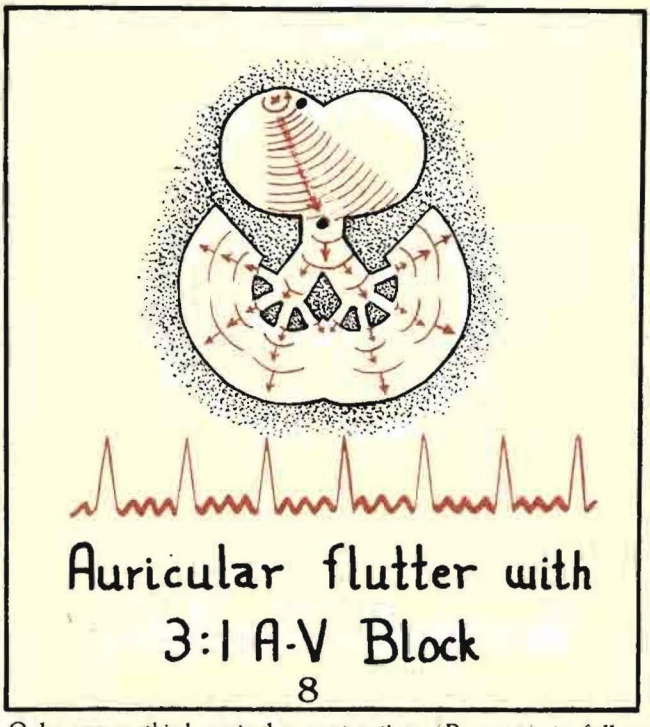
No. 5. — The extra auricular contraction is indicated by an inverted P wave and the subsequent ventricular systole is again followed by a compensatory pause.



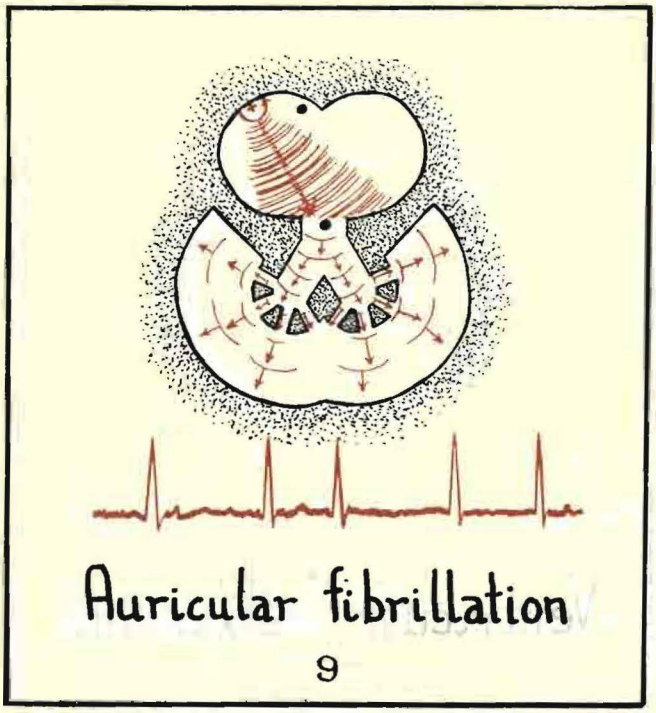
No. 6. — The speed of the heart is increased but each contraction involves both auricles and ventricles.



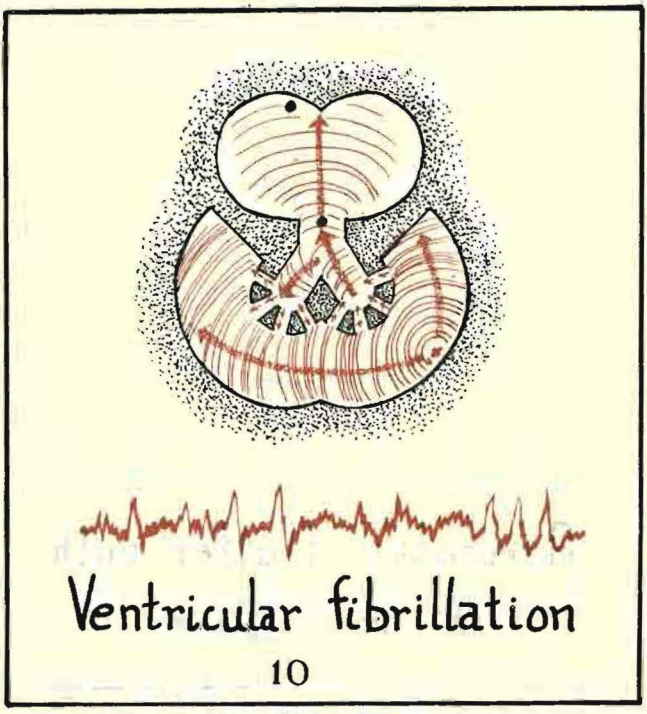
No. 7. — The increased ventricular rhythm is independent of the auricles.



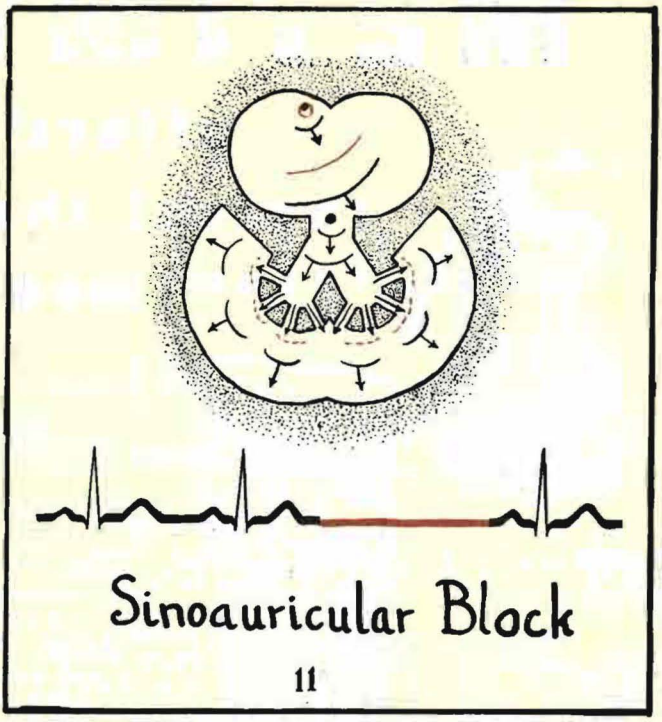
No. 8. — Only every third auricular contraction (P wave) is followed by a ventricular contraction.



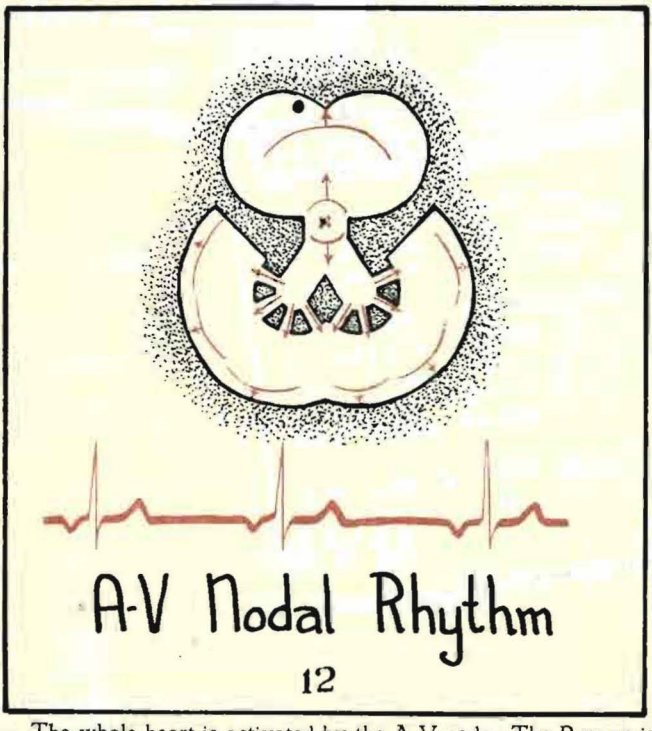
No. 9. — The ventricular rhythm is independent of the auricles due to the refractory period of the ventricular musculature.



No. 10. — The entire rhythm is disorganised.



No. 11. — One complete cycle is omitted due to the failure of the auricles to contract.



No. 12. — The whole heart is activated by the A-V node. The P wave is inverted due to the abnormal direction of the wave of contraction over the auricles.

METIMYD

offers more topical therapy eye, ear,

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 1", 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.

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van inflammasie
Prednisolone Asetaat
5 mgm. (0.5%)

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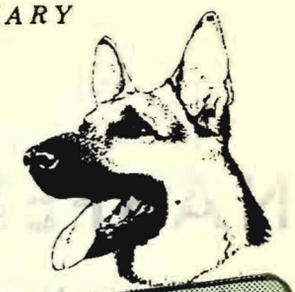
ANTIBAKTERIESE - ANTIBIOTIESE
uitwerking
Natrium Sulfasetamied 100 mgm. (10%)
Neomisien Sulfaat 2.5 mgm. (0.25%)

Ointment with Neomycin
VETERINARY

effective in skin disorders!



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.

Oogontsteking

Edeem

Allergie

Nie - spesifieke

ekseem

Akute en

kroniese otitis externa

an
excellent
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item

METIMYD *Sche*

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JOHANNESBURG

DIE BELANGRIKHEID VAN GENETIESE FAKTORE VAN DIE MIKRO-ORGANISME BY DIE GEBRUIK VAN ANTIBIOTIESE MIDDELS

deur

C. F. B. HOFMEYR

Pretoria

(Voorsittersrede gelewer by die Jaarvergadering van die Afdeling Geneeskunde, Suid-Afrikaanse Akademie vir Wetenskap en Kuns, gehou aan die Mediese Skool, Johannesburg, op Vrydag, 22 Junie 1956)

In hierdie tyd van antibiotiese middels hoor mens soveel van middelbestandheid dat die begeerte hom voordoen om iets meer te weet van die meganisme, wat tot hierdie verskynsel lei. Ek tree dus hier voor u op nóg as bakterioloog nóg as genetikus, maar bloot as iemand wat somtyds graag so 'n bietjie snuffel ten einde effens meer te probeer weet van die hoekom en waarom van dinge wat mens teëkom maar alleen vaagweg verstaan.

Sedert die Oostenrykse monnik, Gregor Mendel, in die sestiger jare van die negentiende eeu sy erflikheidsproewe op pronkertjies uitgevoer het, is daar 'n massa kennis ingesamel oor die genetika. Dié kennis het natuurlik in die eerste instansie betrekking gehad op hoër organismes met die gevolg dat sulke uiteenlopende probleme soos vasstelling van vaderskap, waar dié in die gedrang gekom het, die oorerwing van die Rh-faktor en die verbetering van oeste daarby gebaat het. Die omvang van genetiese navorsing het geleidelik breër geword, totdat betreklik onlangs bevind is dat die erflikheid van eensellige organismes nie in enige wesenlike mate van dié van hoër organismes verskil nie.

Ons kan t.o.v. bakterieë dus ook praat van die *genotipe*, d.w.s. die erflikheidsamestelling, of die som van oordraagbare eienskappe van die indiwidu. Indien omgewingsfaktore enige verandering in die organisme teweegbring, kan hy verworwe eienskappe toon wat *nie* erflik is nie, d.w.s. hy toon die *fenotipe*, bv. bruingebrande witmense, dus 'n fenotipe sal kinders wat wit is voortbring, die genotipe. Daar kan egter in sekere mate verwarring ontstaan, omdat onder sekere toestande die fenotipiese voorkoms van een genotipe dieselfde mag wees as 'n ander genotipe. Om weer die voorbeeld van witmense te noem, 'n bruingebrande witman mag net so lyk soos 'n kleurling. Verder, volgens dieselfde reël, sal 'n bakterium met sweephare bakterieë voortbring met sweephare al is die moedersel se sweephare ook verwyder met fenolbehandeling.

Hoewel, dus, verworwe eienskappe nie oorerflik is nie, is dit ook nie reg om die genotipe as 'n statiese entiteit te beskou nie.

'n Verdere welbekende feit is, dat die indiwidue wat die beste voldoen aan die eise van die omgewing die beste kans het om hulleself voort te plant.

Dit is verder bekend dat fisiologiese en morfologiese eienskappe onder die direkte of indirekte beheer van erflike bepaling staan. Algemeen gesproke, word die potensialiteit om iets te verrig oorgeërf en nie die direkte bekwaamheid nie. Die laaste volg alleen as die eerste benut kan word, bv. die bekwame geneeskundige volg eers nadat hy sy basiese potensialiteite ontwikkel het. Hier dink mens onwillekeurig aan die gelykenis in die Bybel waar verskillende talente gegee is en wat daarmee gedoen is.

U is verder almal bekend met die feit dat sekere kernstrukture verantwoordelik is vir oordrag van die meeste eienskappe. Hulle is nl. die chromosome, bestaande uit nukleoproteïene en hulle tree op as draers van genetiese komplekse, wat die ingebore eienskappe oordra. Daar word vandag geglo dat hierdie chemiese komplekse uit makromolekules bestaan wat in 'n ry op die chromosome gerangskik is.

U is verder vertroud met die verskynsel van mitose waarby daar by selverdeling geen verskil optree in die aantal chromosome nie. Hierdie sel met newestaande *pare* van chromosome word diploïed genoem. Wanneer daar egter by reduksieleiding (soos by meiose) net een chromosoom van elke paar voorkom, is hierdie selle *haploïed*. Met die optrede van vermeerdering van die stelle van chromosome pare in die sel, soos bv. verkry kan word deur sekere chemiese stowwe soos kamfer en kolchisien, word die term poliploïed gebesig.

Wanneer nou 'n paar chromosome langs mekaar lê, lê hulle ooreenkomstige gene ook teenoormekaar. Hoewel die gene dieselfde relatiewe *loci* beklee, is hulle nie noodwendig identies nie, omdat elke ouer een van 'n paar chromosome voorsien. Die effek van ooreenkomstige gene of allelle mag gesamentlik die werking van die twee wees, of een mag dominant wees en een resessief.

Sekere fenomena wat voorkom i.v.m. die verskuiwing van die relatiewe posisies van die gene, hoewel interessant, speel 'n onbelangrike rol in bakteriëe en sal ons dus nie verder besighou nie.

Soos reeds genoem, kan die genotipe nie as 'n heeltemal statiese element beskou word nie en dit is, omdat, volgens die mening van outoriteite, betreklik stabiele veranderinge in die makromolekules van die geen kan optree, d.w.s. 'n *mutasie*. Hierdie veranderinge tree spontaan op, onafhanklik van omgewingsfaktore en met 'n taamlik konstante frekwensie.

In hoër organismes is bevind dat mutasies optree in die verhouding 1×10^{-5} tot 1×10^{-8} per geen per generasie, bv. die mutasie verantwoordelik vir haemofilie kom gewoonlik voor 1×10^{-5}. 'n Verandering tas gewoonlik net een lid van die ooreenkomstige paar gene in 'n diploïede organisme aan. Die meeste mutasies is *resessief* en kan dus nie onmiddellik tot uitdrukking kom nie, omdat hulle oorheers word deur die dominante allelle.

Bakterieë word gewoonlik beskou haploïed te wees. 'n Resessiewe geen kan dus nie onderdruk word deur 'n normale allel nie. Derhalwe sal 'n mutasie spoedig te voorskyn kom.

Dit is egter van belang om te beklemtoon dat daar in haploïede organismes 'n toestand mag optree wat die onderdrukking van resessiewe gene tot gevolg het. Hierdie toestand, wat van groot belang in swamme is en blykbaar ook in bakterieë, verskyn wanneer die kern vinniger kan verdeel as die selle, sodat toestande optree waar een sel veelvuldige kerns het.

'n Dominante mutasie sal in so 'n geval natuurlik nie verhinder word om te voorskyn te kom nie. Die resessiewe mutasie, daarenteen, sal homself alleen kan toon wanneer die selverdeling die kernverdeling ingehaal het. Soos reeds gesê, die meeste mutasies is resessief.

Soms kan die mutasiespoed verhoog word deur sg. *mutagene* soos X-strale, ultravioletbestraling en mosterdgas.

Belangrik is ook dat, waar hierdie mutagene 'n posisieverskuiwing van 'n geen bewerkstellig, die werking van hierdie geen mag verander. Dit lyk dus asof die uitwerking van 'n geen in 'n mate verbonde is aan sy posisie op die chromosoom en dit mag selfs wees dat sekere eienskappe in die indiwidu nie deur afsonderlike nie, maar wel deur die gesamentlike werking van sekere gene oorgedra word. Sommige gene blyk die werking van ander gene te wysig en 'n verdere groep gene kan skynbaar die werking van ander onderdruk.

Dit wil voorkom asof die geen sekere werkings aan die gang sit. Hierdie werking mag talryke stadiums hê voordat die finale eienskap gevorm is. 'n Onderbreking van die effek van die geen op verskillende stadiums kan dus wyduiteenlopende gevolge hê. As voorbeeld kan dit miskien vergelyk word met die verskeidenheid van tekens, wat getoon mag word, wanneer die A. Carotis internus op verskillende hoogtes verstop word.

Die werking van gene is ook in baie opsigte gelykstaande aan die van ensieme. Sommige werkers het reeds aangevoer dat hulle identies is. Dit kan egter met sekerheid gekonstateer word dat gene die tydstip bepaal, wanneer 'n biochemiese proses moet plaasvind.

Tot dusver het ons alleen aandag geskenk aan oorerwing soos bepaal deur onderdele van die kern. Dit is egter bekend dat die sitoplasma van Paramoecium, plante en selfs 'n Drosophila 'n rol mag speel in die oordraging van eienskappe. Aangesien die grootste hoeveelheid van die sitoplasma van moederskant afkomstig is, word hierdie eienskappe alleen van hierdie kant oorgedra. Omdat, egter, dergelike oorerwing nog nie in bakterieë gedemonstreer is nie, sal ons nie langer hierby stilstaan nie.

Organismes, beide die wat geslagtelik en ongeslagtelik voortplant, ondergaan mutasie in 'n groot verskeidenheid van gene. Genetiese studies het verder bewys dat Darwin se beginsels aangaande naturlike seleksie nie foutief was nie. Dit is verder duidelik dat 'n mutasie in een soort omgewing skadelik mag wees

en in 'n ander gunstig. In werklikheid is die organismes van vandag almal voorbeelde van suksesvolle mutasies.

Ons kan dus sê dat die indiwidue van 'n gemeenskap nie verandering ondergaan as gevolg van die omgewing nie, maar dat dié wat die eienskappe het wat die geskikste is vir die omgewing, die beste geleentheid het om die ras voort te plant. Dit volg verder dat sodra organismes met omgewingstoestande waarmee dit nog nie voorheen in aanraking gekom het nie, te kampe het, mutasies wat voorheen van geen waarde was nie, nou van betekenis is.

Wat betref bakteriese erflikheid was die oorspronklike gedagte dat hulle ontwikkel het deur direkte oorerwing van aanpassings. Bakteriologie kan dus beskou word as die laaste vesting van Lamarckisme. Die Franse bioloog Lamarck (1744-1829) was een van die vroegste en bekendste voorstanders van die idee van oordraging van verworwe eienskappe. Dit is maar eers gedurende die laaste 15 jaar of so dat bevind is dat genetiese beginsels van hoër diere en plante ook op mikroörganismes van toepassing is. Deur weinig navorsers is eers besef dat mutasies in werklikheid vir verandering van bakteriese populasie verantwoordelik kan wees.

'n Opvatting wat in die dertiger jare van hierdie eeu baie algemeen was, was dié van siklusontwikkeling van bakterieë, soortgelyk aan dié van protosoë, en dat verskillende fases van 'n siklus hulleself voordoen wanneer toestande geskik is. Daar is toe gepraat van „dissosiasie".

Genetici is nou van mening dat hierdie „dissosiasies" die gevolg van mutasies is en dat moontlik die enigste verskynsel wat aan 'n lewensiklus herinner die van sg. L-vorms is.

Die aanvaarding van 'n erflikheidsmeganisme veronderstel die teenwoordigheid van kerns. Vereers kon sitologiese waarnemings nie sulke strukture in bakterieë toon nie. Verbeterde tegniek het egter wel die herkenning van kerns moontlik gemaak, veral nadat bevind is dat die ribonukleïensuur van die bakteriële sitoplasma die kleurreaksies van die organismes verbloem.

Die getal van kerns mag wissel in die rustende sel. Kokke is gewoonlik eenkernig terwyl basille dikwels meerkernig mag wees. Lg. bevat dikwels twee kerns of veelvoude van twee, veral gedurende tye van aktiewe groei. Blootstelling aan ultravioletbestraling of penisillien mag ook veelkernige toestande in die hand werk, omdat daar vertraging van seldeling mag optree nog voordat kernindeling stadiger gemaak is.

Soos reeds aangetoon, mag die teenwoordigheid van meer as een kern in 'n sel die haploïede toestand verberg. Poliploïede is nog nie in bakterieë opgemerk nie, behalwe reuseselle van Pasteurella pestis wat mag ontwikkel na behandeling met 'n stof soos kamfer.

Mutasies in bakterieë kan voorkom met 'n frekwensie van 1×10^{-4} tot 1×10^{-10} per bakterium per generasie. Mutasie vanaf streptomisiengevoeligheid tot weerstand vir 'n sekere stam van Escherichia coli is bevind te wees 1×10^{-10}.

Dit is belangrik om te beklemtoon dat, hoewel mutasies spontaan optree en afgesien van omgewingsfaktore, dit nie noodwendig volg

dat die reaksies van so 'n organisme dieselfde sal wees in die aanwesigheid van die spesifieke omgewing nie, bv. streptomisien-bestande mutante se metabolisme mag verskil of dit groei in die aanwesigheid of in die afwesigheid van streptomisien. Daarom mag 'n mutasie die sel in staat stel om sekere funksies te vervul of in 'n sekere fenotipe te ontwikkel onder geskikte omgewingstoestande. Hierdie mutasies is besonder stabiel.

Die vraag *waarom* spontane mutasie optree, is nog onopgelos en is seker een van die moeilikste in die genetika.

Wat betref mutante wat bestand is teen antibiotiese middels, tree sommige op as volledig bestand, d.w.s. bestand in een stap, en andere as gedeeltelik bestand, d.w.s. in hierdie gevalle is die bestandheid in die eerste instansie van 'n laer graad.

Onder antibiotiese middels word streptomisien gekenmerk deurdat bestande mutante tot volledige bestandheid in een stap kan verander. Die frekwensie hier is ongeveer 1×10^{-9}.

Dit is interessant en belangrik dat hierdie verskynsel nie t.o.v. ander antibiotiese middels aangetref word nie. Die mutante toon hier net 'n laegraadse weerstand, wat eventueel, deur verdere mutasies, trapsgewyse opgebou word tot 'n volledige weerstand. Aangesien die mutasietempo laag is, is dit dus feitlik buite die kwessie dat twee verskillende grade van weerstand gelyktydig voorkom.

Gestel nou dat die mutasiefrekwensie van eerste trap penisillien bestandheid 1×10^{-6} is. Die tweede trap mutasie kom voor met dieselfde frekwensie as voorheen, m.a.w. een uit 'n miljoen van die trap een bestande organismes kan dus muteer tot trap twee bestandheid. Dit beteken dat trap twee bestandheid alleen kan voorkom in een uit miljoen miljoen organismes, stap drie proporsioneel weer meer ens.

Die verskynsel van kollaterale weerstand is uiters belangrik en kan as volg verklaar word deur:

(1) Die chemiese verwantskap van die gifstowwe.
(2) Soortgelyke werking van hierdie stowwe op die metaboliese prosesse van die sel, in welke geval die bestande sel, deur alternatiewe metaboliese bane, die toksiese werking vryspring.
(3) Die ontwikkeling van nie-spesifieke biologiese verandering soos verminderde permeabiliteit.
(4) 'n Verandering wat die voorafgaande reaksie van verskillende prosesse raak.

Daar is verder aanduidings van antibiotiese weerstand, wat optree *sonder* mutasie deur 'n proses van geleidelike aanpassing na langdurige blootstelling. Hieromtrent is min bekend en, indien dit bevestig word, sal dit waarskynlik bevind word as van sitoplasmiese afkoms te wees.

KLINIESE TOEPASSING

Uit bostaande blyk duidelik dat volledige weerstand teen streptomisien kan voorkom, al word hoë dosisse van die middel van die begin op die pasiënt gebruik (enkele stap bestandheid). Deur

die gebruik van genoegsame hoë dosisse moet verhoed word, dat eerstestadiumbestandheid ontwikkel by organismes wat bestandheid trapsgewyse toon (soos teen penisillien, aureomisien, chloramphenicol ens.). Om te herhaal, veronderstel dat, in 'n organisme met 'n mutasiefrekwensie van 1 x 10^{-6} vir trap een weerstand, die dosis van die antibiotiese middel hoog is, dan sal een uit 'n miljoen organismes oorbly,maar, as gevolg van gebrek aan kompetisie in voedsel ens. word die kiembevolking spoedig opgebou wat nou uitsluitlik bestaan uit indiwidue met trap een bestandheid behalwe die een per miljoen wat trap twee mutant word. Met geleidelike verhoging van die dosis van die middel word die proses herhaal totdat 'n nuwe geslag van hoogbestande organismes geskep is.

Om terug te keer tot die streptomisien — daar is gelukkig geen kruisbestandheid tussen streptomisien en die ander antibiotiese middels soos aureomisien nie. Die toevallige voorkoms van gelyktydige bestandheid in een organisme teen beide middels is dus feitlik uitgesluit. Waar moontlik, sal die gelyktydige gebruik van 'n ander antibiotiese middel met streptomisien van groot waarde wees om die ontwikkeling van n streptomisienvaste kiembevolking te vermy. Dit geld ongelukkig natuurlik alleen vir die bakterieë wat vatbaarheid het vir beide groepe van middels.

In hierdie kort en skematiese praatjie het ek gepoog om iets te skets van die agtergrond van middelvastheid. Mag dit van waarde wees.

SUMMARY

THE IMPORTANCE OF GENETIC FACTORS OF MICRO-ORGANISMS WHEN ANTIBIOTICS ARE USED

DR. C. F. B. HOFMEYR
Pretoria

(Chairman's address delivered at the Annual Meeting of the Division of Medical Sciences, Suid-Afrikaanse Akademie vir Wetenskap en Kuns, held at the Medical School, Johannesburg, on Friday, 22nd Junee, 1956)

It has been found recently that the genetics of lower and unicellular organisms do not differ materially from those of higher organisms.

The genotype cannot be considered as a completely static element, because fairly stable changes can occur in the macro-molecules of the gene, i.e., *mutations*. These changes occur spontaneously irrespective of environmental factors and with a fairly constant frequency. In higher organisms it has been found that mutations occur in the ratio of 1×10^{-5} to 1×10^{-8} per gene per generation, e.g., the mutation responsible for haemophilia occurs at the rate of 1×10^{-5}. A change usually affects only one member of a corresponding pair of genes in a diploid organism. Most mutations are recessive and are not shown immediately. Bacteria are haploid. A recessive gene can thus not be repressed by a normal allell. A mutation thus shows itself early.

In bacteria a condition can occur whereby a recessive gene may be repressed, i.e., associated with the phenomenon of a faster division rate of the nucleus compared with the cell leading to multinucleated cells. The rate of mutation can be heightened by gamma rays, ultraviolet irradiation and mustard gas.

The action of genes may be compared with that of enzymes. Some workers maintain that they are identical. The stage of a biochemical process at which a gene may exert its influence can be responsible for a tremendous difference in the final effect. Compare the varying effects that may be obtained by blocking the Arteria carotis internus at different levels.

A mutation may be to the benefit or disadvantage of the organism, depending on the environment. All organisms of today are in effect the result of successful mutations. *We can thus say that the individual of a society does not mutate as a result of the environment, but that those mutations best suited to the environment are best to ensure the survival of the individual.* Meaningless mutations may suddenly under altered environmental conditions acquire survival value.

As bacteria were first considered to exist through direct transmission of acquired characteristics, bacteriology can be considered

as the last stronghold of Lamarckism. During the nineteen thirties bacterial "dissociation" was regarded as a manifestation of a developmental cycle and not in the light of mutation. Improved technique has made it possible to demonstrate nuclei with their genetic apparatus in bacteria, as the ribonucleic acid of the cytoplasm previously masked the colour reactions of the nucleus.

Mutation in bacteria occurs at the rate of 1×10^{-4} to 1×10^{-10} per bacterium per generation. Mutation from streptomycin sensitivity to resistance in a strain of Escherichia coli was found to be 1×10^{-10}. Even though mutation occurs irrespective of environmental factors, it does not follow that the biological processes will be the same in the presence or absence of the specific environment, e.g., the metabolism of streptomycin resistant strains may differ in the presence or the absence of streptomycin. The problem as to *why* mutations appear as yet awaits solution.

The resistance to antibiotics is of two types, e.g., complete resistance to streptomycin develops in one step, whereas in others like penicillin it develops by degrees for each of which a separate mutation is necessary, while in the former case only one mutation is required. Collateral resistance can be explained as follows:

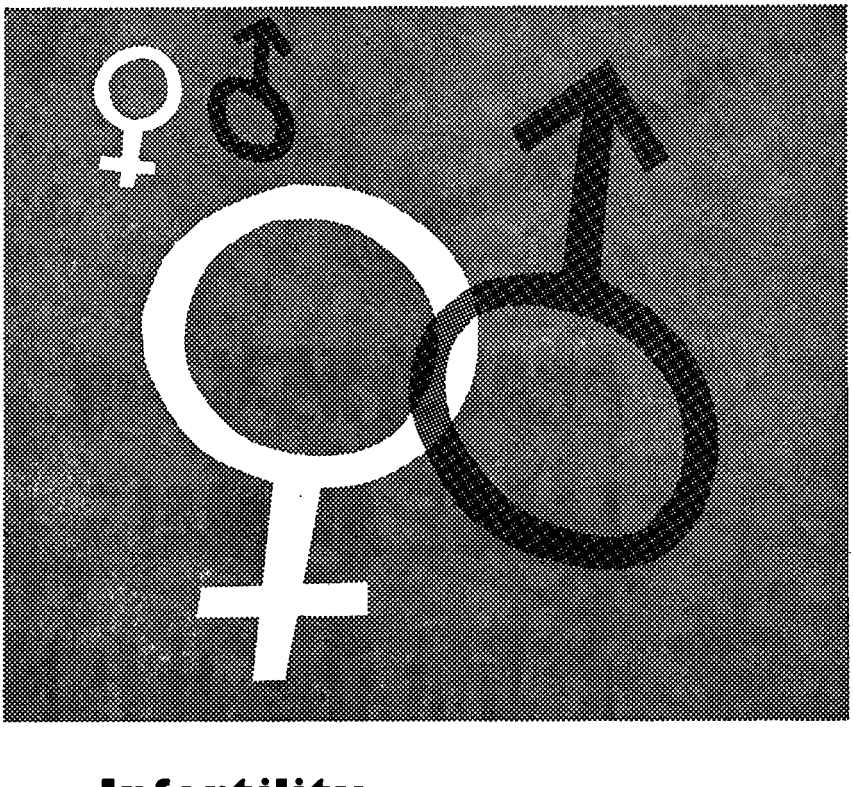
(1) Chemical relationship of the toxic agents.
(2) Similar action of the toxic substances on the metabolic processes of the cell in which case the cell can escape the toxic action of all by developing an alternative metabolic process.
(3) The development of a non-specific biological change like decreased permeability.
(4) A change that touches the preliminary reaction of different processes.

CLINICAL APPLICATION

It is evident from the above that complete resistance to streptomycin is possible even though adequate doses have been used (one stage resistance). In organisms that may show resistance in various stages against certain antibiotics (e.g., penicillin) sufficiently high doses must be used from the beginning. Suppose that in an organism with a mutative frequency of 1×10^{-6} a fairly low dosage of penicillin is used, the one out of a million organisms surviving will, through lack of competition for nutriment, multiply rapidly, so that the surviving organisms are nearly all of stage one resistance. Of these one per million will be of stage two resistance. With inadequate dosage the same story is repeated until finally the bacterial population is fully resistant.

No cross resistance exists between streptomycin and other antibiotics. Simultaneous resistance against both would thus be almost impossible. Where an organism is sensitive to streptomycin and another antibiotic, the simultaneous employment of both is indicated, i.e., care being taken always to employ adequate dosage.

This article has been brief and schematic. May it be of service.



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SOME SIMPLE LABORATORY PROCEDURES FOR THE PRACTITIONER

J. M. M. BROWN

Onderstepoort

Every busy practitioner has at some time or other felt the need for extending his examination of a case presented, to the chemical determination of certain blood or urine constituents to help him arrive at or confirm his diagnosis. We are all familiar with the usual chemical tests carried out on urine, or the usual haematological procedures, but unfortunately in many cases encountered by the practitioner these alone are not enough. In many instances the considerable distances over which blood samples have to be sent for analysis, or the subsequent delay in receiving the report, often make this examination impractical. In other cases the laboratory equipment required is far too expensive for most practitioners or the analytical procedures are too complicated or time consuming. It is to assist the practitioner in this respect that this series of articles is presented. The few analytical procedures which will be described are those which it is felt, are usually required by the average practitioner. All the procedures are extremely easy to carry out, may be done very quickly, the apparatus required is cheap and easily obtainable, and many of the determinations may easily be carried out in the field.

1

The determination of Blood Urea. The method presented here is that of Hench and Aldrich[1] as set out by Bild[2] with a few minor alterations and added comments. This procedure has been used extensively by myself in the field and in the laboratory, and once the practitioner has had sufficient practice in its use, the results obtained compare very favourably with those obtained by using the more complicated colorimetric methods. Its simplicity alone should justify its extensive use by practitioners.

Principle: Mercuric salts combine with urea to form a double salt of urea and mercury. The excess mercury salt is then titrated using sodium carbonate as an indicator.

Apparatus required:

(1) A three inch glass funnel and suitable filter paper.
(2) A micro burette or pipette graduated to 0.1 ml.
(3) A small beaker of about 50 ml. capacity.
(4) A glass flask of about 100 ml. capacity.
(5) A porcelain spot plate or tile.
(6) A few medicine droppers.

Reagents:

(a) 10% Aqueous solution of Trichloracetic acid.
(b) 5% Aqueous solution of mercuric chloride.
(c) Saturated solution of Sodium carbonate.

Method:

(i) To 5 ml. fresh blood add 5 ml. of the trichloracetic acid. Mix well and allow to stand a few minutes for complete protein precipitation.

(ii) Filter. The filtrate should be water clear. If not, refilter the first few drops or use a thicker filter paper. If a centrifuge is available the mixture may be centrifuged instead of filtering; but in practice filtration has been found to be quite adequate.

In large animals where there is no difficulty in obtaining sufficient blood, it has been found more practical to use 10 ml. of blood and 10 ml. of the acid to ensure sufficient filtrate for the test. In the case of small animals one may have to resort to centrifuging if not more than 5 ml. of blood can be withdrawn, to ensure adequate supernatant.

(iii) Fill the micro burette with 5% mercuric chloride. A graduated pipette may be used instead.

(iv) Place 5 ml. of the filtrate or supernatant in the small beaker.

(v) Place numerous drops of the sodium carbonate solution on a porcelain spot plate or tile. Any white surface will do.

(vi) Begin the titration by adding the mercuric chloride drop by drop to the solution in the beaker. After each drop added, mix well and transfer one drop of the mixture to a drop of sodium carbonate on the tile. The end point of the reaction is reached when a brick red precipitate forms within 3 seconds, in the sodium carbonate drop. One very often notices the following sequence of colour changes; curdy white, whitish yellow, yellow and then red, especially when high blood ureas are obtained. It is necessary to go on titrating until the brick red colour just appears. It has been found convenient to run in about 1.5 ml. of the mercuric chloride solution, mixing and then start adding the chloride drop by drop, otherwise the titration may become tedious. Most normal blood ureas lie in the range of 1.7 to 2.5 ml. of the mercuric chloride solution.

(vii) Calculation: (Number of mls. Mercuric chloride x 40) —60=mgm % urea.
Mgm % Urea Nitrogen = mgm % urea x 0.46.

NOTE: The blood of patients receiving sulphonamides may give false positive high reactions, as also will bloods with high uric acid levels. The latter will however not affect the final calculation very much.

Normal urea nitrogen values in mgm% for the various domestic animals are given by Dukes[3] as being:—

Equines: 10-20; Bovines: 6-27; Sheep: 9-20; Dogs: 10-20; Pigs: 8-24.

Allowances should be made for slightly higher values in older animals.

2

The determination of Serum Calcium. This determination will find application particularly in the diagnosis of milk fever in pigs, eclampsia in dogs and the diagnosis of milk fever in cows, especially those atypical or chronic forms. The method used is that of Clark and Collip[4] as cited by Parker[5]. It is extremely simple and gives results in excellent accordance with the more difficult colorimetric methods.

Principle: The serum calcium is precipitated as the insoluble oxalate. It is then determined by titrating the oxalic acid, liberated by acid, with standard permanganate.

Apparatus required:

(1) A centrifuge.
(2) A microburette or graduated pipette (up to 0.01 ml.).
(3) An electric hot plate or bunsen.
(4) A glass beaker of about 50-100 ml. capacity.

Reagents:

(a) $\frac{N}{10}$ — Potassium permanganate

Potassium permanganate 3.5 gms. (accurate).
Water to 1,000 ml. (exactly).

(b) $\frac{N}{100}$ —— Potassium permanganate.

Dilute 10 ml. of the above to 100 ml. with distilled water. Make up fresh from the stock (solution a) for each determination.

(c) Sulphuric Acid (approximately normal).
Concentrated sulphuric acid 28 ml.
Water to 1,000 ml.

N.B.: Add acid carefully to the water, very small quantities at a time.

(d) 4% Ammonium oxalate in water.
(e) 2% Ammonia. (Take 2 ml. of liquor ammon. fort. and make up to 100 ml. with water).

Method:

(i) Pipette into a clean centrifuge tube 2 ml. of fresh serum, 2 ml. of distilled water and 1 ml. of 4% ammonium oxalate. Mix well.
(ii) Allow to stand for at least 30 minutes.
(iii) Centrifuge at 2,000 r.p.m. for about 15 minutes.
(iv) Pour off the supernatant liquid carefully. Allow the tube to drain, and then wipe the mouth of the tube with a piece of filter paper.

(v) By means of a pipette, stir up the precipitate and wash the sides of the tube with about 3-4 mls. of 2% ammonia. Blow it in a fine stream on to the precipitate.

(vi) Centrifuge and drain as before.

(vii) Add 2 ml. of normal sulphuric acid as in (v) above.

(viii) Place in a boiling water bath for about one minute.

(ix) Titrate with $\frac{N}{100}$ Potassium permanganate using a micro-burette or micropipette and carrying out the titration over a white background with the tube partly immersed in a water bath at a temperature of 70-75 degrees C. Add the permanganate drop by drop. The second drop should not be added until the pink colour given by the first has disappeared. Stir after each addition by giving the tube a gentle whipping motion. The end joint of the titration is to be taken as the faintest persisting pink colour that can be recognized when looking down the tube against a white background. The temperature of the water bath should be between 70-75 deg. C at the start and not lower than 60 deg. C at the end of the titration, as too much permanganate may be used.

(x) Calculation:

Number of mls. $\frac{N}{100}$ permanganate X10=mgms.% Calcium.

NOTE: (1) Only distilled water should be used for making up all the reagents. Tap water contains considerable amounts of calcium.

(2) All glass apparatus should be thoroughly cleaned and rinsed with distilled water.

(3) The Blood used for the determination may be clotted whole blood from which the serum has been separated or heparinized blood. Heparin may be used on condition that it is not calcium heparin. Oxalate and citrate should be avoided as anti-coagulants for this determination as they remove all the calcium from the blood.

(4) The blood containers should be washed with distilled water and dried, prior to use.

Normal serum calcium values for the various domestic animals as given by Dukes[3] are:—

Bovines: 9-12; Sheep: 9-12; Pigs: 9-15; Horses: 9-15; Dogs: 9-11. All the values given are in mgm% (mgm. per 100 ml. of serum). I have used this method extensively in the field and laboratory and have found it to be reliable and a valuable aid to diagnosis. It is surprising how many unsuspected cases of hypo-calcaemia one can find by doing the serum calcium determination. I have also found it very useful in following the course of "milk

fever" cases which have a tendency to relapse, and also as a tool in determining whether calcium borogluconate therapy has been adequate in stubborn cases.

(To be continued)

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THE REACTION OF SHEEP TO SUBCUTANEOUS INJECTION OF SULPHURIC ACID IN A SERIES OF SOLUTIONS OF DIFFERENT STRENGTHS

G. D. SUTTON

Onderstepoort

The following experiment was only done because the information was needed for medico-legal purposes. The result is recorded in case it proves of use to someone else.

A series of sulphuric acid solutions was injected under the skin of the inside of the hind leg of sheep to find out what reaction would occur. The strengths and quantities given were as follows:—

$\frac{N}{100}$ — 1 Ml (2 sheep)

$\frac{N}{10}$ — 1 ml (2 sheep)

N — ml (2 sheep)

5N — 1 ml (2 sheep)

9N — 1 ml (2 sheep)

(N solution is approximately a 5% solution by weight.)

The sheep given the $\frac{N}{100}$, $\frac{N}{10}$ and N solutions showed no reaction. There were no visible signs of irritation or discomfort and no reaction developed at the injection site.

The sheep given the 5N and 9N solutions showed discomfort. The leg was either lifted and carried or lifted and stamped. This continued for 1-2 minutes after the injection, after which the sheep started feeding. After 24 hours the skin over the injection site was thickened, oedematous and slightly red in colour. After 48-72 hours the injection site was markedly swollen, oedematous and dark red in colour. The swelling reached a size of 3-5 inches in diameter. After 96 hours a greenish grey area developed in the centre of the swelling, the perimeter remaining swollen, oedematous and dark red in colour. The central greenish grey area persisted for up to 15 days after the injection. It then sloughed away leaving a raw, red crater like wound about an inch in diameter and $\frac{1}{8}$ inch deep with a hard-thickened edge. By this time the swelling and redness had almost disappeared. This wound took from 4-6 weeks to heal and left a puckered scar.

One sheep was slaughtered 8 days after it had been injected. Pathological examination of tissue from the injection site of this sheep showed that the condition which had been caused was a sterile abscess.

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MULTI-BENHEX

Alle bosluise op beeste behalwe die bestande Bloubosluis

'n 10% Gamma BHC poeier vir die dip en spuit van beeste. Maak luise dood en genees skurfte. Maak arseendip doeltreffender vir algemene bosluisbeheer.

SPECIAL BETHEX

Bestande Bloubosluis en alle ander bosluise op beeste

'n BHC en DDT benatbare poeier teen alle bosluise. Voeg net water by. Dood ook luise en bestry skurfte. Aanbeveel vir gebruik sonder arseen teen die bestande Bloubosluis. Ideaal vir spuit van stamboekvee en melkbeeste.

2. VEEDIP-EMULSIES

E 20 VEEDIP-EMULSIE

Bestande Bloubosluis en alle ander bosluise

27½% DDT (20% para para). Ideaal vir spuitdip. Dood vlieë, luise en alle bosluise. Dit verhoog die doeltreffendheid van arseendipmengsels teen geringe koste. Spesiaal aanbeveel as byvoegsel tot Dubble Benhex, Multi Benhex en arseendipmengsel vir bestryding van die bestande Bloubosluis.

3. MENGBARE OLIE

BONT-TOX

Bontbosluise en alle ander bosluise op beeste, behalwe die bestande Bloubosluis

Bevat 75% Toxaphene. Dood bosluise, luise en vlieë; genees skurfte. Bestry die Bontbosluis, draer van die dodelike Hartwater. Maak arseendipmengsels meer effektief vir algemene bosluisbestryding. Moet nie by 'n benatbare poeierdip gegooi word nie.

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PARALYSIS OF THE BRANCHES OF THE NERVUS VAGUS — N. RECURRENS, N. PHARYNGEUS AND N. LARYNGEUS CRANIALIS — AS AN AETIOLOGICAL FACTOR IN "WHISTLING" AND "ROARING" IN HORSES; WITH SOME REMARKS ON ITS HEREDITY AND SURGICAL PROCEDURES IN ITS TREATMENT

J. QUINLAN and D. D. MORTON*

Durban Viljoenskroon

It is generally accepted in horse-breeding countries that hemiplegia laryngis and paraplegia laryngis, as causes of "whistling" and "roaring" in horses, are hereditary. During a long association as a veterinarian, with the breeding of horses, I had always been prepared to accept this view. However, there would appear to be no statistical record, based on research observation, to prove it. Further, I know of no recent research which might end the controversial opinions that have existed between authorities for more than a century.

De Solleysel (1672) was apparently the first to describe "whistling" in equines. According to McQueen (1896) the first researches on recurrent nerve paralysis were made by Prof. Dupuy of Alfort in 1807, Youatt (1833) and John Field (1837). Gunther (Sr.) (1834) described in great detail the pathological conditions of the larynx, associated with "whistling", which he had observed. Gunther apparently was the first worker to observe paralysis of the nervus recurrens following acute febrile disease. Gunther (Jr.) (1866) made notable observations on hemi-plegia laryngis. He described the pathological lesions found in recurrent nerve paralysis with great exactitude. He considered "whistling" and "roaring" to be hereditary. Gunther was also the first worker to perform ventriculectomy as a surgical measure to overcome the resulting dyspnoea.

The literature between 1866 and 1914 has been discussed by Vermeulen (1914). Consequently it is unnecessary to go into further detail in this paper.

Quinlan and Steyn (1939) reviewed the literature, relevant to their paper, then available. The results of observations by research workers up to that date, as to the heredity of vagal nerve branch paralysis, were not conclusive. Vermeulen (1914), Mettam (1899), Haslam (1895), Weiss (1937), Wester (1937) and Argyle (1933/34) suggested various aetiological factors which were not hereditary. These factors are too well known to vete-

* Major D. D. Morton was the officer in charge of the S.A.P. Remount Station at the time these observations were made.

rinarians to necessitate discussion; they are summarised by Vermeulen (1914) and Argyle (1933/34).

That "whistling" and "roaring" were hereditary was the conclusion of those authorities responsible for horse-improvement schemes in most countries: Belgium, Holland, Germany, England, France and Ireland. This conclusion was based on observation without research foundation. It would appear that there was little doubt that heredity was an aetiological factor, either as a direct or predisposing cause, when certain stallions and mares were selected and the history of their progeny analysed. However, one cannot select the progeny of certain unsound horses for analysis and discard others with a similar history of unsoundness.

It is realised that hemiplegia laryngis was considered hereditary by the authorities responsible for most horse-improvement schemes. However, their conclusions were based on observations, not exact research.

In the rules regulating the admission of mares and stallions to these schemes, it was scheduled as a disqualification. In 1889 the Royal Commission on Horse-Breeding took evidence from leading authorities in Britain. Many breeders and veterinarians considered it should be included in the schedule of hereditary diseases. Others, equally well informed, did not subscribe to this view.

The same controversial opinions are heard today whenever hemiplegia laryngis is being discussed. The dogmatism of some of the older observers would appear to be unfounded. Graves (1889) believed it began at the time of mating and nothing could alter it. Williams (1902) challenged this opinion. In support of his view he drew attention to the fact that the great Ormonde, a "roarer", produced some affected progeny in England, but when exported to the U.S.A. he produced sound horses. What is the inference from this? Does it not appear that hemiplegia laryngis is environmental rather than hereditary.

De Roo (1911) is equally emphatic that it is a sequel to infectious disease and is not hereditary.

Certain diseases of the limbs were, and still are, also considered hereditary under horse-improvement schemes: ring bone, side bone, navicular disease, bone spavin and curb. These diseases are partly due to malformation of the limbs and the type of work horses are called upon to perform. The pathological lesion in itself is not hereditary. Moreover, modern research on nutrition has shown the part played by mineral imbalance in the aetiology of bone diseases such as those mentioned.

All zootechnicians are aware that malformations of the limbs and other bony structures may be passed on by parents to their offspring. These malformations, when the limbs are involved, result in certain weakness and consequent inability to stand strain, with resulting pathological lesions: curb, ring bone, spavin, splints, etc.

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* In this paper "whistling" and "roaring" can be considered synonymous. "Grunting" may in most cases, be concurrent with "roaring".

The lesion is not in itself hereditary: It is the malformation, predisposing to it, which is hereditary.

May it not be the same with hemiplegia laryngis? There are certain types of horses that we find frequently affected, those with long, light necks, with lack of width between the mandibles. Perhaps this conformation causes horses to become more susceptible to hemiplegia laryngis as a sequel to certain diseases, especially those of the respiratory tract: pneumonia, influenza, bronchitis, strangles, or, in fact, any acute febrile disease. Any acute febrile condition, which lasts four to five days, may have, as a sequel vagal nerve branch paralysis. There is no doubt that hemiplegia laryngis is a sequel to these conditions in horses which were known to be sound prior to an attack.

It would scarcely be correct to say that such cases were hereditary, although the predisposing factor may be present without being capable of observation.

Have we a precedent in human medicine where we can point out a monoplegia that is hereditary? So far as I am aware we have not.

We have, however, the case of lead poisoning where the musculo-spiral nerve is selected for degeneration in humans. In the horse, Mosselman and Hebrandt (1899) and Gombault (1880-1) describe the left recurrent nerve as the predilection site for degeneration in lead poisoning.

Recurrent nerve paralysis also follows jugular phlebitis caused by faulty intravenous injection of such drugs as gonacrine, and chloral hydrate, and the resulting cicatrisation of the jugular furrow. I have observed three such cases and have operated on two of them.

The cause in this case is involvement of the recurrent nerve in the acute inflammatory process and the resulting cicatrix. It was not a sequel to Babesiases for which the gonacrine was being used, because it was the off side vocal cord that was involved, the side on which the phlebitis developed. .

It may also result as a sequel to Forssell's operation for cribbiting should a severe inflammatory reaction, resulting from infection, follow.

Another cause of dyspnoea in race-horses, which I have no doubt is due to the same aetiological factor as hemiplegia laryngis is paresis or paralysis of the soft palate — palatum molle (Quinlan et al. 1949). The muscles of the soft palate M. palatinus, M. levator veli palatini get their sensory supply from the glossopharyngeal nerve. The motor supply is from the pharyngeal branch of the vagus. The M. tensor veli palatini is supplied by the mandibular branch of the trigeminal nerve. The dyspnoea in this case is produced by the soft palate falling across the glottis. The sound is produced during expiration. It is of a snoring nature. Another factor of great importance is that it is not always present during forced exercise as true "roaring" is. I have observed several

of these cases and previously described the condition and the operation for shortening the soft palate (Quinlan et al 1949).

Paresis of the soft palate does not appear to have been described in equines in Europe. I have requested colleagues, who may have had experience of the condition to communicate with me through the Veterinary Record, but have had no replies.

Another aetiological factor for "roaring" which does not appear to have been described is unilateral or bilateral disturbance of the anterior laryngeal branch of the vagus nerve. Paralysis of this nerve causes relaxation of one or both vocal cords. It would appear that this nerve can be affected without involvement of the recurrent nerve in the same way as the pharyngeal branch. It supplies motor-fibres for the crico-thyroid muscle which tenses the vocal cords. It is not infrequent, on opening the larynx for operation for "roaring", to find bilateral movements, but the vocal cords do not appear to become normally tense. There is some movement in them. The usual, almost static condition of the left vocal cord is not evident as with recurrent nerve paralysis.

Gunther (1866) described hypertrophy of the crico-thyroid muscle, in certain cases of recurrent nerve paralysis, no doubt the result of extra effort of the muscle to relieve the dyspnoea. Vermeulen (1814) describes a similar case.

When I first began observations on "roaring" I was perturbed about the correctness of my diagnoses when I encountered this relaxation of the vocal cords without actual paralysis of the left cord. Now I consider the relaxation to be caused by paralysis of the anterior laryngeal branch of the vagus, without disturbance of function of the intrinsic muscles of the larynx supplied by the recurrent laryngeal nerve. Unfortunately I have been unable to perform autopsies on these cases to establish this suggestion. In any case, difference in the noise made by certain horses, unsound of wind, needs explanation.

The operation of chordo-ventriculectomy is equally effective in "roaring" caused by paralysis of either nerve.

Runciman (1940) and Bogue (1956) state that "roaring" is increasing in this country since the introduction of unsound sires for breeding purposes. These veterinarians, who have spent their long professional lives in close association with thoroughbred horses, have arrived at this conclusion from observations not based on research. I admit research into the problem is most difficult on account of the reluctance of breeders to use unsound horses. However, such an opportunity was made available by Major-General I. P. de Villiers, late Commissioner of Police, at their large stud for remount breeding at Grootdam, near Kimberley. This work was done in co-operation with my colleague, Major D. D. Morton, who was in charge of the stud. Major Morton had an open mind on the heredity of roaring and his co-operation was readily given.

When the experimental observations commenced it was the

intention to breed from sound and unsound sires in a long range experiment. Then when the selected mares reached four years old, they were to be mated again to sound and unsound sires, so that there would have been at least two crosses of sound or unsound blood in the second generation. When the third cross had been reached, further planning would have been considered depending upon whether statistical analysis would prove or disprove the transmission of hemiplegia laryngis and less frequently, paraplegia laryngis from recurrent nerve paralysis, or one of the other aetiological factors discussed above.

Unfortunately before sufficient offspring from the second generation could be bred the stud had to be sold for economic reasons and the fact that the police force had, by that time, become more or less mechanised. This, of course, was in the nature of a tragedy in relation to this observation, because so far as I am aware, no such opportunity had previously been presented to the veterinary profession. However, even when the first generation only could be dealt with, some important conclusions could be drawn.

The observations were initiated in 1942 and breeding continued until 1949, when, for the reasons stated, they had to be discontinued. From the foals bred during this period, 488 were examined. These were mostly geldings. The fillies were, as a rule, not used for police work and consequently, were not available for examination. It was intended that the fillies, after selection, would be returned to the stud for breeding purposes. When the stud was closed, all of them were sold by public auction and became unavailable for further observation.

It is realised that it would have been preferable to have selected stallions without any "roaring" blood to use as controls. However, this was found to be most difficult because there were few stallions, which became available for purchase, without some "roaring" blood in their pedigrees. Consequently five sound stallions were included, as controls, which had a higher saturation of "roaring" blood than was desirable. They should have been excluded, and would have been, had the long range observation been possible to carry out. All these stallions were sound of wind and the nearest "roaring" blood was in No. 13 in generation three, and in Nos. 11, 14, 20 and 21 in generation four; No. 17 had a saturation of 21; 875 all from the sire's side in generations five and six.

These stallions were selected as controls because they were sound of wind and with the exception of No. 13, the closest "roaring" blood was in the 4th generation.

The breeding records of these stallions is interesting: No. 11 had 10 of his progeny tested, one was unsound. No. 13 had 27 of his progeny tested: All were sound. No. 14 had 24 of his progeny tested: There was one unsound. No. 20 had 24 of his

progeny tested: There was one unsound. No. 21 had 35 of his progeny tested: Two were unsound.

It is also realised that the suspected sires could have been more ideal. However, seven were "roarers". Four of them had a saturation of 50 or over. Two were sound of wind with a saturation of 59.375 and 78.125 respectively.

The average saturation in the suspected stallions was 50, and in the controls 12.375.

One important point would seem to be that the control stallions had "roaring" blood close up in their pedigrees, six in generation two, two in generation three, one in generation six only; this stallion produced three "roarers" in 47 of his progeny.

The stallions were thoroughbred, seven imported and 17 South African bred.

Twenty-four stallions were used for service during the period of the observation. Of the control group fifteen were sound of wind. However, 12 had some roaring blood in their predigrees. The saturation of roaring blood in these sires is shown in Table I.

Of the suspected stallions seven were "roarers". Two were sound of wind, but had roaring blood in the second generation on the male side of the pedigree and in the third or fourth generation on the female side. The saturation of "roaring" blood in these sires is shown in Table 2.

TABLE I

Control Sires

Saturation of "Roaring" Blood in Pedigree

Number of Sire and condition of wind	Sire's Sire: Saturation and generation in which "roaring" occurred	Sire's Dam: Saturation and generation in which "roaring" occurred	Total Saturation
10 (S)*	3.125 (g.6)*	3.125 (g.6)	6.25
11 (S)	—	12.5 (g.4)	12.5
12 (S)	(—)	(—)	(—)
13 (S)	3.125 (g.6)	25.0 (g.3): 3.125: 3.124 (g.6)	34.375
14 (S)	12.5 (g.4)	3.125: 3.125: 3.125 (g.6)	21.875
15 (S)	3.125 (g.6)	3.125 (g.6)	6.25
16 (S)	3.125 (g.6): 6.25: 6.25: 6.25 (g.5)	—	21.875
18 (S)	—	6.25 (g.5)	6.25
19 (S)	—	3.125: 3.125 (g.6)	6.25
20 (S)	3.125: 3.125: 3.125 (g.6)	12.5 (g.4): 6.25 (g.5)	25.125
21 (S)	(—)	12.5 (g.4): 6.25 (g.5)	18.75
22 (S)	(—)	(—)	(—)
23 (S)	(—)	(—)	(—)
24 (S)	12.5 (g. 4)	(—)	12.5
Average saturation			12.375

TABLE II

Suspected Sires

Saturation of "Roaring" Blood in Pedigree

Number of Sire and condition of wind	Sire's Sire: Saturation and generation in which "roaring" occurred.	Sire's Dam and generation in which "roaring" occurred	Total Saturation
1 (R)*	12.5 (g.3)	3.125, 3.125, 3.125 (g.96)	21.875
2 (R)	12.5 (g.3)	—	12.5
3 (R)	50.0 (g.2)	25.0 (g.3)	75.0
4 (R)	3.125 (g.6)	(—)	3.125
5 (Sr)	50.0 (g.2)	25.0 (g.3): 3.125 (g.6)	78.125
6 (R)	50.0 (g.2)	25.0 (g.3)	75.0
7 (R)	50.0 (g.2)	25.0 (g.3)	75.0
8 (R)	50.0 (g.2)	(—)	50.0
9 (Sr)	50.0 (g.2)	6.25 (g.5): 3.125 (g.6)	59.375
Average saturation			50.0

Footnote: (S) = Sound.
(Sr) = Sound of wind, but from "roaring" parentage close up in pedigree.
(R) = "Roarer."
(G) = Generation.
(—) = No "roaring" blood.

In analyzing the pedigree for saturation, when in any generation a parent has been found unsound, and this parent was the progeny of an unsound parent in a previous generation, no notice was taken of the latter parent for purposes of estimating saturation.

For the purpose of estimating saturation the sire and dam of any horse is estimated at 50: the grandsire and granddam at 25: the great-grandsire and great-granddam at 12.5: the great-great-grandsire and great-great-granddam at 6.25: the g.g.g.-grandsire and g.g.g.-granddam 3.125: although the sire's blood has been traced to the 7th generation it is not included in the saturation. When more than one figure appears in the same column it indicates that there were an equal number of ancestors in the pedigree affected with "roaring" and the generation in which it occurred.

The progeny of control and suspected sires was analyzed to ascertain if any individual showed a tendency to produce a higher percentage of "roarers" than others. When the progeny of individual sires was in sufficient numbers to draw conclusions there was no significant difference in either. For instance, sire No. 4 produced three roarers from 47; No. 6, three from 28; 8 from 47; No. 2, two from 16; No. 9, one from 14.

Of the control sires, No. 16 produced three "roarers" out of 41; No. 20, one from 24; No. 21, two from 35. Sires 12, progeny 10, No. 22, progeny 5, No. 23, progeny 7, which had no "roaring" blood in six generations, produced no "roarers". Unfortunately these were sires not long in the stud. Consequently the number

of their progeny is insufficient to draw precise conclusions. This was the type of sire originally aimed at. Unfortunately they were purchased too late to be used exclusively in the observation.

The veterinary history of the stallions, prior to purchase, was unknown. Consequently it is not known whether they developed "roaring" subsequent to an acute febrile attack or not. No stallion purchased sound developed "roaring" subsequently. The mares used were a mixed lot. Some were by Clydesdale sires from local mares; others were by thoroughbred sires out of local mares. Our local horses are seldom affected with "whistling" or "roaring". They are notoriously sound and hardy, and ideal for an observation such as this.

The stallions were stabled throughout, being exercised daily in an enclosed yard. The mares and the young horses ran on the veld in the Kimberley district. All the horses were inoculated against horsesickness.

During the breeding season the mares were brought into kraals in the forenoon for testing for oestrus and mating. The colts were gelded at about 18 months. They were stabled at approximately three and a half years. After this age, they were broken in and stabled continuously. They were fed crushed yellow maize, lucerne hay and oat hay. They had, of course, strong daily exercise to fit them for police work.

They were galloped at three and three-quarter years and some as late as five and six years old. Suspicious cases were re-tested after about 3 months.

The dams of unsound horses were tested whenever possible. It is interesting to note that not a single mare was found to be unsound. This is characteristic of local horses under natural environmental conditions.

The veterinary history of the horses was recorded. The only acute febrile disease, which might have an influence on the result of the experiment, that was observed, was strangles. No doubt the young horses became naturally immune to Babesiosis and Nuttalliosis unnoticed, while foals.

One hundred and fifteen horses developed strangles. Of these only five subsequently developed "roaring": Three were the progeny of control sires and two were from suspected sires, quite an insignificant difference.

In the final statistical anylysis, all horses have been included and a further analysis has been made in which all horses that had been sick with strangles were excluded. (Tables 3 and 4).

A most interesting result of the analysis is that recurrent nerve paralysis and strangles were unrelated, i.e. the occurrence of the one did not imply the occurrence of the other. One would have expected a higher percentage of unsound horses amongst those that developed strangles. This was not the case with the cross-bred horses used, under the environmental and nutritional conditions prevailing on the S.A.P. Stud Farm at Grootdam,

Kimberley. The horses were not stabled during the outbreak. Owing to the large numbers of young unhandled horses it was impossible to treat them individually. Only those requiring urgent surgical intervention had individual attention.

The following tables show the results of the examinations.

TABLE 3

ALL HORSES

	Unsound	(%)	Sound	(%)	Total
Suspected	21	(10.71) +	175	(89.29) —	196
Controls	14	(4.8) —	278	(95.2) +	292
Totals	35	(7.2)	453	(92.8)	488

TABLE 4

SICK HORSES OMITTED

	Unsound	(%)	Sound	(%)	Total
Suspected	19	(11.73) +	143	(88.27) —	162
Controls	11	(5.2) —	200	(94.79) +	211
Total	30	(8.0)	343	(92.0)	373

These data show 10.71 per cent of all the progeny of suspected sires were "whistlers" or "roarers": 4.8 per cent of the progeny of control sires were similarly affected.

When horses that had been sick with febrile diseases were excluded 11.73 per cent of the progeny of suspected sires were unsound, while 5.21 per cent of the progeny of control sires were affected.

It will also be observed that febrile disease did not increase the incidence of unsoundness in the progeny of the suspected or the control sires. There was no significance in comparative figures obtained in the analysis of Tables 3 and 4. In fact the similarity of the percentages is striking.

Statistical analysis applied to answer the main question: "Is there any dependence of unsoundness on suspected pedigree" showed that there is a definite dependence of the occurrence of unsoundness, i.e. vagal nerve branch paralysis, on the suspected pedigree in the horses that had no record of sickness and in those that had suffered an attack of strangles.

This proves that the tendency to "whistling" and "roaring" is inherited in the cross-bred horses observed under the nutritional environmental conditions experienced in the Kimberley district. There is little doubt that in a changed nutritional and

climatical environment, experienced outside South Africa, that results would, at least have been similar. This reference is drawn because vagal nerve branch paralysis would appear to be more frequent in the environmental conditions experienced in Europe than in the drier climate of South Africa.

An important point to be noted in these observations is that all the stallions were not entirely suitable, because of the relatively high saturation of "roaring" blood in the pedigrees of some of the control group. It was impossible to plan an ideal observation partly because only three stallions free from "roaring" blood in six generations became available for purchase, and partly for economic reasons.

Surgical treatment of "whistling" and "roaring".

The operation simple ventriculectomy, which was first done by Gunther (Jr.) 1866, and was later adopted by the veterinary profession as the most successful operation for the relief of the dyspnoea caused by vagal nerve branch paralysis, has now been modified into the more radical operation of chordo-ventriculectomy. The vocal cords (labium vocale) are partially excised as well as the mucous membrane of the lateral ventricle (ventriculus lateralis laryngis) and the laryngeal saccule (Sacculus laryngis).

In South Africa the procedure is similar to that described by Quinlan and Steyn (1939) up to the stage of eversion of the ventricular mucosa. At this stage the labium vocale is penetrated at its centre with a small, sharp-pointed scalpel until the point enters the pouch created by the eversion of the mucosa of the laryngeal saccule. Portion of the m. vocalis is included as well as the ligamentum vocale and the plica vocalis. The vocal cord is then severed towards the arytenoid cartilage, and by reversing the scalpel, towards the body of the thyroid cartilage. Care is taken not to expose the cartilages. The severed portion of the vocal cord is then picked up with a vulsellum forceps and detached at either extremity with a small, curved scissors. Care is taken, while the scissors is being used, that the cartilages are not exposed. The mucosa on either side of the laryngotomy incision must be carefully preserved, otherwise there is danger of the resulting cicatrix causing laryngeal stenosis.

The everted mucosa, still held in the forceps, as described by Quinlan and Steyn (1939) is now detached with the curved scissors.

The operation is completed by removing the mucosa of the plica ventricularis with forceps and scissors.

The resulting wound is cleaned of blood clot and dusted with sulphamezathine and penicillin powder (I.C.P.)

The after treatment is similar to that described by Quinlan and Steyn (1939).

The results obtained by this operation in 74 cases have been exellent compared with those obtained by the author (Q) by other methods used:- simple ventriculectomy (Gunther 1866) and

the technique previously employed by Quinlan and Steyn (1939). Almost all the operated cases were sound. There were four cases that still continued to make a harsh inspiratory noise following operation, but two of these won races without distress. One case was not improved. This was a case due to cicatrisation in the jugular furrow caused by a misplaced injection of gonacrine. One case developed peri-laryngeal ossification. The larynx was partially encased in ossified tissue although the laryngeal cartilage did not seem to be ossified. The horse was destroyed for autopsy.

Many of the operated horses have won large sums of money. Three of them turned out to be our best handicap horses, including our highest stakes winner in South Africa.

The operation for shortening of the soft palate (palatum molle) has been fully described by Quinlan, van Rensburg and Starke (1949) and needs no further description here. It is sufficient to say that the operation has been successful in four cases in which it has been performed.

My late partners Faull and Burgess (1956) have operated on 41 horses using the technique described for chordo-ventriculectomy: 39 were sound: two made a noise subsequently. The latter two were reoperated upon successfully. They have also shortened the soft palate successfully in two cases of paresis. They state that in one of these cases the condition may have been congenital.

ACKNOWLEDGEMENT.

The authors wish to express appreciation of the attitude of Major General I. P. de Villiers, late Commissioner of Police, towards the experimental work. He allowed the purchase of "roaring" sires which were to be used at the stud. Besides he gave us every facility to conduct the observations until the stud had to be sold, by placing his staff at Grootdam and Kamfersdam at our disposal. The Director of Veterinary Service is also thanked for allowing the statistical records to be drawn up by his staff.

CONCLUSIONS.

The limitations in exactitude of the observations are realised, as explained in the text. Owing to the long range observation originally planned having to be curtailed these limitations became unavoidable. However, the results prove that there is a hereditary tendency to vagal nerve branch paralysis in horses, the progeny of "roaring" thoroughbred sires, when bred in the nutritional and environment conditions prevailing in the Kimberley district in South Africa.

There was no increase in the incidence of "roaring" following an outbreak of strangles, in affected horses.

The comparison was made between the progeny of sires with a saturation of "roaring" blood estimated at an average of 50, as compared with controls, sound of wind with an average saturation of 12.375.

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COUNCIL MATTERS

THE SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

Minutes of the Council Meeting held on Thursday, 22nd November, 1956, at the Meat Board Building, Pretoria.

Preseent: Drs. P. S. Snyman (President), L. W. v.d. Heever, M. C. Robinson, M. de Lange, E. M. Robinson, R. M. du Toit, A. C. Kirkpatrick, H. P. Steyn, M. C. Lambrechts, S. W. J. van Rensburg, C. F. B. Hofmeyr, and S. van Heerden (Hon. Secretary).

Present by Invitation: Dr. A. M. Diesel.

Apologies: Drs. P. J. du Toit, R. Clark, R. A. Alexander, G. D. Sutton.

The President welcomed Dr. Diesel to the meeting. Drs. H. P. Steyn and M. C. Lambrechts were welcomed back to Council.

II. *Matters arising from Minutes*:—

(a) *Ante and post-mortem inspection of meat.*

Dr. Snyman, the Convenor of the sub-committee nominated by Council, reported that as Dr. Alexander, the Director of Veterinary Services, had undertaken to have informal discussions with the Secretary for Health, in regard to these matters, he did not convene a meeting of the sub-committee. Progress reported.

(b) *Scale of Directive Fees.*

The Secretary reported on the progress made in this matter. It was agreed to appoint a subcommittee to co-ordinate and collate the views of branches, and to submit a schedule of fees and a report thereon to Council at its next meeting.

Drs. H. P. Steyn (Convenor), C. F. B. Hofmeyr and L. W. v.d. Heever were nominated. Agreed.

(c) *Co-operative Employment of Veterinarians.*

The Secretary reported that all members of the Association had been circularised, in accordance with the decision taken at the Annual General Meeting. Members who had not registered at the Congress had been sent a copy of the report as well as the extract of the discussions at the Annual General Meeting.

Dr. Diesel asked that the branches be asked to collate the views of their respective members, as this would facilitate the work of the sub-committee immensely.

Dr. van Rensburg pointed out that it was for Council to decide whether a Special General Meeting should be called to discuss this matter further, or whether it should be held over for discussion at the next Annual General Meeting.

The sub-committee would submit a memorandum on the comments of branches/members for consideration at the next Council meeting.

Dr. van den Heever felt that the sub-committee should also consider the views of members expressed at the Annual General Meeting. After some discussion it was agreed that all members be circularised and requested to re-submit their views to their respective branches.

(d) *Report by Convenor of General Purposes Committee on matters raised in memo. by Dr. Grosskopf.*

The Secretary reported that Dr. Clark together with other members of the General Purposes Committee had arranged to meet representatives of the Pharmacetical Society on December 4th, to discuss matters. — Noted.

(e) No other matters were raised.

III. *Redrafting the Constitution of the S.A.V.M.A.*

(a) The President asked Dr. Diesel to address the Council.

Dr. Diesel said that he had had an informal discussion with the Registrar of Companies, who had given him much information and several suggestions and reasons for redrafting the Constitution.

It was suggested to him that Council obtain the assistance of a Mr. S. Wegger, Charter House, Pretoria, to draft a new constitution.

Dr. Diesel said that his information was that Mr. Wegger's charges would be extremely reasonable. Dr. Diesel felt that the sub-committee charged with this task should seek out all the resolutions from minutes of previous meetings of members, and consider these when re-writing the constitution.

It was agreed that the General Purposes Committee, together with Dr. Diesel, and any of the previous Hon. Secretaries, be charged with this matter.

(b) *Resolution submitted by Natal Branch re Veterinary Internships.*

Dr. Hofmeyr felt that since Faculty matters were rather unsettled, we should postpone investigation of this resolution until a later stage.

Dr. Steyn said that this resolution embodied post-graduate experience and qualifications. To him it appeared as though some members wanted to obtain cheap veterinary assistance from young graduates. Notwithstanding this, however, a sub-committee should be appointed to investigate the matter so as to enable Council to report back to the next Annual General Meeting.

In Dr. du Toit's view, this resolution attempted to remedy the lack of confidence a young graduate had in himself, and also to enrich his human knowledge.

Dr. van Rensburg asked whether we should not advise the Natal branch of the letter obtained from Faculty in reply to a similar matter — although not quite so embracing — raised by Dr. Loveday.

The President said that Council had been entrusted with a task by the Annual General Meeting and that Council would have to give account at the next Annual General Meeting.

After further discussion it was agreed to postpone this matter until the next Council meeting.

(c) *Resolution by Dr. Paine*: "Council to investigate ways and means of making strychnine less freely available to the general public."

The Secretary reported that Dr. Clark had intimated that the General Purposes Committee would also discuss this matter with the Pharmaceutical Society representatives on the 4th of December. Noted and agreed.

IV. *The Professional Provident Society of South Africa.*

The President introduced the matter and indicated that this scheme could be of great value, particularly to private practitioners.

Dr. Steyn proposed that Council accept the conditions as set out in Appendix A. Members should then be advised through the medium of the Journal and be advised to apply direct to the Society for membership.

Dr. Hofmeyr thought that additional measures were necessary to bring the benefits of this Society to the notice of new members, e.g., brochures to be sent annually to the new graduates, or a free advert in the Journal annually.

Council agreed to accept the conditions as set out in the letter dated the 26th September, from the Professional Provident Society of S.A. (Appendix A) and to leave this matter to the General Purposes Committee to work out the details for putting the scheme to members.

V. *Representation of the Veterinary Profession on the A.I. Board.*

After some discussion it was agreed that the matter should not be considered closed by the letter of the Secretary for Agriculture.

It was further agreed that a letter be directed to the Secretary for Agriculture, pointing out that, although, we are cognisant and appreciative of the fact that two veterinarians are members of the Board, it cannot be accepted that these members represent the veterinary profession.

VI. *General Correspondence.*

(a) Letter from Dr. H. P. Steyn re telephone services read.

It was agreed to write to the Postmaster-General, to request that the telephones of veterinarians in practice, be included in the list of telephones which are regarded as essential services.

(b) Letter from Dr. Rossiter read re bars on Presidential ribbon.

It was agreed that Dr. Rossiter's suggestion, that the names of all the past Presidents be inscribed on bars on the ribbon of the Presidential Jewel, be put into effect, and that the Hon. Treasurer be asked to have this work put in hand.

(c) Letter from Dr. Flight read re memorandum of Federated S.P.C.A.s on Bill to amend Laws relating to cruelty to animals.

It was agreed to ask Dr. Flight to obtain 24 copies of the amended memorandum, for distribution to branches and Council members. The General Purposes Committee should then collate the views of branches and Council members and submit these to Dr. V. Shearer and copies thereof to go to Dr. Flight for transmission to the Federation of S.P.C.A.s.

VII. *General.*

(a) Dr. Diesel reported that the address list of members was incomplete and that the Registrar of Veterinarians also did not have an up to date list of current addresses of registered veterinarians.

After some discussion it was agreed that the Editor should once a year, insert a notice in the Journal, drawing attention of members to the provisions of the veterinary Act No. 16 of 1933, relating to notification of change of address. [Section 13 (1) (a).] In the notice members would also be urged to notify the Registrar of Veterinarians forthwith of any change of address.

THE PROFESSIONAL PROVIDENT SOCIETY OF SOUTH AFRICA

The Council of the S.A. Veterinary Medical Association is pleased to advise its members that it has successfully concluded negotiations with the Professional Provident Society of South Africa for the admission of Veterinary Surgeons. A prospectus setting out the benefits offered by the Society is published hereunder, and application forms may be obtained from the Secretary of the Society or the undersigned, on request.

As veterinary surgeons are subject to a greater accident risk in the performance of their professional duties than the members of the other professions currently admitted to membership, the imposition of a small loading has been deemed necessary. This loading has been actuarially assessed and your Society is advised that while it is felt that this loading will be adequate, only experience over a period of years will reveal whether it is adequate or excessive, and that any adjustment necessary will be made in the light of this experience.

While the Balance Sheet of the Society for 1956 is not yet available, it can be reported that the assets of the Society at the end of 1956 were approximately £195,000. The Annual Report and Balance Sheet of the Society will be available to members in due course.

S. VAN HEERDEN,
Hon. Secretary, S.A.V.M.A.

THE PROFESSIONAL PROVIDENT SOCIETY OF SOUTH AFRICA

P.O. Box: 6268
Telephone: 23-1560

1004, Cavendish Chambers,
Jeppe Street,
Johannesburg.

The Society was established by members of the Dental profession some fifteen years ago and is an entirely *mutual* organisation devoted solely to the interests of its members.

MEMBERSHIP

Membership has now been extended to include members of the Dental and Medical Associations of South Africa, the Pharmaceutical Society of South Africa and the Societies of Advocates of South Africa. An applicant for membership must be under 51 years of age and must submit satisfactory evidence of good health in the form of a medical certificate, the cost of which must be borne by the applicant. Applicants of 45 years of age and over are required to submit in addition, recent X-Rays of the chest and a recent E.C.G. tracing.

Although the Society has the right to refuse to admit applicants because of an unsatisfactory medical report, it will in certain instances grant membership subject to a restriction precluding benefits payable in respect of a sickness directly attributable to a specific complaint from which the applicant is suffering or has suffered at the time of admission to membership. Such restriction may be imposed for a limited period, or permanently.

SUBSCRIPTION RATES

An entrance fee of one guinea (£1.1.0d.) is payable on acceptance as a member.

Subscriptions are assessed on an age basis according to the number of shares for which a member subscribes, the maximum being fifty (50) shares. The following are the subscription rates:

Age next birthday at date of purchase of shares	Normal Subscription Rate per Share		Additional subscriptions per share for Veterinary Surgeons	
	Monthly	Yearly	Monthly	Yearly
30 or under	2/9	£1 13 0	2d.	2/–
31 to 35	2/10	£1 14 0	2d.	2/–
36 to 40	3/–	£1 16 0	2d.	2/–
41 to 45	3/3	£1 19 0	2d.	2/–
46 to 50	3/6	£2 2 0	3d.	3/–
51 to 55	3/9	£2 5 0	3d.	3/–

The monthly subscription rate at entry remains unchanged throughout membership. Increases in shareholdings are assessed at the rate applicable to the age at the date of purchase of the additional shares.

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maximum holding of shares will allow sick pay at the rate of £31.5.0d. per week or £135 per month.

Provision is made in the Rules of the Society for an incapacity benefit in the case of permanent incapacity.

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Illnesses do not affect membership, as once accepted, a member is covered for any number of illnesses up to the age of retirement from practice.

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Only a comparatively small proportion of the subscriptions paid is utilised for providing sick pay benefits, the balance being used to secure the provident aspect of membership. The total subscription income of the Society, after deduction of administration and sick pay costs, at the end of each financial year, is apportioned to members on a pro rata basis and placed to the credit of their respective Apportionment Accounts. The amount credited to a member's Apportionment Account is in no way affected by any sick pay benefits received by him during the year.

The average rate of allocation to members' Apportionment Accounts for the past five years has exceeded 2/6d. per share per month, and in 1955 was 2/8.85d. per share per month. For example, on the basis of a 2/6d. dividend rate, a member with the maximum shareholding at 2/9d. per share, pays £82.10.0d. per annum in subscriptions and receives a dividend of £75.0.0d. which means that the sick pay cover has cost him only £7.10.0d. for the year. In the case of a member with the maximum shares at 3/6d. per share, this cover is £30.0.0d. per annum (i.e., £105.0.0d. subscriptions less £75.0.0d. dividend).

Apportionment Accounts earn interest at the average rate of interest earned by the Society's investments which, for a number of years now, has been approximately 5% per annum.

PROVIDENT FUND BENEFITS

The balance standing to the credit of members' Apportionment Accounts is payable on retirement at 60 or 63 years, or in the event of prior death or of retirement from practice. This also applies in the case of permanent departure from the Union. In the event of resignation for reasons other than given above, the balance in the Apportionment Account is paid out but subject to a small penalty.

Although it would be difficult to give any accurate figures of the ultimate value of an Apportionment Account, a rough calculation on the basis of the present figures would suggest that a member holding the full 50 shares would, after 20 years of membership, be entitled to over £2,200, while the figure after 30 years of membership would probably exceed £4,400. These figures are, of course, subject to the present dividend and interest rates being maintained.

TAX PROVISIONS

Subscriptions qualify for the rebate of 1/3d. in the £ allowed for insurance premiums while sick pay benefits and all other payments made to the member are free from taxation.

MANAGEMENT

The management of the Society is vested in the members through a Board of Management appointed at the Annual General Meeting. This Board has at its disposal, the services of a consultant actuary, and the Society also has three trustees who are required to approve all investments which do not fall under the categories of investments required by Government regulations for such Societies.

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INTER AFRICAN BUREAU FOR EPIZOOTIC DISEASES

Mr. W. G. Beaton, Director of the Bureau is to be congratulated on having been made a Companion of the Order of the British Empire.

BOOK REVIEW

The Physiology and Biochemistry of Lactation," by S. J. Folley. Oliver and Boyd, Edinburgh, 153 pages. Price 18/6.

To those interested in the physiology of the mammary gland, the name of S. J. Folley requires no introduction. In this handy sized book the author gives a very concise but full and lucid appraisal of our knowledge in this field. The book is divided into six chapters as follows:—

(1) The development of the mammary gland and the hormonal influences acting thereon.
(2) The initiation of milk secretion (Lactogenesis) and the control of lactation induction at parturition.
(3) The maintenance of milk secretion (Galactopoiesis) with special reference to the interaction of prolactin and somatotrophin, the pituitary-adrenal mechanism and the pituitary-thyroid mechanism.
(4) The physiology of suckling and milking which deals mainly with the milk ejection reflex.
(5) The biosynthesis of milk fat and its hormonal control.
(6) The biosynthesis of lactose and milk proteins.

The approach is fundamental, not applied, but the book can be recommended to all veterinarians who wish to keep abreast of modern knowledge in this vital field of their work. Although much of the experimental work quoted was done on small laboratory animals, the cow, sheep and goat receive considerable attention and much of the information can be applied directly to these animals. Of particular interest to the veterinarian is the demonstration that the mammary gland of ruminants is especially adapted to the synthesis of milk fat from acetate. This again emphasizes the close connection between normal ruminal fermentation and butter fat production.

R. CLARK.

VETERINARY CODE OF ETHICS

Copies of the Veterinary Code of Ethics can be obtained from the Registrar of the Veterinary Board, P.O. Onderstepoort (Mr. van Jaarsveld).

INTERNATIONAL ASSOCIATION OF VETERINARY TROPICAL MEDICINE

This Association has been formed with the object of uniting all persons interested in the study of animal diseases of tropical and equatorial regions. It is affiliated to the International Congress of Veterinary Medicine and may be represented in the National Committees or on the Permanent Committee by two veterinarians, a titular and an adjunct member. The Permanent Committee of the International Congress of Veterinary Medicine has the right of representation at association meetings.

The Association proposes to organize meetings with the object of studying tropical diseases of domestic stock, encouraging research in this domain and to encourage contacts, exchange of information and of material between interested individuals and organizations.

Full details of the rules of the association and the requirements for membership, etc., can be obtained from the Editor, S.A.V.M.A. Journal.

GENOOTSKAP NEDERLAND-SUID-AFRIKA
(Sekretariaat: Posbus 1219, Pretoria)

REGLEMENT

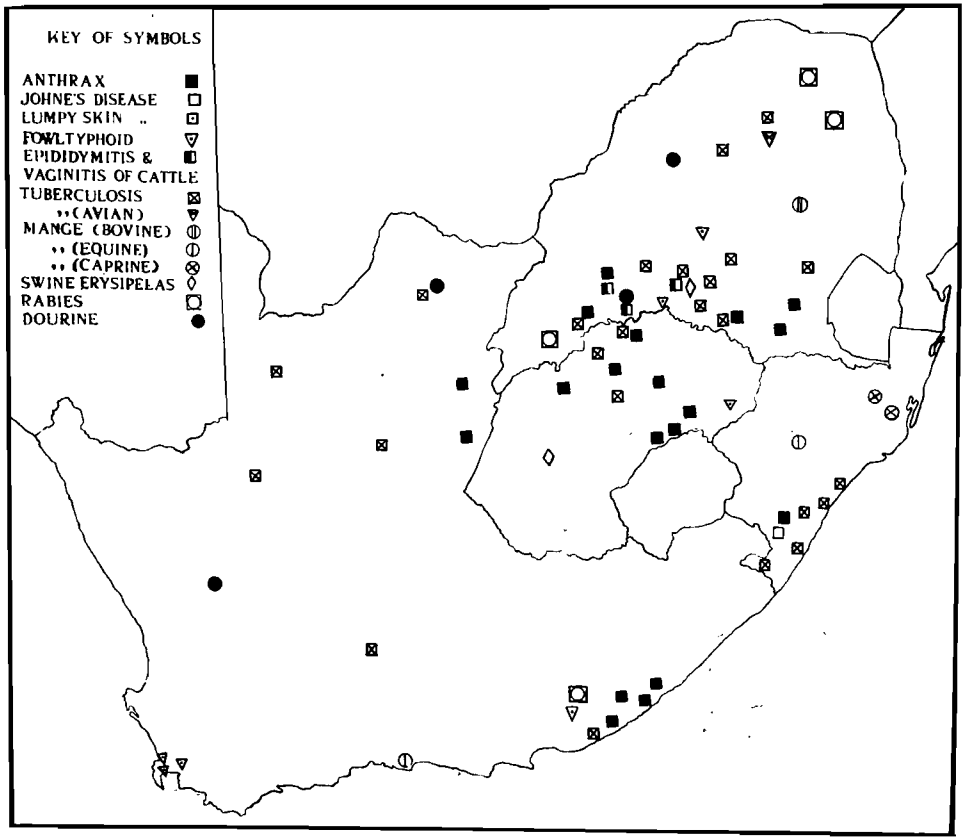
VIR STUDENTE WAT AANSOEK DOEN OM 'N BEURS VAN DIE „STUDIEFONDS VOOR ZUID-AFRIKAANSE STUDENTEN" IN NEDERLAND

1. Die Bestuur van die Studiefonds voor Zuid-Afrikaanse Studenten in Nederland ken op grond van die advies van die plaaslike komitees van die Genootskap Nederland-Suid-Afrika in die verskillende universiteitstede bekragtig deur die Komitee van Toepassing en Advies onder die Kultuurverdrag met Nederland, beurse toe met die oog op studie aan 'n Nederlandse universiteit.
2. Die genoemde beurse bedra fl. 2500 (ongeveer £250) per jaar. Die beurshouer word vrygestel van studiegeld deur die Universiteite van Leiden, Utrecht, Groningen, Amsterdam, Delft en Wageningen. Die studiegeld bedra fl. 300 (ongeveer £30) per jaar.
3. Die beurse is beskikbaar vir studente en oud-studente van 'n doserende Suid-Afrikaanse universiteit, *wat nie ouer as 25 jaar is nie,* wat minstens 3 jaar as student van die betrokke universiteit geregistreer was en wat een of meer van die volgende grade behaal het: M.A., M.Sc., B.D., LL.B., M.Com., M.B.Ch.B., M.Ed., *B.VSc.,* M.Sc. Agric., B.Sc. Ing., en wat beoog om aan 'n Nederlandse universiteit in hulle vak verder te studeer met die doel om 'n graad te behaal of minstens een of ander eksamen te doen.
4. By uitsondering kan ook beurse toegeken word aan studente wat slegs 'n drie- of vierjarige B.A., B.Sc. of B.Com.-graad behaal het, maar alleen in geval hulle met 'n bepaalde omskrewe opdrag van hulle betrokke professor die studie in Nederland gaan voortsit, of ook aan studente wat die ouderdom van 25 jaar *oorskry* het of vir 'n korter periode dan 3 jaar as student van die betrokke universiteit geregistreer was.
5. Aansoeke vir beurse moet by die sekretaris van die plaaslike Komitee van Advies ingedien word voor die 1ste April of die 1ste September. Onmiddellik daarna vergader die Komitee van Advies om oor die aansoeke te oordeel en sy advies te gee.
6. Aansoeke moet in tweevoud ingedien word en moet vermeld:
   a. 'n volledige opgaaf van die akademiese kwalifikasies van die aansoeker en 'n uiteensetting van die loop van sy studies sinds matrikulasie;
   b. sy plan van studie in Nederland met vermelding van die graad wat hy beoog om te behaal, of die eksamen wat hy beoog om te doen, die vermoedelike duur van die studie, die universiteit waar hy wil studeer en, indien moontlik, ook die professor onder wie se leiding hy wil studeer;
   c. besonderhede insake beurse wat alreeds aan hom verleen is of waarvoor hy ook aansoek gedoen het of wil doen;
   d. 'n getuigskrif, in tweevoud, van die dekaan van sy fakulteit of die hoof van sy departement tot ondersteuning van die aansoek.
7. In geval 'n beurshouer nog 'n ander beurs ontvang, is die Komitee van Advies bevoeg om 'n beurs wat alreeds toegeken is, weer in te trek.
8. 'n Beurshouer moet ieder jaar by die Bestuur van die Studiefonds in Nederland 'n verslag indien oor die vordering en die resultate van sy studie, wat goedgekeur is deur die professor onder wie se leiding hy studeer. Goedkeuring blyk uit die handtekening van die professor.
9. 'n Beurshouer is nie bevoeg om sy studieplan te verander sonder goedkeuring van die Bestuur van die Studiefonds nie.
10. 'n Beurs verval sodra die beurshouer sy studieplan voltooi of sy studie staak. Promosie tot die doktorsgraad word beskou as voltooiing van studieplan.
11. Die beurse word in Nederland uitbetaal aan die einde van 'n kwartaal deur die penningmeester van die Bestuur van die Studiefonds. Hulle begin loop op die eerste dag van die maand waarin die beurshouer sy studie in Nederland begin.

12. Beurse word telkens vir die tydperk van *een jaar* toegeken, maar word solank die studie normaal verloop, op versoek van die beurshouer verleng. 'n Versoek om verlenging moet skriftelik voor 1 Mei by die sekretaris van die Studiefonds ingedien word, saam met die genoemde verslag.
13. 'n Beurshouer aan wie in die eerste semester 'n beurs toegeken word, moet uiterlik op 1 Oktober daaropvolgende, en 'n beurshouer aan wie in die tweede semester 'n beurs toegeken word, moet uiterlik op 1 Februarie daaropvolgende sy studie in Nederland begin. Anders verval die beurs.
14. Van 'n beurshouer word verwag om by aankoms in Nederland hom met die Sekretaris van die Bestuur van die Studiefonds, Keizersgracht 141, Amsterdam, in verbinding te stel.
15. Van 'n beurshouer word verwag dat hy onmiddellik nadat hy berig ontvang het van toekenning van 'n beurs, of indien hy daartoe in die geleentheid is reeds daarvoor, hom in verbinding sal stel met die sekretaris van die Raad van die Fakulteit waarin, of die professor onder wie se leiding hy in Nederland wil gaan studeer, om te verneem wat die eise is wat aan hom gestel sal word om tot die beoogde studie toegelaat te word en om vas te stel in hoever hy hom voor sy vertrek uit Suid-Afrika reeds vir die verdere studie in Nederland kan voorberei.

OUTBREAKS OF SCHEDULED DISEASES IN THE UNION OF SOUTH AFRICA DURING THE PERIOD 1/12/1956 TO 1/3/1957

Disease	Province and District (Number of outbreaks in parentheses)			
	Cape	Natal	O.F.S.	Transvaal
Anthrax	(1) Warrenton (6) East London (1) Komgha (2) King William's Town (3) Barkly West (1) Peddie (1) Kimberley	(1) Richmond	(1) Hoopstad (1) Vredefort (3) Kroonsatd (2) Ficksburg (1) Lindley (1) Fouriesburg (2) Bethlehem	(1) Ermelo (1) Amersfoort (1) Klerksdorp (1) Ventersdorp (1) Standerton
Epididymitis and Vaginitis in cattle	—	—	—	(4) Ventersdorp (2) Potchefsrtoom (1) Germiston
Fowl Typhoid	(2) Albany (1) Wynberg (2) Stellenbosch (1) Cape Town	—	(1) Harrismith	(1) Vereengiing (2) Pretoria
Johnes Disease	—	(1) Ixopo	—	—
Lumpy Skin Disease	Fairly widespread over the Union except in Western and South Western Districts were isolated cases have been reported.			
Mange	(1) Knysna (Equine) (Note in the Transkei this disease is endemic in equines and caprines)	(1) Hlabisa (goat) (1) Nongoma (goat) (1) Msinga (equine)	—	(1) Lydenburg (Bovine)
Rabies	(1) Fort Beaufort	—	—	(1) Wolmaransstad (1) Letaba (8) Zoutpansberg
Swine Erysipelas	—	—	(1) Bloemfontein	(1) Brakpan
Trypanosomiasis (Dourine)	(1) Vryburg (1) Calvinia	—	—	(6) Potchefstroom (1) Waterberg
Tuberculosis (Bovine)	(1) Beaufort West (1) Hav (1) Vryburg (1) Kenhardt (2) Gordonia (1) Bathurst	(1) Umzinto (1) Harding (1) Inanda (1) Lower Tugela (1) Camperdown	(1) Vredefort (1) Viljoenskroon (1) Ventersburg (1) Koppies	(2) Delmas (2) Witbank (1) Standerton (2) Potgietersrust (1) Randfontein (1) Germiston (1) Kempton Park (1) Pietersburg (1) Klerksdorp (1) Nigel (1) Carolina (1) Viljoenskroon
Tuberculosis (Avian)	—	—	—	(1) Pietersburg


KEY OF SYMBOLS
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JOHNE'S DISEASE
LUMPY SKIN ..
FOWLTYPHOID
EPIDIDYMITIS &
VAGINITIS OF CATTLE
TUBERCULOSIS
,,(AVIAN)
MANGE (BOVINE)
,, (EQUINE)
,, (CAPRINE)
SWINE ERYSIPELAS
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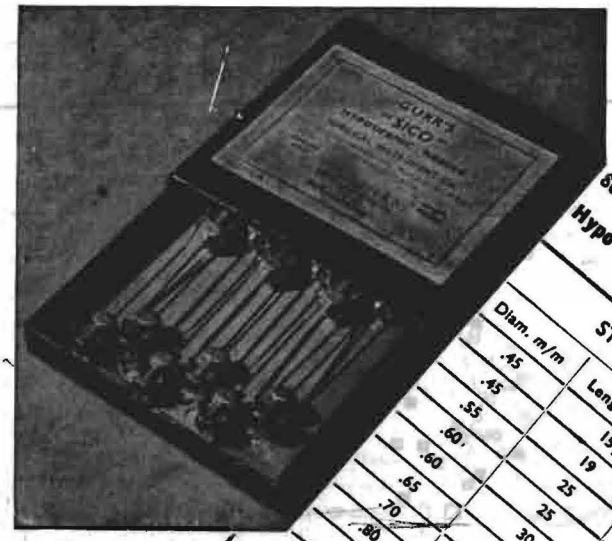

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Jl. S.A.V.M.A. 28 (2), 1957

REFRESHER COURSES IN PHYSIOLOGY

II THE PHYSIOLOGY OF THE LIVER IN HEALTH AND DISEASE WITH REFERENCE TO CLINICAL PATTERNS OF HEPATIC DYSFUNCTION

J. M. M. BROWN
Onderstepoort

Although this paper is intended to serve primarily as an introduction to one on the use of liver function tests in veterinary medicine, to be published later in this journal in the current series of "Some simple laboratory procedures for the Practitioner," it is hoped that this review of the normal and pathological physiology of the liver will also be of some assistance to the practitioner in forming his diagnosis of liver disease in domestic animals, purely from the symptomatology shown by the particular case.

The diagnosis of most of the forms of liver disease encountered in our domestic animals, purely by clinical methods, is at the best of times a very difficult matter. The symptoms exhibited are often very vague and diverse in nature, often appearing to be quite unrelated to the liver, and thus all the more confusing. However, a serious study of the normal functions of the liver and the aberrations produced by various forms of liver dysfunction will assist greatly in correlating these various symptoms, and so serve to build up a picture which will at least suggest the existence of liver lesions.

The liver, besides being the largest gland in the body, is the site of continuous and considerable metabolic activity, and has, therefore, numerous very important functions. It is also an organ which possesses a tremendous reserve and regenerative power and it is usually only when the damage to the liver is severe or extensive that many of its functions become impaired. There are, however, a few conditions such as Tribulosis or early obstructive icterus where only one or more of these functions may be seriously disturbed, without very much apparent injury to the liver cells themselves. In the latter condition, however, severe damage will ensue if the condition persists, and the pathological picture changes accordingly.

Before passing on to a consideration of the functions of the liver, it would be of advantage to review briefly the histological structure of this organ. The liver can be regarded as a modified compound tubular gland with cells of the serous type. It is enclosed in a connective tissue capsule covered by visceral peritoneum. From the capsule proceed connective tissue trabeculae, (ill defined in most of our domestic animals) referred to collectively as Glisson's

capsule, which run between the hepatic lobules. Each of these lobules has roughly the form of a polygonal prism, along the centre of which runs a central vein into which drain numerous intralobular sinusoidal capillaries, the liver sinusoids. The cells lining these spaces are unlike those of the ordinary blood capillaries, in that the outlines of individual cells cannot be distinguished. There are also present amongst these cells larger stellate phagocytic cells, the Kupffer cells, which belong to the reticulo-endothelial system. These cells possess a selective ability to take up particulate matter such as particles of certain dyestuffs or fragments of red blood cells. These particles may be stored in these cells or transferred to the parenchymal cells of the liver and thence be eliminated from the body via the bile, either in the original form or in the form of degradation products (e.g. the bile pigments). These Kupffer cells often become separated from the linings of the sinusoids and appear in the blood stream as macrophages.

The sinusoids run radially within the lobules between cords of hepatic cells and open into the central veins. These in turn coalesce to form the hepatic veins which drain into the posterior vena cava.

The parenchymal cells in keeping with those of most glands have at least one surface in contact with secretory passages, the bile canaliculi, and another to a blood vessel, the sinusoids. These cells are subject to marked changes corresponding to the different functional states of the organ. For instance during and immediately after a meal, they are distended with large fat droplets and stores of glycogen (in the form of granules) and proteins. In the fasting state they are proportionately smaller with only small amounts of fat and glycogen. In conditions of starvation this glycogen reserve may become entirely depleted.

The bile canaliculi drain into the interlobular bile ducts, which in turn unite to form radicles of the hepatic duct.

The functions of the liver are more numerous and complex than those of any other organ and fall roughly under two headings, namely metabolic and excretory activities, of which the former are by far the most important. All these functions may be grouped into five main classes, which are subdivided accordingly, namely:-

1. *Circulatory Functions*:- The liver is intimately concerned in the transfer of blood through a highly complex vascular system from the portal to the systemic circulation. Marked alterations in the hepatic cells will produce disturbances in the blood flow through this organ, being reflected by the appearance of clinical signs such as ascites etc. The liver is also one of the so-called "blood reservoirs" of the body, and the liver, spleen and portal system contain at rest about one quarter to one third of the total blood volume in the dog and cat.

During a severe haemorrhage blood is forced out of the liver and spleen in an attempt by the body to maintain the arterial

blood pressure. A large proportion of the liver's blood content can thus be expelled into the general circulation. This can also be brought about by injections of adrenalin or stimulation of the sympathetic nerves.

The liver capillaries appear to be specially permeable by plasma proteins. By virtue of its Kupffer cells it is also an integral part of the reticulo-endothelial system. The main function of the hepatic artery would appear to be the maintenance of the oxygen tension in the liver.

2. *Secretory Functions*:- The secretion of bile is one of the many functions of the liver and probably not its most important one in the higher animals especially herbivorous animals.

Bile can best be described as a dual purpose secretion containing on the one hand secretory products used for digestion and excretory products such as the bile pigments. It is a viscid greenish or golden coloured liquid with a composition that is complex and varies considerably in the different species. The gallbladder is capable of exerting a marked concentrating effect on bile, water and inorganic salts (especially chlorides, bicarbonate and sodium ions) being reabsorbed.

The secretion of bile is continuous, although the rate varies with conditions like fasting, feeding, etc. Bile is stored in the gallbladder and expelled into the duodenum whenever the products of gastric digestion enter this organ. This action is mediated by a hormone cholecystokinin, which is formed in the duodenal mucosa in the presence of gastric ingesta especially fats. In ruminants, where the passage of ingesta is more or less continuous, the expulsion of bile is presumably also continuous. In equidae the bile passes directly into the intestine.

Bile contains the following characteristic substances; bile salts or bile acids, bile pigments, cholesterol and variable amounts of mucin, lecithin, fats, soaps, urea, inorganic salts and other substances. Bile functions chiefly as a digestive juice by virtue of its bile salts. These function in the following manner:- (a) by activating pancreatic lipase and to a much lesser degree pancreatic amylase (b) by emulsifying fats and so aiding in the direct absorption of a large proportion of the dietary fat as neutral fat. Emulsification also increases the surface area acted upon by the lipase. When bile is not allowed to enter the intestine, fat digestion and absorption is definitely decreased, the faeces develop a very offensive odour, become clay coloured, increased in amount and greasy in appearance. This is naturally of more importance in omnivora and carnivora than in the case of herbivora, (c) bile salts possess the property of forming hydrotropic compounds with many water insoluble products of digestion such as fatty acids, higher alcohols, sterols and fat soluble vitamins e.g. vitamins A, D, E and K. In biliary obstruction these vitamins are not absorbed, and the consequent loss of vitamin K has special clinical significance as we shall see later. In the same way large amounts of dietary

calcium are lost as insoluble calcium soaps. (d) Bile salts have a choleretic action and a mildly laxative effect. A large percentage of the bile salts are reabsorbed from the intestine and are conveyed back to the liver for resecretion into the bile, thus stimulating the flow of more bile. (e) They serve to keep cholesterol in solution in the bile.

Bile is alkaline and by playing a role in the neutralization of the acid gastric juices, assists in maintaining an optimum pH in the small intestine.

The bile salts are usually the sodium salts of the bile acids. There are a number of these, all derivatives of cholic acid, which is allied structurally to cholesterol, the adreno-cortical hormones and the sex hormones. Only a few of the acids are present in the bile of any particular species e.g. taurocholic and glycocholic acids.

The cholesterol present in bile is excess cholesterol which is eliminated via the bile. The sources of biliary cholesterol are (i) synthesis by the liver parenchyma (ii) from the breakdown of red blood cells and (iii) dietary cholesterol, especially in omnivora and carnivora. As cholesterol is often the chief constituent of gallstones, and the bile salts maintain it in solution, it is probable, therefore, that a decrease in bile salts is a factor in the precipitation of cholesterol to form these stones. The inflamed gallbladder moreover is permeable to these salts and thus the lowered concentration creates favourable conditions for the deposition of cholesterol.

In the blood cholesterol may occur in the free form or esterified with fatty acids, whose transport it probably aids. Some 60-70 percent of the serum cholesterol is in the form of these esters. It is well known that the hepatic cells esterify free cholestrol and are responsible for the presence of these esters in the blood.

The bile pigments, biliverdin and bilirubin are also excretion products and impart the typical colour to bile. The amount of each present varies with the species of animal. For instance biliverdin is plentiful in the bile of herbivora and birds and bilirubin is present in the bile of all vertebrates. It is usually the only pigment present in the bile of dogs (Dukes [1]) and predominates in the bile of humans. Under circumstances of starvation biliverdin is also excreted in the bile of dogs. (Winton and Bayliss.[2]).

As these pigments are of considerable clinical significance it will be advantageous to discuss briefly their formation and excretion at this stage. They are derived from Haem, which is the pigment moeity of haemoglobin and myoglobin. It is generally believed that only about one quarter to one third of the bile pigments originate from myoglobin, the majority coming from haemoglobin.

According to recent work the average life span of the erythrocyte is approximately 120 days (Lemburg and Legge [3]). These degenerating red cells then disintegrate and the pigment is taken up by the reticulo-endothelial cells of the bone marrow, spleen and liver, where degradation to bile pigments then commences. Most of these pigments are probably formed in the extra hepatic reticulo-endothelial cells.

Many of the intermediate stages in the degradation of haem to bilirubin are not yet clear, but the process proceeds along the following lines. Haem is a ferroprotoporphyrin IX. A series of oxidative reactions appear to take place at the alpha methene bridge of the haem molecule giving rise to a series of verdohaemochromes. Rupture of the porphyrin ring now occurs at the alpha methene position, the iron is removed and the resulting pigment is biliverdin.

It is a green protein bound pigment at this stage. Reduction now occurs at the alpha methene bridge yielding bilirubin. The bilirubin protein complex, designated "free" bilirubin is now transferred to the hepatic cells by means of the blood circulation. This is the pigment which gives the "indirect" reaction with the van den Bergh test. It is insoluble in water, and various changes now take place in the liver cells rendering it water soluble, (Cole, Billing and Lathe [4], Billing [5, 5a], Schmid [6]) over a wide pH range. The protein molecule is probably split off at this stage. This protein is possibly one of the plasma albumin fractions. The body is unable to excrete "free" bilirubin because of its insolubility. It is normally present in minute amounts in the blood and in large amounts in haemolytic diseases.

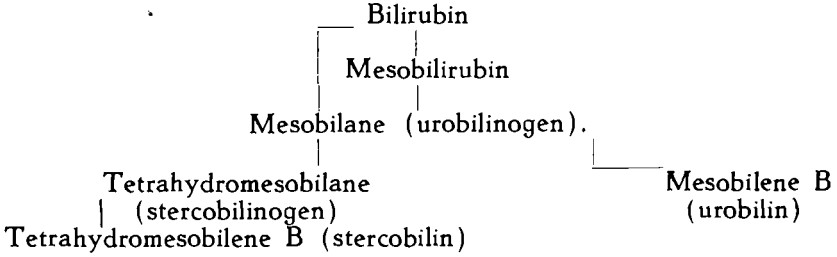
The hepatic cells now convert this into a water soluble pigment by conjugating it, at the alpha-alpha[1] positions with glucuronic acid. First one molecule of glucuronic acid is added yielding a monoglucuronide and then the second molecule follows giving the fully conjugated diglucuronide (Schmid [6], Billing [5a]). These conjugates are referred to collectively as "conjugated" bilirubin and give the "direct" reaction with the van den Bergh test.

They are then excreted in the bile, usually in the diglucuronide form. The kidneys are also able to excrete bilirubin in this form and hence fresh urine will only give a "direct" van den Bergh reaction when bilirubin is present.

The monoglucuronide is more abundant than the fully conjugated form in the urine of human obstructive jaundice cases (Billing [5, 5a]), especially when secondary liver damage supervenes. The diglucuronide is most abundant in normal bile (Billing [5, 5a]).

The exact means by which these pigments are excreted by the hepatic cells into the bile is not clear, but it is most probably not by the process of simple diffusion. It is becoming clear that a complex enzyme system is largely responsible, from work at present in progress on Tribulosis (unpublished).

In the intestines and especially the large intestine, bacterial reduction of bilirubin takes place and the following series of compounds arise:-



The stercobilin then passes out in the faeces, but a small amount of it together with the urobilin is reabsorbed and carried by the portal circulation back to the liver. The stercobilin fraction is converted back to urobilin and most of this is re-excreted in the bile, while the rest is excreted by the kidneys and appears in the urine. Stercobilin and urobilin contribute to a large extent in giving the typical colour to the faeces, and urobilin contributes to a small extent in giving urine its normal colour. In the latter case, however, urochromes play the greatest role as only very small amounts of urobilin are normally present in the urine.

Other biliary excretion products are porphyrins, dyes like Rose Bengal and Bromsulphalein, serum alkaline phosphatase and serum amylase. The dyes are only excreted after administration.

3. *Metabolic Functions*: The metabolism and storage of foodstuffs, their by-products, hormones, enzymes and electrolytes as well as the release of these products in accordance with the requirements of the body, is one of the most important facets of liver activity.

(a) *Water and Electrolyte metabolism*:- The water balance of the tissues depends on the normal functioning of the liver in at least three ways, viz:- (i) by its maintenance of the normal plasma protein levels (ii) by its storage and release of electrolytes, especially potassium, and (iii) by its handling of the pituitary antidiuretic principle (Lederle Bulletin [7]). Water is produced in the liver during the course of physiological oxidation of nutrients.

Besides acting as a storehouse for mineral elements, e.g. iron, it also enters actively into the maintenance of the electrolyte balance of the body.

Potassium is stored and released in a cyclic manner related to the activity of muscles and carbohydrate metabolism.

The potassium level in the blood rises and falls with the lactic acid level and blood sugar concentration (7). It falls after insulin administration and rises after adrenalin has been given (7).

Sodium and chlorine retention have been observed in liver disease, but the significance of this is not clear (7).

Ziady (8) refers to a "pigment specific gravity dissociation" in the urine of patients suffering from various forms of liver disease and noted that when bile pigments appeared in quantity in the urine the specific gravity fell to a low level, which remained fixed while the pigments were present in quantity. He does not, however, attempt to explain this fully. This has been observed during the course of recent experiments conducted on sheep in our present series of research work on bile duct ligation and icterogenin poisoning. (Unpublished) and has always been accompanied by a marked polyuria. Whenever our experimental sheep showed a marked bilirubinuria the specific gravity dropped to a constant low level around about 1.010 until the bile pigments started disappearing from the urine, when the polyuria ceased and the specific gravity returned to normal. It would appear that this is an emergency mechanism whereby the body is able to eliminate excess bile pigment. Whether the liver plays any role in this or not is not yet clear.

It is of interest to note that Bodansky and Bodansky (9) cite references to the occurence of an antidiuretic principle present in the urine of cirrhotic patients.

(b) *Carbohydrate Metabolism*:- The liver is the principal organ concerned with carbohydrate metabolism and the regulation of the blood sugar and carbohydrate level in other tissues. In the non-ruminant, carbohydrates are absorbed as monosaccharides and are conveyed to the liver as such. That portion of these products which is not used by other tissues for energy purposes immediately is converted in the liver to glycogen, in which form carbohydrates are then stored.

When the energy requirements of the body demand it this liver glycogen is then converted to glucose by the process of glycogenolysis and poured into the blood stream. Excess blood glucose can then be reconverted back into liver glycogen or stored in the muscles as muscle glycogen.

During muscle activity part of the lactic acid, which is formed from muscle glycogen, is converted by the liver back into glycogen and then to glucose, which is transferred to the muscles for future activity after being resynthesized once more to glycogen.

Glycogen is a colloidal polysaccharide resembling starch. It is deposited in large amounts in the hepatic cells of all animals, which vary according to the state of activity of the animal. The liver of a normal pregnant ewe contains on the average 3.8% of glycogen, while that of the normal dog contains 3-4% (Dukes [1]).

We see then that the liver serves to maintain the normal levels of blood glucose and muscle glycogen. It is also largely concerned in the process of gluconeogenesis by which glucose can be formed from noncarbohydrate substances such as amino acids, glycerol and possibly fat.

All these reactions take place by means of delicately balanced enzyme systems and stand under regulation of hormones like Adrenalin, Insulin and certain of the adrenocortical steroids. Thyroxin influences metabolic processes as a whole.

In ruminants the majority of the dietary carbohydrates, including cellulose, is fermented in the rumen to the lower fatty acids of which acetic, propionic and butyric are most important. Acetic acid can be utilized directly or used for fat synthesis. Propionic acid is converted to glucose by the liver and apparently forms the main source of liver glycogen and blood sugar. Butyric acid is converted by the ruminal wall and liver to ketone bodies. (Clark and Weiss[10]).

According to Dukes (1) it seems evident that the process of gluconeogenesis is quantitatively much more important than liver glycogen as a source of blood sugar in the absence of dietary sources e.g. in starvation. Gluconeogenesis takes place under practically all normal conditions but varies in magnitude from time to time.

It is of value to note at this stage certain compounds which play an enormous biological role in metabolism and which, therefore, may be used therapeutically in certain forms of liver dysfunction. Thiamine forms an integral part of the co-enzyme cocarboxylase and is essential to a number of enzyme systems particularly those in which pyruvic acid, an intermediate of carbohydrate meta-

bolism, is dehydrogenated. Riboflavine forms part of enzymes known as flavo-proteins, which function in tissue respiration. Nicotinic acid forms part of two very important enzymes namely co-enzymes I and II, which are concerned in tissue respiration and sugar breakdown. Ascorbic acid may act as an important hydrogen carrier and may be concerned in the degradation of haem to bilirubin. Glutathione is an essential part of oxidation reduction systems. Methionine is an important agent concerned in the transference of sulphydryl and methyl groups and is also one of the so-called lipotropic factors.

(c) *Nitrogen Metabolism*: This embraces the metabolism of proteins, amino acids, urea, ammonia, creatine, creatinine, the purines and pyrimidines and is one of the foremost of the liver's functions. Proteins in the normal diet of any animal are digested almost completely in the alimentary tract, amino acids being the final stage in which they are then absorbed. Some of the amino acids in the portal blood pass through the liver for utilization by other tissues, the greater part, however, is removed and undergoes certain important metabolic changes. A certain percentage of these are retained by the liver for synthesis of body proteins but the excess undergoes oxidative deamination. The amino group is split off, enters a complex enzyme cycle and is converted into urea. It is believed that the liver is the only site of urea formation and the chief site of deamination in the body. Urea formation in the liver is dependant on the presence of an enzyme arginase. This does not occur in the liver of birds and in them the final product of deamination is uric acid, which is then eliminated as in the case of urea chiefly through kidneys. Pyridoxine plays an essential role in amino acid metabolism although its precise role is not quite clear.

The non-nitrogenous residue of the amino acids may then be oxidised by the tissues for energy purposes. Certain amino acids give rise to ketonic compounds, others to glucose, which are then both metabolized by the body.

The normal body is in a very delicate state of nitrogen balance, in which the amount of nitrogen lost in a twenty four hour period is approximately the same as that consumed. The liver is a very important agent in maintaining this balance, by virtue of the fact that it is concerned with the synthesis of plasma proteins and is also a valuable pool of protein and amino acids on which the body can draw in times of need.

The liver is the chief site of formation and storage of the plasma proteins. Some of the globulin fractions may originate extrahepatically. The plasma protein concentration and the ratio of the different proteins to each other remains remarkably constant. Serious disturbances of hepatic function will upset the equilibrium of the circulating plasma proteins and will also then naturally upset the colloidal osmotic activity of the blood. Albumin accounts for almost 80% of this osmotic activity. Depletion of hepatic protein reserves affects the production of albumin more than that of globulin and we usually encounter a reversal of the albulin globulin ratio.

The liver is also the site of prothrombin formation in the body. Serious hepatic dysfunction causes marked alterations in the clotting time of the blood. Vitamin K is a prerequisite for the formation

of prothrombin and it will be remembered that this vitamin is fat soluble. Hence any interference with bile secretion will interfere with vitamin K absorption. As has already been indicated this is also true for vitamins A, D and E. Severe bony changes may thus follow on prolonged obstructive liver disease.

It is of interest to note that neither deamination nor urea formation is noticeably affected until almost 90% of the liver has been destroyed.

The liver and kidneys are the two chief organs responsible for the production of creatine.

The purines and pyrimidines are products of the digestion of nucleoproteins in the intestines or may occur in other forms in the normal diet. Under the influence of various enzymes in the liver the purines are converted to uric acid. The livers of all animals except man and the higher apes contain an enzyme uricase which converts uric acid to allantoin. Uricase is also present in the bovine kidney. We therefore find that the end product of purine metabolism appearing in the urine of man is uric acid, while in our domestic animals it is allantoin. In cattle the average percentage of allantoin in the urine is 92.1 compared with the percentage distribution of 7.3 for uric acid, while in man the ratio is 2.0:90.0 (Dukes [1]). The remaining percentage in both cases is made up of other purines. Uric acid is a constant constituent of blood, but in most mammals other than man the amount is small. For example the range in the blood of man is 0.7-3.7 mgm. per 100 ml. of blood, while in bovines it is 0.05-0.2 mgm. per 100 ml. (Folin and Denis[11]).

The only known site of uric acid conversion in dogs is believed to be the liver, therefore in hepatic dysfunction in these animals, one would expect the blood uric acid level to rise and the allantoin level to fall, It is interesting to note that in the Dalmatian only about one third of the uric acid formed is converted to allantoin, whereas in other dogs the conversion is almost complete.

Very little is known about the fate of the pyrimidines in the body.

(d) *Lipid Metabolism*:- Fat is the form in which the body can store the greatest amount of reserve energy. It will be remembered that the liver is the chief site of the formation of cholesterol esters with fatty acids and probably also the main site of phospholipid formation, both processes assisting in the transport of lipid derivatives in the body. A large proportion of the absorbed fat is conveyed to the liver for temporary storage. A very large part of it is then transformed into phospholipid in which form it is then conveyed to the various tissues.

If this conversion of fat to phospholipid is prevented at all there will be a deposition of fat in the liver. For the synthesis of lecithin, one of the main phospholipids involved, choline is required. Choline is one of the so-called lipotropic factors. Normally the quantity of fat in the liver is fairly constant. An accumulation of excess fat in this organ is indicative of an alteration in fat metabolism. The production of "fatty livers" is generally due either to a failure of fat transport from the liver or to an increased mobili-

zation of fat from the depots at such a rate that the liver cannot cope with it.

Two chief types of fatty livers are encountered namely fatty degenerations and fatty infiltrations. The former is due to physical changes in the hepatic cells and is not necessarily an increase in the total fat content of these cells, whereas in the latter there is an increase in intracellular fat. Substances capable of preventing or curing fatty infiltration of the liver are designated lipotropic factors. A number of these are known namely; choline, which is today classed as a member of the vitamin B complex, manganese, which seems to be necessary in traces and has some interaction with choline, methionine, betaine, triethylcholine and ethanolamine. These substances all owe their lipotropic activity presumably to the fact that they are precursors of choline. Certain proteins which have a high methionine content, for example casein, may also be regarded as being lipotropic. Other lipotropic factors whose precise mode of action are not yet clear are lipocaiac (a substance found in the pancreas) and inositol.

It is of interest to note that diets which produce fatty livers fail to do so unless thiamine is present (Dukes [1]). Deficiencies of pyridoxine and thiamine are associated with a decreased fat synthesis. An excess of these vitamins increases fat synthesis and deposition of fat in the liver, especially if choline is deficient at the same time. It is, therefore, very essential to give a balanced vitamin regimen when treating hepatic disease. Pantothenic acid is also essential for fat metabolism but its precise role is still obscure. The work of Fischer et al. (12) on lipotropic liver support therapy in schizophrenia, illustrates very well the value of a balanced vitamin regimen in the treatment of liver disorders. The liver is also concerned with the synthesis of fatty acids from carbohydrates, amino acids and proteins and with interconversions of one fatty acid to another. The latter process occurs during the so-called beta oxidation of fatty acids. It is also the sole site of ketone body formation from fatty acids in non-ruminants, although these are probably mainly utilized elsewhere in the body.

(e) *Mineral Metabolism*:- The liver is concerned with the fixation, transformation and utilization of iron, although it plays a secondary role to the spleen in this respect. Iron is stored chiefly in the liver, spleen and bone marrow. Copper, manganese, zinc and probably cobalt are stored in the liver. The liver is also able to store certain toxic ions such as lead, arsenic and probably also antimony and thallium until they reach a concentration toxic for the liver or the organism.

(f) *Vitamin and Hormone Metabolism*:- Certain of the vitamins and hormones connected with the metabolic processes in the liver have already been discussed. There are still a few others which require some comment.

Vitamins A and D are stored in the liver and it is the largest storehouse in the body for vitamin C. Damage to the liver may result in inadequate storage capacity, or failure of the liver to secrete bile may result in disturbed absorption of A and D. It may also then fail to metabolize these vitamins when it needs them most.

The liver is responsible for the conversion of carotenes to vitamin A and for the destruction of excess vitamin A and carotenes.

Vitamin B 12 and Folic acid are both stored in the liver and consequently severe prolonged liver disease will result in decreased storage and utilization of these factors, and anaemic changes.

The liver is concerned with the destruction of oestrogens or their conjugation with glucuronic acid prior to elimination via the urine. In females the uterus and ovaries also play a role in this respect. It is of interest to note that in human cases of certain forms of liver disease of long standing gynaecomastia is often seen indicating deficient destruction or elimination of these oestrogens. The liver is probably also concerned in the conjugation or alteration of other steroidal hormones prior to elimination. It would appear that there is a close connection between liver function, adrenal function and activity of the gonads. Coodley and Molle[13]).

(g) *Enzyme Relationships*:- Serum amylase has already been mentioned and although this enzyme is produced primarily in the pancreas and salivary glands work by Cajori and Vars (14) indicates clearly that some of it originates in the liver as well. They report a marked fall in the serum amylase following experimentally induced severe liver damage. Somogyi (15, 15a) found low serum amylase in clinical cases of liver injury such as hepatitis, cirrhosis and liver carcinoma. It is also reported to be low in liver abscesses and liver damage due to chloroform poisoning. (Parker[16]). Rachmilewitz (17) also reported low serum amylase in hepatic disease, as did Ravdin, Vars and Goldschmidt (18). Increased serum amylase values are found in common bile duct obstruction (Parker 16).

Serum alkaline phosphatase is excreted to a large degree by the liver and increased values are found in diseases of the liver and biliary tract, especially in obstructive icterus. The hepatic cell is involved in this activity in contrast to the excretion of dyes in which the reticulo-endothelial cell is the functioning element, thus alkaline phophatase will accumulate in the serum following bile duct obstruction or from liver cell injury especially if the hepatic damage is caused by a toxic agent rather than by infection.

(h) *Porphyrin Metabolism*:- The role of the liver in porphyrin metabolism is not yet clear. It is apparently concerned in the interconversion of porphyrins. Dobriner and Rhoads (19) give an interesting and extensive review of porphyrins in liver disease. Quin and co-workers (20) found that Phylloerythrin, a porphyrin derivative of chlorophyll, is normally excreted in fair quantities in the bile of ruminants. Interference with phylloerythrin excretion, as in the case of Tribulosis, leads to marked photosensitization. Therefore we may expect to find photosensitization in all cases of liver damage in ruminants where excretion of this porphyrin is apparently interferred with. This has been observed in certain forms of liver damage in calves brought into Onderstepoort for treatment (unpublished).

(1) *Heat Production by the Liver.* The liver is second to the muscles in producing some of the body's heat, by virtue of its

active metabolic processes and large size. It, therefore, plays a role in the heat regulating mechanism of the body.

4. *Protective Functions*:- An important aspect of the activities of the liver is the mechanism by which it is able to protect the body from certain injurious agents. This is accomplished by:-

(a) Scavenger action; as we have seen, the Kupffer cells possess marked phagocytic activity removing foreign particles e.g. colloidal dye stuffs from the blood. Many of these substances are then passed on to the liver cells to be ultimately excreted via the bile into the intestine. Certain liver function tests are based on this ability of the liver to excrete certain dyes e.g. Bromsulphalein or Rose bengal. These dyes are injected into the bloodstream and their rate of disappearance over a selected time is noted, retention being indicative of hepatic disease.

(b) *Resistance to infections*: the reticulo endothelial cells are also believed to be engaged in the production of antibody globulins.

(c) *Detoxication*: toxic substances reaching the liver, provided that they do not do so in quantities sufficient to cause rapid and overwhelming damage to the hepatic cells, are handled in such a manner as to render them harmless and they are then usually eliminated via the urine. This is accomplished by (i) oxidation or reduction of the compound, as in the case of cincophen, or (ii) acetylation as occurs with the sulphonamides or (iii) conjugation with glycine, glucuronic acid or sulphates, as in the case of benzoic acid (Hippuric acid being formed) aspirin or sex hormones (glucuronates being formed) and Indole (Indican resulting) respectively. Conjugation is probably by far the most important of these mechanisms.

The ability of the liver to conjugate and excrete substances like aspirin or camphor, in the form of glucuronates is used as a liver function test. In cattle and horses there is normally a considerable amount of glucuronic acid present in the urine and its absence for any reasonable length of time may be taken as evidence of liver dysfunction. (Boddie [21]).

5. *Blood Coagulation*: Protection of the body from excessive loss of blood is another function of the liver by virtue of its production of prothrombin and fibrinogen. The liver also contains as a balancing factor large amounts of heparin which is produced by the mast cells of the body.

6. *Haemopoietic Functions*:- The role of the liver in erythrocyte formation is probably one of its most vital functions. In the foetus it is one of the organs concerned with red blood cell formation, while in post-natal life it serves as a store house of iron, vitamin B 12, folic acid, copper and cobalt, all of which are necessary for the maturation of the erythrocyte and haemoglobin formation.

Having now considered the normal physiology of the liver, it is expedient to review briefly at this stage the general pathogenesis of liver disease, before discussing the pathological physiology encountered in liver dysfunction.

The first consideration in the pathogenesis of liver disease is damage to the parenchymal cells. These being very specialised cells are easily affected by toxic agents or circulatory interference,

much more so than the cells of the biliary system, sinusoids or the Kupffer cells. The parenchymal cells may be affected by circulatory factors such as inadequate blood flow, excessive accumulation of venous blood, as would occur in chronic heart disease, and a low oxygen tension in the blood, or by some toxic agent either damaging the cells directly or interfering with the utilization of nutrients by these cells.

The degree of damage produced is not only dependant on the causal agent or factor, but also on the position of the cells concerned, in the lobule. For instance one of the first results of damage to the liver is swelling of the cells, circulation being impeded and the parenchymal cells furthest from their blood supply suffer first, or as in the case of passive venous congestion those around the central veins are affected first.

The liver cells might also be affected indirectly by certain conditions like contiguous neoplasms or abscesses, which may exert pressure on the area of the liver concerned as well as being the source of circulating toxic products, or cholecystitis and obstruction of the bile duct.

The parenchymal cells will respond firstly to any of these damaging influences by simple cloudy swelling, which may or may not be associated with fatty changes. The changes which then follow depend on the causal agent, its severity and duration of action and whether it is removed or not. They may be severe fatty changes, necrosis and cirrhosis or regeneration and complete recovery. Severe cytotoxic agents may cause extensive and permanent damage from the start and cause the death of the organism within a short space of time.

Repair of liver damage may take place in two ways viz. by replacement of the necrotic cell masses with fibrous tissue or regeneration and hyperplasia of the surviving intact liver tissue. The two types of change may occur concurrently, the severity of the damage determining which will predominate and to what degree the blood circulation is restored.

Cirrhotic changes may develop either as a fine network of fibrous tissue diffused throughout the liver or as coarser cords of fibrous tissue separating nodules of regenerating parenchymal cells. The former results from a more chronic and widespread type of injury, the latter from a more acute and severe form causing patches of necrosis.

In this country extensive damage to the liver cells may be brought about by helminth parasites such as fasciola hepatica or cysticercus tenuicollis (hepatitis cysticercosa), especially in cattle and sheep, purely by mechanical irritation to the bile ducts or in the case of the latter, burrowing into the hepatic tissue.

Let us now consider the pathological physiology seen in liver dysfunction and the clinical signs or biochemical changes which one would expect to encounter in these conditions. These vary widely with the liver condition concerned and also the degree of involvement of the liver in these conditions. We must bear in mind the great natural hepatic reserve and the enormous regenerative ability of the organ and remember that while certain functions

may be impaired with moderate liver damage, the majority are only involved when the liver is seriously injured. In the discussion which now follows we will consider liver dysfunction as a whole including thus all the changes seen in minor, moderate and severe grades of insufficiency to enable us to see a clear and complete picture of the pathological physiology.

The volume of the blood in the liver may or may not be increased in inflammatory conditions involving it. It is increased in chronic venous congestion and decreased in cirrhosis, cloudy swelling and so on.

Both the secretory and excretory functions will be affected to a marked degree and interference with these functions produces some of the symptoms so suggestive of liver involvement. Dysfunction of the hepatic cells causes a failure or decrease in the synthesis of the bile salts and swelling of these cells will interfere with bile secretion. There arises thus a deficiency of bile in the intestine and certain characteristic digestive disturbances appear. Pancreatic amylase and lipase are not sufficiently activated, the emulsification and absorption of fats and fat soluble compounds is hindered, the pH of the small intestine is upset and conditions may become unfavourable for some of the other digestive enzymes. Constipation tends to develop and the faeces are clay coloured, increased in amount, greasy in appearance and have a very offensive odour. In long standing cases of liver dysfunction, bone anomalies tend to develop because of impairment with the absorption of vitamin D, calcium and phosphorus. Because of the digestive disturbances, intestinal putrefaction tends to occur, flatulence develops and the breath has a foetid odour, the so-called "fetor hepaticus." Diarrhoea may then also develop. There is a rise in the bile salt concentration in the urine as these "dam back" into the blood stream and start to appear in the urine in increased amounts.

There arises a failure on the part of the liver to form cholesterol esters, so the level of these compounds in the blood may fall, although the total cholestrol level may rise, due to deficient excretion through the bile of cholesterol. The transport of fatty acids is thus impaired. The synthesis of bile acids from cholesterol is hindered and a deficiency of these may then arise. The liver may also be unable to store cholesterol.

The formation and excretion of bile pigments is also seriously interfered with. Bile pigment in the form of free bilirubin is conveyed from the reticulo-endothelial cells to the liver parenchyma for excretion. The liver cells may be unable to conjugate this or only conjugate it to a certain extent depending on the severity of the damage. Thus free bilirubin accumulates in the blood stream and giving the indirect van den Bergh reaction, when this test is applied to the serum. Owing to swelling of the cells or impairment of the excretory mechanism, what conjugated pigment has been formed may then also accumulate in the blood stream giving a positive direct van den Bergh reaction as well as the indirect reaction. Whichever pigment is present clinical icterus will develop. This then is icterus of hepatogenous origen, or hepatocellular icterus. Under these conditions conjugated bilirubin is then excreted in the urine and a bilirubinuria becomes apparent.

Owing to the impairment of bile pigment excretion the amount of stercobilinogen in the faeces gradually decreases in proportion to the severity of the condition and the faeces may then become completely acholic giving them their typical clay-like colour. Thus also the amount of urobilinogen in the urine decreases and absence of urobilinogen from the urine may indicate complete biliary obstruction.

It is expedient at this stage to note two other types of icterus, namely obstructive or regurgitation icterus and haemolytic icterus. In the former regurgitation of bile pigment into the blood stream occurs as a result of complete or partial obstruction of the bile duct. As this pigment has already been conjugated by the hepatic cells the blood serum will, in the initial stages of the condition, give the direct van den Bergh reaction. As the condition progresses secondary damage to the liver cells occurs and free bilirubin starts to appear in the blood in addition to the conjugated pigments. The van den Bergh test will, therefore, give us both types of reactions with the direct reaction predominating until the conjugating mechanism breaks down entirely.

In the case of haemolytic icterus the rate of red cell breakdown is so rapid that the liver cells are unable to keep pace and "free" bilirubin accumulates in the blood. If secondary hepatic damage now also occurs, swelling of the cells may impede excretion of the conjugated pigment and this will start to accumulate in the blood as well, and a bilirubinuria then also appears. The van den Bergh test will then give first the indirect reaction and then a mixture of both with the indirect reaction predominating. In this case the urine will also contain increased amounts of urobilinogen owing to the increased amount of bile pigment being poured into the intestine and urobilinogen increasing in the blood faster than the liver can rehandle it for excretion. The faeces then also contain increased amounts of stercobilinogen. If secondary hepatic damage now supervenes the amount of urinary urobilinogen may then decrease as also the amount of faecal sterobilinogen. It is of interest to note that even in complete biliary obstruction small amounts of sterobilinogen may later appear in the stools. This is due to the fact that bilirubin being a "tissue invader" may accumulate in the cells of the intestinal mucosa and these cells may be in state to excrete it into the intestine, where bacterial action will convert it to sterobilinogen. It is the tendency at present to group both haemolytic icterus and hepatogenous icterus under the term "retention icteru". In both cases this term should strictly only apply when "free" bilirubin is the predominant pigment.

Marked interference with dye excretion also results and as the condition progresses complete retention of dyes like bromsulphalein may even occur. This liver function test is regarded by many as the most sensitive, detecting even early liver damage (King and Wootton [22], Mateer et al [23]) and indeed this has been our own experience in our work on tribulosis (unpublished). It is regarded by many as the most reliable and sensitive test for use in canine medicine. (Coffin [24], Sverbely et al [25]).

There will also be interference with the excretion of enzymes like serum alkaline phosphatase and serum amylase. The blood levels of the former rise very high in biliary obstruction. Garner (26) states that this test is unsatisfactory in cattle owing to the relatively wide normal range of this enzyme in the blood of these animals. These enzymes will be discussed more fully in a later paper.

In severe liver damage there is a failure of hepatic control of the water balance of the body. This may be due to peripheral vascular collapse similar to that seen in shock, usually seen in acute liver disease, or oedema, ascites and oliguria, found in advanced chronic liver disease and due to lowered serum albumin, portal hypertension and cardiovascular failure. György and Goldblatt (27) mention sodium retention and the presence of an excess of water retaining steroid hormones in the blood in certain forms of liver disease. There is naturally also a decrease of water produced by the liver in parenchymal damage.

Glycogenesis is markedly interfered with and the glycogen reserve of the liver will become depleted in severe prolonged liver disease. The same holds good for gluconeogenesis and glycogenolysis in the liver. The blood sugar level can then be expected to fall, while the lactic acid level would be expected to rise. A depletion of muscle glycogen can also be anticipated and the body easily becomes fatigued after exertion. The animal can be expected to show a decreased glucose tolerance and decreased utilization of galactose. The latter has been demonstrated by Garner (26a) in liver dysfunction in cattle.

In complete liver collapse, like acute yellow atrophy, an insufficiency in the deamination of amino acids arises, with a subsequent decrease in urea formation. The blood amino acid level, therefore, rises while that of the urea falls to below normal limits (Graf 28). Ammonia also appears in the blood in increased amounts, while aminoaciduria becomes apparent and tyrosine crystals are often seen in this urine.

A decreased synthesis and storage of protein in the liver can be expected, with also decreased synthesis of the plasma proteins especially albumins, fibrinogen and prothrombin. A reversal of the albumin globulin ratio occurs with its attendant vascular effects. Glycine synthesis is impaired and hence detoxication of benzoic acid may be expected to be interfered with, although it may take place in the kidneys of ruminants and dogs as well.

In our domestic animals defects in the conversion of uric acid to allantoin will arise, with a reversal of the ratio of these compounds. Bloom (29) regards this as a sensitive and accurate indicator of even mild liver damage in the dog.

As we have seen choline synthesis is disturbed and fatty infiltration may commence. Defective utilization of vitamin K will produce a delayed clotting time and a tendency to haemorrhage. Defects in the absorption storage and utilization of vitamins A and C also arise and symptoms related to deficiencies of these vitamins may become evident. Excess oestrogens may accumulate in males with concomitant mammary development or gynaecomastia

Other steroidal hormones are affected in the same manner. Grave interferences with the detoxicating mechanism arise. In chronic hepatic disease a macrocytic anaemia arises due to defective utilization of the antianaemic factors. The possible development of signs of photosensitization in ruminant liver disease has already been referred to.

It must be borne in mind that all the changes just mentioned are those one would expect to find in extremely severe or total liver dysfunction and have been presented to enable the practitioner to gain overall knowledge of the various changes which can take place in liver dysfunction varying from minor to severe grades of insufficiency. Later papers will deal with the actual biochemical changes encountered in various forms of liver dysfunction and with liver function tests. However, Lichtman (30) has listed the functions of the human liver which become impaired after varying degrees of damage as follows:-

Liver dysfunction in minor grade insufficiency.

Bile salt synthesis.
Glucuronic acid production.
Detoxicating mechanisms
Glycine synthesis.
Glycogen synthesis and storage.
Bilirubin excretion.
Bromsulphalein excretion.
Plasma protein synthesis.
Electrophoretic changes in plasma proteins.

Liver dysfunction in moderate grade insufficiency.

Blood cholesterol, total and ester fractions.
Prothrombin: Vitamin K response.
Serum alkaline phosphatase level.
Carotene, Vitamin A synthesis and storage.
Thiamine, Nicotinic Acid metabolism.
Water Metabolism.
Blood Glucose homeostasis.
Oestrogen inactivation.
Lipid mobilization and metabolism.
Iron, Copper storage and utilization.

Liver dysfunction in severe grade insufficiency.

Deamination of amino acids.
Glycogenolysis, gluconeogenesis.
Ketone body formation.
Electrolyte imbalance.
Blood volume control
pulmonary oedema.

It is very likely that this scheme is also applicable to liver dysfunction in most species of our domestic animals.

As mentioned earlier a very wide variety of symptoms may be expected in liver dysfunction which depend on the type of

damage, its course and its severity. Primary liver disease will often produce disease in other organs e.g. the heart and kidneys and conversely primary lesions elsewhere often lead to secondary liver lesions. These factors often obscure the clinical pattern. In general the following symptom patterns indicate various types of liver disease:-

(i) *Acute Liver Disease*:- Anorexia, thirst, vomiting, epigastric pain, hepatic enlargement, icterus, constipation or a putrid diarrhoea, fever or subnormal temperature, psychic disturbances, muscular twitchings and cramps, ataxia, delirium, coma and death. There may also be dilation of the pupils and abnormal distention due to flatulence.

(ii) *Subacute Liver Disease*:- Anorexia, icterus, intermittent chills and fever, epigastric pain, hepatic enlargement, constipation or diarrhoea, and often neutrophilia.

(iii) *Chronic Liver Disease*:- Anorexia, cachexia, unthriftiness and gradual loss of weight, lassitude, fetor hepaticus, mild icterus or none at all, anaemia, ascites, anasarca, increase or decrease in liver size, sensitiveness over the liver, caput medusae, tumor splenis, bleeding tendencies, vague digestive disturbances, nausea, cyanosis, bony charges, steatorrhoea, psychic disturbances, motor symptoms like muscular irritability, twitchings, cramps, convulsions or paralysis.

(iv) *Biliary obstruction*:- Icterus, offensive clay coloured faeces in non-ruminants, constipation or diarrhoea, anorexia, flatulence, vomiting, pruritus, delayed clotting time, normal or subnormal temperatures and in long standing cases, bony changes and the nervous symptoms listed above.

The psychic disturbances produced in liver disease are diverse varying from mania and aggressiveness to dullness or stupidity. One has only to consider conditions like acute "Chase valley disease," acute liver atrophy in horses or chronic seneciosis to appreciate how frequently these mental disturbances are associated with liver disease in our domestic animals. Hofmeyr (31) described mental aberrations in a case of biliary obstruction with associated parenchymal damage in a Great Dane bitch.

Unfortunately only the advanced stages of hepatic disease are easily recognizable. Prior to this, signs and symptoms may be few or lacking, especially in bovines and sheep. Acute liver disease is distinguished by a short and violent course, followed often by collapse, while the course of chronic liver disease is very prolonged. There are, however, a few "common denominators" in all these patterns which one could almost regard as cardinal signs of liver disease when taken together. They are:-

(a) digestive disturbances:- anorexia, vomiting, flatulence, constipation, diarrhoea, with copious clay coloured foul smelling greasy stools.

(b) Pain or tenderness over the liver.

(c) Icterus and bilirubinuria.

(d) Nervous symptoms, excitability or dullness, twitchings, convulsions, delirium and coma.

Icterus is often absent even in some severe forms of liver disease. It is at once obvious that a carefully elicited history of the case presented is essential and the importance of a meticulous examination cannot be overstressed.

Lichtman (30) has tabulated the chief patterns in humans together with the associated symptomatology as follows:-

Primary Parenchymal Defects

Anorexia	Arthralgia	Anaemia
Fatigability	Dyspoea	Fœctor hepaticus
Myasthenia	Constipation	Hepatic coma
Myalgia	Diarrhoea	Mental depression
	Acneform rash	Hypoproteinaemia

Primary Vascular, Circulatory, Haemodynamic defects

Splenomegaly	Venous collaterals	Venous bruit
Ascites	Caput medusae	Epigastrium
	Oesophageal varices	Haematemesis
	Haemorrhoids	

Primary Biliary stasis or failure

Xanthoma	Osteomalacia	Diarrhoea
Pruritus	Osteoporosis	Steatorrhoea
Nyctalopia	Constipation	Bleeding tendency

Parenchymal Vascular defects

Spider angiomata	Oedema (general or pulmonary)	Hypoproteinaemia

Parenchymal Biliary defects

Icterus	Epigastric distress	Bleeding tendency
Steatorrhoea	Pruritus	Hypoprothrombin-
	Cholaemia	aemia.

Most of the table above is probably applicable to liver disease in our animals, but some of the symptoms listed are seldom if ever seen, namely oesophageal varices, spider angiomata, haemorrhoids and Xanthoma. Hofmeyr (31) described caput medusae and venous collaterals seen in one case.

It is hoped that this review may help the practioner in some small way to understanding more fully the clinical pathology of liver disease and that it will be of some assistance in helping him to correlate all the vague and varied symptoms shown in these conditions.

SUMMARY

A brief review of the normal physiology of the liver is presented, followed by a short note on the pathogenesis of liver dysfunction in general and a discussion of the pathological physiology which may be expected in liver disease. A short discussion on the clinical patterns of liver dysfunction is then given.

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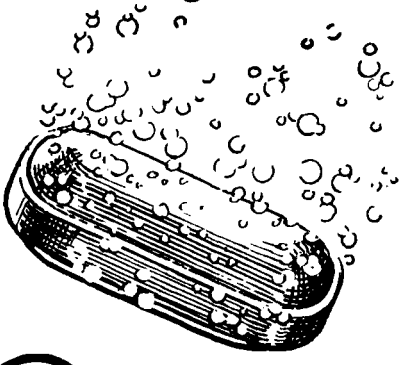

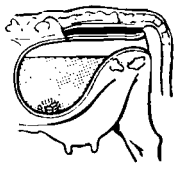

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IMPRESSIONS OF VETERINARY PROBLEMS IN GREAT BRITAIN

B. C. Jansen
Onderstepoort

During my visit in the United Kingdom I was impressed by the fundamental similarity between the problems confronting the veterinary profession in that country and ours. This applies to bacterial diseases, metabolic disorders, vegetable, mineral and industrial poisoning and also parasitism, although they are free of the tropical diseases which cause our persistent headaches. Through local circumstances, however, some disease conditions have come into prominence and in the following passages I shall elaborate on a few of these.

On account of the competition in the production of pork and bacon offered by the Scandinavian countries much attention has lately been focused on the British pig industry both from a point of husbandry and the study of disease. The wide distribution and economic importance of virus pneumonia of pigs (V.P.P.) and atrophic rhinitis are realized and have led to intensive research. V.P.P. affects pigs of all ages but most frequently young ones. A great deal of confusion exists about the aetiology of this condition. Whittlestone, at Cambridge, has identified the causal organism as a filterable agent larger than 0.7 u. The virus is capable of causing extensive pneumonia by itself, but in the majority of natural cases secondary bacteria are also present. The lesions persist in infected pigs for a long time, even though coughing has stopped. These cases perpetuate infection in a breeding establishment.

Once V.P.P. is established it persists in an endemic form and new introductions become affected in two to three weeks. When the infection is first introduced into a fully susceptible herd the morbidity is 100%, with a high mortality rate, but in practice most cases are chronic. When piglets are infected from the sow they show symptoms at three to six weeks. There is a dry, non-productive cough which is most marked in the morning but can be elicited by vigorous exercise. The cough may disappear after some weeks and there may be a short period of abortive sneezing. The pigs do not thrive although their appetites remain good. A number usually develop an acute, fatal pneumonia. If the pigs are kept under optimal conditions the effect of the disease is mitigated.

At post mortem the lesions are mainly confined to the ventral portions of the cardiac and apical lobes of the lungs. They are greyish and resemble lymphoid tissue, clearly demarcated from the normal tissue. Frequently pleurisy and pericarditis are seen and the bronchial and mediastinal lymph glands are enlarged and oedematous.

The present scheme of eradication depends on the rearing of litters in isolation. The sows are farrowed down in out-of-door

fold units and the litters are observed closely for coughing; any that show it are regarded as infected. If no coughing is observed up to 8 weeks of age the litter is weaned but retained in isolation for a further 3 weeks. If there is still no coughing the young pigs are regarded as probably free from infection. They may then be mixed with other similar litters but segregated from the rest of the herd.

At weaning or at pork or bacon weight those of the "clean" pigs not required for breeding are sent to slaughter and their lungs examined for pneumonia lesions. The original breeding stock is eventually replaced by clean pigs.

As the disease spreads by droplet infection, clean pigs should be well separated from possibly infected ones and examination of lungs at slaughter must continue for months to confirm complete freedom.

I was fortunate in seeing atrophic rhinitis illustrated in all its different phases at Weybridge. Its aetiology is as yet undetermined. Atrophic rhinitis produces its severest effects and is most readily recognised in rapidly growing pigs in the first three months of life. It frequently co-exists with V.P.P. and causes considerable economic loss. It spreads by contact or short range droplet infection, usually from the sow in the first week of the piglets' life. Infective material will survive freezing, but the infection does not persist in a contaminated piggery for as long as three weeks under ordinary circumstances.

The incubation period is 10-14 days and in the acute stage sneezing or snuffling spreads in the litter. There are signs of nasal irritation and a discharge. Conjunctivitis is present with closure of the lachrymal duct and overflow of tears. The acute symptoms usually pass but the growth of the piglets is checked. About 10% of piglets die in the acute phase and a considerable number become chronically affected: they show occasional sneezing and a progressive deformity of the snout. Epistaxis and snorting are common. The snout becomes thickened and the skin over the nose corrugated; protrusion of the lower incisors follows and the snout may be drawn to one side. Brown lines down the face from the medial canthi of the eyes are caused by the constant flow of tears. When fully-grown pigs are infected they show light, transitory symptoms but remain infective.

At postmortem, acute cases show a congestion and catarrh of the nasal mucosa. J. Done has described inclusion bodies in the cells of the nasal mucosa during the acute stage. Chronic cases show a deformity, contraction and softening of the turbinate bones and bowing of the septum nasi. These changes are best seen if the snout is sawn transversely in front of the second upper premolars.

The only way to control this disease is by aseptic delivery and artificial rearing of piglets. In aseptic delivery each piglet is removed from the sow as it emerges from the birth canal or, alternately, all the piglets are removed at once by Caeserean section. As may be realised, the completely artificial rearing of piglets has offered many difficulties but I feel convinced that the method has been proved successful. This drastic procedure can also be applied to the eradication of V.P.P.

While in S.A. we are acquainted with large scale inoculation against Anthrax, a completely different attitude is adopted in Great Britain due to the characteristic epidemiology of this disease in that country. In the neighbourhood of tanneries regular inoculation is undertaken since anthrax often occurs as a result of spores contaminating the factory effluent. On farms removed from these areas only single cases seem to occur as a result of contaminated feed material and the disease does not establish itself in the soil. Consequently only disposal of the carcase by burning or deep burial is undertaken where a case has occurred.

An interesting, though serious, situation has arisen in some areas where tuberculosis has been eradicated, in so far as Johne's disease is showing an increased incidence. The theory, that the state of premunity resulting from subclinical tuberculosis infections protects against other acid-fast bacterial diseases, has been advanced.

In Britain considerable concern is caused by the disease scrapie affecting sheep. This is characterised by symptoms of intense and progressive pruritus, progressive asthenia and locomotor in-co-ordination. The causal factor is an infective agent of which the precise nature has not yet been determined but it is suspected of being a virus. The agent can withstand boiling for 30 minutes and a concentration of 0.35% formalin. It is believed that infection occurs congenitally through either parent and the disease can be transmitted by intracerebral, subcutaneous, intravenous and intradermal injection of infective material. The incubation period may be up to three years but is usually about five months experimentally and is seldom observed in sheep under 18 months old. Scrapie is present in Britain, Germany and France and was imported into Canada, U.S.A., Australia and New Zealand.

The onset of the disease is insidious: nervousness and excitability and slight tremors of the head and neck become evident. If affected sheep are rounded up they become tense with excitement and show rapid muscular tremors. The sick animal has a staring, fixed facial expression, its ears assume an unnatural position, it shows a marked pruritus, and feeding and rumination are interfered with. Skin abrasions and loss of wool result. Emaciation and weakness are progressive and aberrations of gait are obvious. Some sheep show epileptiform convulsions. In a few cases no appreciable skin irritation is present.

Scrapie is regarded as invariably fatal with only a few spontaneous recoveries. Some animals take six months to die.

Recently Bosanquet, Daniel and Parry (The Lancet, October 13, 1956, page 737) have, by approaching the problem in an unconventional fashion, produced convincing evidence that, pathologically, scrapie is a myopathy. All the clinical manifestations could be traced to the muscular lesions.

During my visits to some of the veterinary institutes I saw very well illustrated the difference between foot-abscess and foot-rot in sheep. The former is caused by Bacillus necrophorus and usually affects only one foot; the disease never assumes the form of an outbreak and is not transmissible artificially. With foot-abscess there is no underrunning of the horn but it appears as a

fistulous condition with involvement of the joint and thickening of the soft tissues around the coronet. It mostly affects adult sheep.

Foot-rot, on the other hand, is caused by a combination of three organisms: Fusiformis nodosus, Spirochaeta penortha and a motile fusiform, and occurs in the form of outbreaks. Noticeable changes consist of an underrunning of the horn tissue with subsequent loosening. This disease is easily transmisible and there is no involvement of the tissues around the coronet.

The system of Veterinary investigation Centres dispersed through the country definitely deserves praise. The Veterinary Officers at these centres have all the facilities for investigating diseases and have the advantage of being close to any point in their operational area. As a result, all specimens are examined in a fresh state which facilitates the diagnosis of conditions caused by sensitive organisms. These people also have a first-hand knowledge of the local conditions in their areas. Although major problems are referred to central research laboratories, diseases like Clostridium welchii type C diarrhoea in piglets and spirochaetosis in cattle have been virtually completely investigated in the field by V.I.O.'s.

TRANSMISSIBLE GASTRO-ENTERITIS OF PIGS

(A review)

M. W. HENNING

Onderstepoort

Transmissible gastro-enteritis is a virus disease of pigs associated with severe vomition, profuse diarrhoea, dehydration and marked loss of condition. It is highly fatal in baby pigs under 10 days of age, but may affect pigs of all ages. The disease was first reported by Doyle and Hutchings (1946). Its natural history and origin have remained obscure so far, and it is not known whether some wild animal acts as a natural reservoir of the infection.

ETIOLOGY

Doyle and Hutchings (1946) showed that the infecting agent was present in the ingesta and the walls of the gastro-intestinal tract, and that it was capable of passing through bacterial filters. Hutchings (1947) was able to maintain this agent by serial passage in baby pigs for 32 generations. Lee, Moro and Baker (1954) report that, during the acute stage of the disease, the virus is present in a high concentration in the intestines and kidneys of the affected pig, but only in a much more diluted form in the blood, liver, spleen, lungs and brain. They were able to recover it from the lungs, blood and kidneys only for a limited time, but found that it persisted in the faeces for at least 8 weeks. They believe that the infecting agent may be maintained in the faeces of a recovered animal for a long time, as happens with the virus of poliomyelitis.

Young, Hinz and Underdahl (1955), by using gradocol membranes, have estimated that the size of the virus particles is approximately 200 m/h.

PROPAGATION

The virus has an affinity for the mucous membranes of the gastro-intestinal tract and the kidneys and occurs in its highest concentrations in these organs. It multiplies rapidly in all parts of the intestine. Intranasal instillation a few hours after birth leads to a rapid multiplication of the virus in the lungs but not in the nasal mucosa. On the other hand, propagation occurs in the nasal mucous membrane, but not in the lungs, in six day old pigs.

There is a viraemia during the acute stages of the disease, but the virus is present in the blood and internal organs for only a short time. It does not occur in the urine (Lee et alii, 1954) of infected pigs.

To be published in the next edition of "Animal Diseases in South Africa."

It has not yet been possible to cultivate the infecting agent in the chick embryo (Haelterman and Hutchings, 1956) but Lee (1956) has succeeded in propagating it in a tissue culture medium containing pig-kidney epithelium. He was able to maintain the virus on this medium for 8 months using 28 serial transfers. There is some evidence that a certain degree of attenuation is effected during the tissue culture.

VIABILITY

Hutchings (1947) states that under natural conditions the virus does not persist in infected premises from one season to another, but he succeeded in maintaining the infection present in the gastro-intestinal tract in the frozen state for 13 months. Bay, Doyle and Hutchings (1952) were able to inactivate the virus with 0.5% phenol, 0.05% formalin or by heating at 56°C for 30 minutes. A suspension of gastro-intestinal tract kept at room temperature remained active for three days but not for 10. The virus present in the ground infected intestinal tract survived after storage at -28°C for three and a half years. Neither the antibiotics, penicillin or streptomycin, nor the sulphonamides had any effect on its viability (Haelterman and Hutchings, 1956).

TRANSMISSION

Doyle and Hutchings (1946) and Bay, Hutchings, Doyle and Bunnell (1949) showed that the disease could be readily transmitted by direct contact or by the oral administration of ground intestine or ingesta from infected pigs. They were also able to infect susceptible pigs by means of bacteria-free filtrates or triturated infected gastro-intestinal tract or infected ingesta given by the mouth.

Reports of transmission by parenteral routes are less satisfactory. Although Bay et alii (1949) succeeded in infecting susceptible pigs parenterally, Lee et alii (1954) could produce no more than a mild temperature reaction. Young et alii (1953) were able to transmit the infection intraperitoneally only after the employment of large amounts of infective material. According to Bay et alii (1949) baby pigs can be infected by infected filtrates in dilutions of one in a million.

PATHOGENICITY

Transmissible gastro-enteritis is a disease confined exclusively to swine. No other species of animal has yet been proved to be susceptible. Bay et alii (1952) failed to transmit it to hamsters, mice, rabbits and guinea-pigs. It is a disease mainly of baby pigs, causing a very high rate of mortality in animals up to about 10 days of age. Older pigs, weaners and even farrowing sows are also susceptible, but although they may develop acute symptoms and lose much weight, they usually recover.

EPIDEMIOLOGY

The source of transmissible gastro-enteritis is not known; it was completely unknown in veterinary literature until Doyle and Hutchings (1946) reported it. The disease often occurs sporadically but is very widespread in the pig raising areas of the United States, and its incidence seems to be increasing (Hutchings, 1947). Bay, Doyle and Hutchings (1953) do not believe that infected sows remain carriers so that the method of propagation of the disease under natural conditions from season to season is not quite clear yet. Sometimes an outbreak follows the introduction of a new pig which may be an inapparent carrier, but more often an enquiry will reveal that, apart from boars, no swine have been brought in for a long time. Nevertheless Young, Underdahl and Hinz (1953), and McNutt (1953) report that the disease appears in a clean herd only following the introduction of new stock, so that the existence of inapparent, symptomless carriers cannot be entirely ruled out.

Transmissible gastro-enteritis often starts in one litter and quickly spreads to others; any fresh pigs farrowed soon also become victims. Most outbreaks are sporadic, usually affecting successive litters only on one farm in a community, although sometimes the infection may spread from farm to farm to involve a large area. The disease often fails to recur on an infected farm even in the progeny of sows whose litters suffered badly during a previous farrowing.

The incidence is seasonal, most cases occurring in winter or spring. Outbreaks in summer are rare. It is believed that there are certain contributory and predisposing factors which favour the pathogenesis. These include the farrowing of a large number of sows during the same season, and the confined quarters used for housing so that some of the houses used are too closely packed with pigs. On the other hand, outbreaks have been reported in pigs that were housed under conditions that were regarded as ideal.

SYMPTOMS

(Doyle and Hutchings, 1946; Feenstra, Thorp, Gray and McMillen, 1948). The incubation period may be as short as 24 hours or less. It is rarely as long as 72 hours. In naturally exposed pigs born in infected quarters symptoms may be observed on the day after birth. The clinical symptoms are constant in pigs under 10 days of age. The first sign of illness is vomition, followed by a profuse diarrhoea characterized by yellowish, greenish, or whitish evacuations, often resembling curdled milk. There may be a rise in temperature but this is usually of short duration, being followed by a normal or subnormal temperature. Dehydration is rapid and an excessive thirst is developed. The pig becomes progressively weaker and rapidly loses weight. Death usually supervenes from the second to the fifth day after the onset. In baby pigs that survive, the diarrhoea may persist for from 5 to 10 days. But when the virus persists the "recovered" pig remains stunted and fails to gain weight normally (Lee et alii, 1954).

The infection soon spreads from the baby pigs to the mother and to other pigs on the premises. But manifestations of disease in swine over two weeks of age are inconstant, and the presence of the infection may not always be apparent. The sow may show symptoms from two to three days after the first piglet sickens. Vomition, diarrhoea, loss of weight, diminution and even complete suppression of milk secretion are the outstanding symptoms. Sometimes, however, the only disturbance is a transient inappetence or there may be no evidence of disease whatsoever. Most of the affected sows recover completely within the course of about five days.

Frequently the epizootic spreads throughout the drove involving, in addition, suckling pigs of two weeks or more, weaners, porkers, baconers and gilts. The symptoms resemble those manifested by the sow. The course is usually short and the diarrhoea seldom lasts for as long as a week.

Most of the older pigs recover within a period varying from two to five days. Spontaneous recovery of most of the pigs over two weeks of age is the rule, and death is rare in this age group. Some pigs that have apparently recovered fail to gain weight, remain stunted, and may act as carriers of the virus for some time (Lee et alii, 1956).

COURSE

In baby pigs exposed to infection the morbidity may be 100% and the mortality over 90%. It is not uncommon for every pig born to succumb. Death usually supervenes in from two to five days. Pigs that survive for more than five days usually recover but are often stunted and may remain carriers for some time. The course is much more favourable in suckling pigs over 10 days of age and in older pigs, recovery being the rule. The symptoms manifested by them may vary considerably in the different animals. In some the gastro-intestinal disturbance may be severe and the loss of condition well-marked. In others the symptoms are very mild and hardly any disturbance can be noticed. In still others no signs of illness whatsoever are manifested.

LESIONS

(Doyle and Hutchings, 1946; Haelterman and Hutchings 1956). The most important lesion presented is a gastro-enteritis, the extent of which may vary greatly from animal to animal. One is often impressed with the apparent lack of gross inflammatory changes which one would expect to find in such a severe involvement of the gastro-intestinal tract. Hutchings (1947) has described the intestinal lesions as a "superficial enteritis." It is possible that some of the changes exhibited are due to the effects of secondary invaders. Haelterman and Hutchings (1956) report that affected pigs kept in isolation may not show any definite changes at death other than dehydration, excessive fluid in the intestine and a slight congestion of the mesenteric blood vessels.

In a number of pigs, however, there is a moderate or severe hyperaemia of the gastric and intestinal mucosae which may be especially marked in the region of the fundus. The hyperaemia can usually be readily observed from the serous aspect. Feenstra et alii (1948) reported haemorrhages in the walls of the stomach. The mesenteric blood vessels are usually markedly engorged; the kidney may exhibit degenerative changes, and often contain urates. A constant feature of nearly all cases of disease is the presence of a large amount of fluid in the intestinal canal, having a yellowish, greenish or whitish colour.

Histologically also no characteristic changes are manifested. There may, however, be some evidence of a superficial necrosis of the intestinal epithelium associated with a neutrophilic infiltration of the mucosa, congestion of the intestinal mucosa and renal medulla, albuminous or fatty degeneration of the tubular epithelium of the cortex of the kidney. Feenstra et alii (1948) reported necrotic foci in the gastric mucosa associated with haemorrhages in the submucosa.

DIAGNOSIS

As noted above, neither the symptomatology nor the pathology can be relied upon for recognizing the disease. The sudden appearance of a markedly infectious, highly fatal disease, associated with severe vomition and profuse diarrhoea, in a litter of baby pigs is usually presumptive evidence of transmissible gastro-enteritis. But the existence of the disease can be definitely proved only by means of biological tests on susceptible baby pigs under 10 days old, exposed to infection with intestinal contents or an intestinal emulsion.

In the live pig vomition is a sign of specific significance, and at post mortem atony and dilatation of the intestine, when present, may be useful evidence. Further proof is the relatively benign nature of the disease in older pigs.

SERUM NEUTRALIZATION TESTS

Young, Hinz and Underdahl (1954) and Haelterman and Hutchings (1956) report encouraging results in their attempts to neutralize the virus in vitro with the sera of recovered swine. But the fact that relatively expensive non-susceptible baby pigs are the only animals that can be used makes the test rather uneconomical.

With the conglutinating complement absorption test Lee (1956) was able to detect the presence of antibodies in animals infected either with natural virus or tissue culture virus. The supernatant fluid of a tissue culture was employed as the source of the antigen.

DIFFERENTIAL DIAGNOSIS

An outbreak of sporadic diarrhoea may be confused with transmissible gastro-enteritis, but the relatively benign nature of this disease and the rapidity with which it responds to treatment

with sulphonamides, bismuth, calcium, antibiotics and other drugs will generally rule out transmissible gastro-enteritis.

Swine fever which may also cause digestive disturbances severely affects pigs of all ages.

TREATMENT

No effective chemotherapeutic agent has yet been found. Various antibiotics, sulphonamides and arsenical preparations have been tested, but not one has exhibited any curative properties (Bay, Doyle and Hutchings, 1952).

IMMUNIZATION

Exposure to infection confers on the recovered pig a definite active immunity, at least of short duration (McNutt, 1953). But as the incubation period of the disease is so short and as baby pigs may start showing clinical symptoms as soon as 24 hours after birth, immunization of piglets obviously does not offer a solution for the control of the disease. Bay (1952), Bay, Doyle, Hutchings (1953), and Haelterman and Hutchings (1956), however have reported that baby pigs, the progeny of sows that had been exposed to oral infection some time before farrowing, exhibit a fair degree of resistance to infection. It is believed to be a colostral immunity conferred on the piglet by immune colostrum. It may be interesting to note that the progeny of sows infected by the intramuscular and subcutaneous routes did not exhibit the same degree of resistance, and that the majority of them succumbed when exposed to infection. Nelson (1954) and Haelterman and Hutchings (1956) partially succeeded in controlling natural outbreaks of the disease by deliberately dosing the breeding sows some time before farrowing with intestinal contents obtained from infected pigs. Further work on the production of a colostral immunity as a means of controlling transmissible gastro-enteritis seems therefore to be justified.

According to Lee (1956) tisue culture propagated virus, though less virulent than the natural infecting agent, nevertheless protects animals that received it against the more pathogenic intestinal propagated virus. An attenated tissue culture virus may, therefore, prove to be of use as an immunizing agent for farrowing sows.

CONTROL

The application of the usual sanitary measures and the careful isolation of farrowing sows in clean quarters are recommended. Where isolation measures are practised it is essential that the sows be maintained at a safe distance from the seat of infection, because the disease is known to spread rapidly under ordinary conditions of farming.

Hutchings (1947) reported serious outbreaks in litters farrowed in well-constructed, hygienic houses. On the other hand,

he states that the infection does not survive on the same premises from one farrowing to another.

Bay (1952) believes that one of the most effective procedures for controlling the disease consists of merely interrupting farrowing on the infected premises for a period of four to eight weeks.

Ordinarily transmissible gastro-enteritis soon disappears from an affected herd, especially if it is a small one, so that only the pigs from one season's farrowing usually suffer.

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STRAIN 19 BRUCELLA VACCINE

IV. REDUCED RISK OF LOCAL REACTION BY SUBCUTANEOUS INOCULATION INTO THE EXTERNAL EAR.

G. C. van Drimmelen.

ABSTRACT: Preliminary results of a vaccination technique intended to obtain a maximal contact between live antigen and the reticulo-endothelial system with a minimal local tissue reaction.

Several attempts at producing a satisfactory immunity against bovine brucellosis with less than the massive dose of live bacteria recommended by the U.S. Bureau of Animal Industry[1] have apparently been successful[2,3,4]. Nevertheless the joint FAO/WHO Expert Committee on Brucellosis state "The minimal dose necessary to produce an adequate protection by the subcutaneous, intradermal, or any other route, is not, however, yet known. It is therefore concluded that, at present, the recommendation for a dose of 5 ml (approximately 60,000 million viable organisms — 60 x 10^9) injected subcutaneously should be adhered to." "The Committee emphasized that, at the actual time of injection, the vaccine must have the full content of viable strain 19 organisms and that these must be in an undissociated state."[5]

At one time it was suggested that a method of intradermal inoculation be adopted in an attempt to obtain the greatest possible benefit from the inadequate vaccine supply available. The suggestion was not supported at Onderstepoort because the shortage could be overcome by other measures. Vaccine production has recently been augmented to meet all demands by means of new culture techniques and concentration before freeze-drying.

The route of inoculation has remained an important consideration, particularly in adult cattle and in connection with pregnancy and lactation. Also, in the case of animals prepared for show purposes, some objections to blemishes alleged to have resulted from earlier inoculation into the side of the neck have been encountered.

Briefly, the reported methods of inoculation are:-

(a) Subcutaneous injection of 5 ml vaccine into the side of the neck. This is the conventional method and it is carried out with an ordinary hypodermic syringe and stout needle.

(b) Intradermal vaccination. This requires a special (dental) syringe and needle, and the greater skill required for the operation would delay progress of an anti-brucellosis campaign with lay inoculators.

(c) Intracaudal inoculation. This method, introduced by Australian workers,[6] requires syringes of exceptionally robust construction. If rubber pistons are employed rapid wear is to be expected. The inflammatory process in the tail tip is

rather distressing although in our hands no serious lesions have been encountered. The local necrosis may be present in 40% to 50% of vaccinated animals but only results in sloughing off of the tip, with hardly noticeable reduction in size of brush.

(d) The caudal fold injection reported by Thorne[7] in Nigeria is affected by the objections under (b). Frequent tick infestation of this part decreases its usefulness.

(e) In a previous publication[8] inoculation into the dewlap was demonstrated and the obvious advantages have often been stressed in recent years. The site of inoculation is, however, low to reach and in a cattle-crush, not easy to handle.

Experiments with subcutaneous injections into the external ear have been initiated. The present paper is a preliminary report on this technique. The purpose of the work is to supply the clinician with an elegant and effective method. A method which causes little trouble and which can be carried out with an ordinary hypodermic syringe and needle, but which at the same time limits the local reaction to a minimum, commends itself to the client and practitioner alike.

MATERIAL AND METHODS

The experiments were carried out with routine Strain 19 *Brucella* vaccine. Freeze-dried vaccine was used at two levels of dosage (1) 20 x 10^9 viable organism per dose of 1 ml.
(2) 100 x 10^9 viable organism per dose of 1 ml.

The latter dose was prepared by reconstituting the concentrated vaccine,[9] consisting of living *Brucella* organisms, freeze-dried in an equal volume of spent medium with added lactose (5%), ascorbic acid (1.5%), thiourea (2.25%), and sodium carboxy-methyl-cellulose (0.06%), to one fifth of the prescribed dilution.

For control purposes a number of animals were given an injection of vaccine by conventional methods under the skin of the neck. On each occasion, two guinea-pigs were given one fifth of the cattle dose in order to compare the antigenic response obtained.

The inoculation of calves was carried out at the age of 6 to 8 months when they were held by hand in the camp (see fig. 1). Large animals such as adult heifers and cows were controlled in a cattle crush with the head firmly tied to the bars. The tip of the ear was held in the left hand and its convex surface cleaned with sterile water. A long needle (5-7 cm, gauge 19) was used on a hypodermic syringe. The point of the needle was inserted under the skin on the posterior (convex) surface of the ear, 1 to 2 cm from the ventral (lower) border and between the middle and the tip of the ear. The needle was then passed in a medial direction between the cartilage and the skin for a distance of 5 cm. The dose was injected during gradual, partial

Fig. 1

Inoculating a heifer with Strain 19 *Brucella* vaccine subcutaneously into the external ear.

withdrawal to produce a weal of 2 to 4 cm long. With the left thumb pressed on the point of penetration the needle was afterwards completely withdrawn. No leakage of vaccine from the wound was allowed. The vaccine was spread under the skin by slight gentle rubbing.

RESULTS

(a) **Temperature reaction.**

Morning and afternoon temperatures of the experimental animals and of an equal number of controls vaccinated by conventional methods are compared in table 1.

(b) **Local reaction.**

Swelling of the skin and subcutaneous tissues was found in some experimental animals but was more common in the controls. A number of the experimental animals that did show a swelling appeared to be reacting to traumatic rather than infectious stimuli. Four animals accidently received their dose of vaccine on the central ridge of the ear, where muscular tissue was involved. Here acute, local oedema was followed by infiltration, necrosis and a cold abscess. This eventually became inspissated and gradually organized into a small thickening of the ear (see figure 2).

TABLE 1

Comparison of the temperature reactions resulting from subcutaneous inoculation of strain 19 Brucella vaccine into the external ear and into the neck.

(degrees Fahrenheit; m = morning; e = evening)

GROUP	NUMBER OF HEIFERS	DOSE OF VIABLE ORGANISMS	AVERAGE TEMPERATURE														MAXIMUM TEMPERATURE
			DAYS BEFORE INOCULATION			DAYS AFTER INOCULATION											
			2	1	0	1	2	3	4	5	6	7	8	9	10	11	
			m e	m e	m e	m e	m e	m e	m e	m e	m e	m e	m e	m e	m e	m e	
Experimental inoculated into the ear Control	13	100 x 10^9	101.3 101.3	101.3 102.6	101.3 102.4	103.1 105.1	106.0 105.7	104.3 104.7	103.9 104.4	102.5 102.8	102.3 102.6	101.8 102.1	101.7 102.2	102.0 102.0	101.5 101.5	101.9 102.7	107.6
inoculated into the side of the neck	13	100 x 10^9	101.6 101.6	101.8 102.6	103.8 101.8	104.0 105.2	105.2 105.4	104.1 104.9	104.3 103.7	102.6 103.2	102.9 103.3	101.8 102.6	101.6 102.3	102.2 102.3	102.0 102.2	102.0 102.6	107.6

TABLE 2

Comparison of the antigenic response resulting from subcutaneous inoculation of strain 19 Brucella vaccine into the external ear and into the neck.

GROUP	NO. OF HEIFERS	DOSE OF VIABLE ORGANISMS	AVERAGE BLOODSERUM AGGLUTINATION TITRE							MAXIMUM TITRE
			Before inoculation	After inoculation						
				2 weeks	1 month	2 months	3 months	4 months	5 months	6 months
Experimental inoculated into the ear	13	100×10^9	<1:10	1:1132	1:440	1:130	1:32	1:48	1:31	1:11	1:2560
Control inoculated into the side of the neck	13	100×10^9	<1:10	1:978	1:140	1:65	1:20	1:15	1:6	1:3.1	1:5120

The primary reaction in experimental cattle was demonstrated by slaughter of two animals on the third and seventh day after inoculation. Sections of the ear at the site of inoculation were microscopically examined (see figure 3). The reaction in the controls from subcutaneous injection of vaccine on the side of the neck produced large, hard, local swellings with swollen prescapular glands.

(c) **Antigenic response.**

The agglutination titre resulting from experimental and control inoculations is compared in table 2.

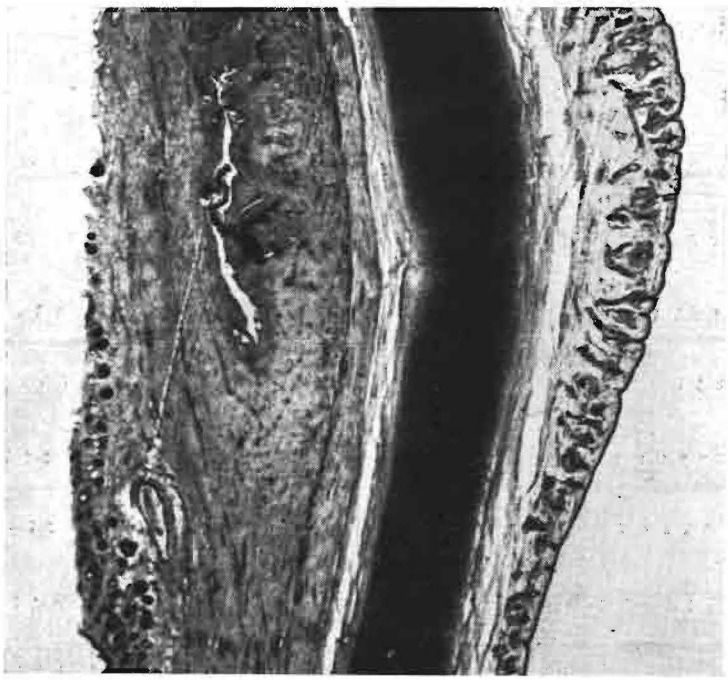

Fig. 2

Cross section of a lesion on the muscular part of the ear three months after inoculation with Strain 19 *Brucella* vaccine.

(d) **Immune response.**

Immunity was tested by a method devised to suit the local facilities and based on the successful tests of earlier workers[10,4,3]. The animals were tested monthly for serum antibodies before and after vaccination and served when convenient. Exposure was commenced during the fourth month of pregnancy and consisted of a monthly dose of 150 million viable *Brucella* organisms from a 48 hour culture of Strain 544 (McEwen) grown in 10% CO_2 atmosphere. The organisms were suspended in 0.1 ml buffered saline and placed in the conjunctival sac. This exposure was repeated monthly until abortion or calving supervened. At each exposure adult male guinea-pigs received one tenth of this dose for control tests. Microscopic, cultural and biological tests

were carried out on foetuses, afterbirths and colostrum. A preliminary test on pregnant cows has been completed (see table 3). A further report on the heifers inoculated will be published after two years.

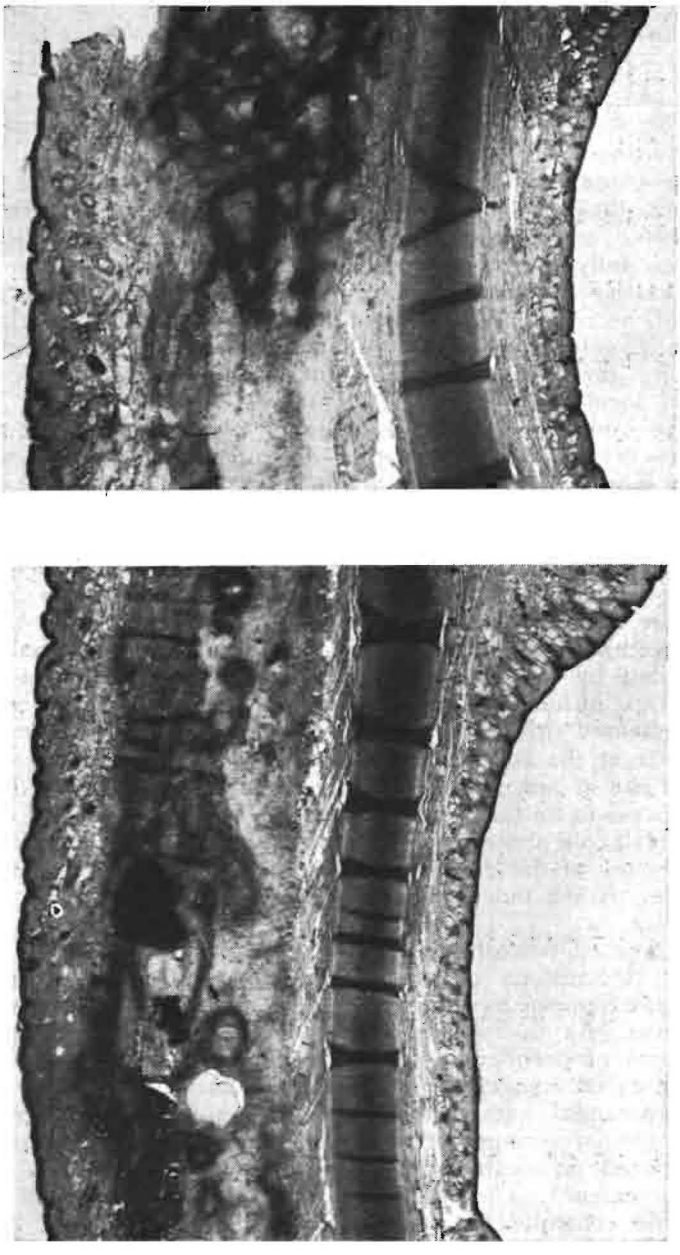

Fig. 3

Sections of ears showing local reactions 3 and 7 days after subcutaneous inoculation of Strain 19 *Brucella* vaccine into the ear.

DISCUSSION

The size of the vaccine dose selected for the test on pregnant cows was suggested by the "intra-caudal" inoculation method of the Australian workers[4,6,10]. When there was however, no longer any reason for using less than the full dose (60 x 10^9 viable *Brucella* organisms), because production methods for Strain 19 *Brucella* organisms were improved to the extent that future shortages became unlikely, the conventional dose was adhered to. For inoculation into the ear the vaccine was reconstituted to one fifth of the prescribed volume and thus 1 ml of the suspension contained the full dose. The view sometimes held by clinicians that the inoculation of *Brucella* vaccine into immune or infected cattle, elicits a tuberculin-like allergic reaction has not been fully investigated. The mere toxic effect of the mass of organisms inoculated could produce a local reaction to the point of necrosis. We believe that the animal by its vigorous ear movements after release following ear inoculation, assists in spreading the vaccine through the tissues, thereby minimizing risks of local reactions.

The preliminary findings in every respect support the view that the response to vaccination by subcutaneous injection into the ear is in no way inferior to the inoculation of vaccine at the side of the neck or into the dewlap.[8]

The advantages to be derived from ear inoculation are:-

(a) Decreased inflammatory reaction at the site of injection. It appears that this difference is the result of absence of muscular tissue.

(b) Decreased risk of functional disturbances. Lameness is excluded by inoculation into the ear. As in the intra-caudal method of inoculation no untoward effects have been observed.

(c) Decreased risk of permanent blemish. When vaccinating adult cattle at the side of the neck a hard swelling of the skin at the site of inoculation has sometimes been encountered. This appears to be the result of connective tissue formation around a small cold abscess possibly caused by toxic necrosis following injection of the dose. This has not been observed in the ear except when inoculation was done over the auricular musculature.

(d) Decreased risk of abortion in pregnant cattle. It is believed that inoculation of pregnant animals into the ear sets up less inflammatory reaction thus reducing the danger to the foetus. As the present cases were vaccinated in the early stages of pregnancy no conclusions can be drawn yet.

(e) No extra equipment is required. Unlike intra-dermal and intra-caudal vaccination, the injection into the ear requires no special syringes and does not increase the wear and tear imposed on ordinary hypodermic syringes.

(f) Convenience. The ear is easily reached both in calves and in cattle controlled in a crush.

Disadvantages which might be brought forward would probably be the leakage of vaccine from the thin skin of the ear and the care required to prevent the needle from penetrating the conchal cartilage.

TABLE 3

Results of Immunity tests after inoculation of Brucella vaccine subcutaneously into the external ear and after inoculation by the conventional route.
(Animals vaccined after service)

Number of Animal	*Method of vaccination*	*Dose of Vaccine*		*Exposed Months of Pregnancy*	*Abortion or Calving*	*Result infected or not*
		Volume	*No. of viable orgs.*			
7889	subcutaneous ear	1.0 ml	20×10^9	3rd, 4th, 5th, 6th, 7th, 8th	Calf No. 8520	not
7919	subcutaneous ear	1.0 ml	20×10^9	5th, 6th, 7th, 8th	Calf No. 8429	not
7800	subcutaneous ear	5.0 ml	100×10^9	3rd, 4th, 5th, 6th, 7th, 8th	Calf No. 8629	not
7801	subcutaneous ear	5.0 ml	100×10^9	3rd, 4th, 5th, 6th, 7th, 8th	Abortion (full term)	not
1482	not	—	—	4th, 5th	Abortion	infected
7574	not	—	—	5th, 6th	(foetus resorbed or aborted and eaten at night)	infected
7577	not	—	—	4th, 5th, 6th	Abortion	infected
7893	not	—	—	3rd, 4th, 5th	Abortion	infected

SUMMARY

The description of a new method of inoculating *Brucella* vaccine is given. The full dose of living organisms can be injected under the skin of the external ear if suspended in one millilitre of water. The relative absence of marked local reactions is demonstrated. Some preliminary comparative data are supplied.

ACKNOWLEDGEMENTS

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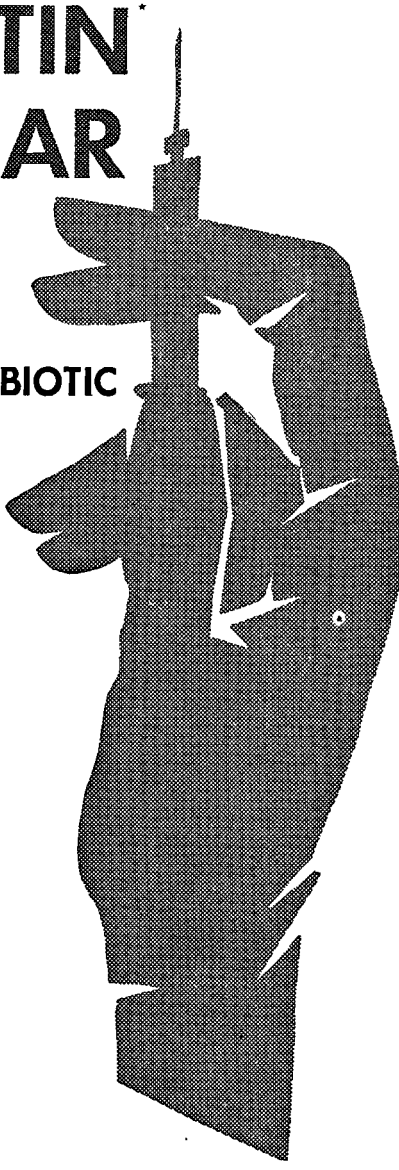

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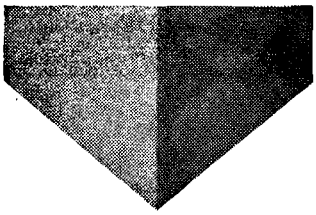

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TRANSMISSION OF BACTERIAL DISEASES BY BACTERIA-FREE FILTRATES

G. C. van Drimmelen

ABSTRACT: A brief review of the role of bacteriophage in bacterial heredity with special reference to some hypothetical aspects of pathogenesis.

The pathogenic properties of bacteria, such as invasiveness, toxin production and multiplication in the tissues of a host, are inherited characteristics. These can be acquired, lost or altered by mutation. The rate of mutation is itself under genetic control, the rate found in normal environment being known as the "Spontaneous Mutation Rate." Artificial or natural variations of the environment may reduce or increase the mutation rate. Many mutations such as the lethal mutations of bacteria cannot be detected, but the rate for some specific genes is constant, occurring once in every 10^6 to 10^8 multiplication divisions. Reverse mutations occur spontaneously with the same frequency.

Alterations of the spontaneous mutation rate of some known characteristics of certain bacteria have been produced by artificial physical, chemical and genetic influences (Griffiths, 1928; Lwoff, 1953; Hewitt, 1954; Shepherd, 1957). As examples may be mentioned:- temperature (Lincoln, 1947); X-rays (Kaufmann, Hollaender and Gay, 1946); caffeine (Novick & Szilard, 1952); extracts of cells e.g. capsular material or desoxyribonucleic acid (McCarty & Avery, 1946); and bacteriophage transduction (Freeman, 1951; Lederberg, Lederberg, Zinder and Lively, 1951).

The purpose of this paper is to examine the possible implications of recent developments in our knowledge of the transfer of inheritable characteristics by means of bacteriophage.

Bacteriophages are protoplasmic particles with nucleoprotein containing desoxyribonucleic acid, which is reproduced by organisation of non'phage material in the bacterial cell. For reproduction a 'phage has to: (1) be absorbed on to a growing bacterium, (2) inject its DNA into the bacterium leaving the empty protein capsule adsorbed onto the surface, (3) undergo a period of complete union with the bacterial cytoplasm (35 minutes or longer), and then commence "replication" of the 'phage DNA in the bacterium and eventually formation of the complete 'phage. When the 'phage population in the cell has reached the "burst size" the cell ruptures liberating about 100 bacteriophages. This is known as "lysis." Bacteria may be "susceptible" or "resistant" to a given 'phage.

If susceptible, they may either be "lysed" or become "lysogenic" when brought in contact with the 'phage.

If they are lysed the bacterium is destroyed as described above.

If they become "lysogenic" then the DNA portion is taken up in the normal heredity replication of the cell components during

binary fission thus producing the hypothetical entity "prophage". About once in ten cell divisions a cell is produced which "lyses" with the liberation of 100 or more 'phage particles identical with the 'phage responsible for the prophage in the growing cells and capable of causing lysis or lysogenic qualities in susceptible bacteria. The 'phage responsible for the "prophage" state in a lysogenic bacterium is known as a "symbiotic" 'phage and a lysogenic strain of bacteria is a "carrier strain." There is, however, no virus particle present in the bacterium but only prophage, (i.e. the capacity to produce 'phage) and this prophage may be indefinitely retained on subculture or may be lost in a few generations.

If bacteria are "resistant" they may be either (1) non-receptive or (2) may neutralize the 'phage, or (3) the 'phage may be adsorbed giving rise to bacteria carrying "latent" or "temperate" 'phage ("symbiosis," "prophage," carrier strains not normally lysogenic).

D'Herelle noted in 1926 (a footnote on page 218 of his book "Le bacteriophage et son comportement) that toxicogenic strains of *Corynebacterium diphtheriae* were carrier strains. The toxic character of the diphtheria bacillus might be dependent on the presence of bacteriophage. Freeman (1951) found that a toxicogenic strain of C. *diphtheriae* could be obtained if cells from a susceptible non-toxicogenic (avirulent) strain were lysed by mixture with cells from a lysogenic, toxicogenic (virulent) strain and the resulting filtrate containing 'phage were transferred to a pure culture of the non-toxicogenic (avirulent) strain. The cells which were not "lysed" grew out and were now fully virulent toxicogenic and lysogenic being permanent hereditary carriers of the original 'phage (prophage). This has been named "transduction."

The dramatic change of C. *diphtheriae* cultures by 'phage induced mutation from avirulence to full virulence has been demonstrated *in vitro* and *in vivo* and in populations as small as 50 bacteria (Hewitt, 1954). Here a lysogenic 'phage, "symbiotic" in a virulent strain, lysed bacteria in a culture of non-virulent organisms, and being "dominant" the 'phage produced not only lysogenesis but also toxicogenic characteristics, in all the surviving bacteria of the strain in which it assumed the prophage state.

Transduction of genetic material by means of 'phage has also been demonstrated in strains of Salmonella (Lederberg, Lederberg, Zinder and Lively, 1951; Zinder and Lederberg, 1952; Stocker, Zinder and Lederberg, 1953). Here flagellar characters were transduced by 'phage but the same 'phage was capable of transducing only one or two of the many genetic characteristics of donor strains to a different bacterial strain and this only to approximately one in a million of the bacteria in which it had symbiotically attained the prophage state.

In Hewitt's work, however, the transducing bacteriophage showed "dominant" properties by means of which the whole population was rapidly homogenized to a toxicogenic strain. Consequently the *in vivo* test was highly successful. Mice injected intradermally with 0.2 ml of avirulent culture or with 0.2 ml of

purified 'phage, showed no reaction. Mice injected with 0.1 ml of avirulent culture followed by 0.1 ml of purified 'phage did, however, show a diphtheritic reaction followed by necrosis similar to that caused by injection of a fully virulent culture.

As continued subculturing of the culture possessing transduced virulent characters acquired by means of the symbiotic dominant 'phage did not alter its toxigenicity, the finding of Hewitt is one which must fundamentally affect the classical views on the transmission of bacterial infections.

Conditions which occur in the presence of large numbers of facultative, pathogenic, parasitic bacteria, may be transmitted by means of filtered lysates from one animal host to another. It is not essential that the virulent bacterial strain be transmitted but merely that the infected host shall carry lysogenic pathogens as well as "'phage-susceptible," avirulent bacteria in its body, and that the liberated 'phage material be transferred to a susceptible animal or person carrying "'phage-susceptible" avirulent bacteria.

The diseased conditions which would be most suited to the suggested circumstances are:-

(1) pneumonia.

(2) gastro-enteritis.

(3) vaginitis, metritis, urethritis, and epididymitis.

In view of the anatomical features of the genital tract it should receive primary consideration. Bacteria-free tissues of the tract are exposed to frequent mechanical invasion by commensals and facultative pathogens from the vagina and prepuce. *Corynebacterium pyogenes* frequently occurs in the genital tract of many species and non-pathogenic parasites are present in smaller or larger numbers near the vulva and prepuce. It may thus be expected that from time to time more pathogenic strains will occur and be transmitted from individual to individual during coitus. Also, as the result of coitus, pregnancy or parturition, a redistribution of infection involving the epididymis, urethra, cervix, uterus etc. is to be expected with opportunities for pathogenesis depending on the resistance, immunity or susceptibility of the host. Here the distribution of the more virulent or less virulent strains of bacteria is a matter of chance with frequent opportunities for mixing of populations and thus bacteriophage activity is favoured.

We may speculate that if a "dominant" 'phage which possessed the property of transducing pathogenic or toxicogenic properties were to be present, its transmission from the prepuce of a male to the vagina of a female could take place either by transfer of the lysogenic bacteria symbiotically carrying this 'phage, or *artificially by a filtrate entirely free from bacteria but containing the bacteriophage lysates of susceptible variants of the same species of bacterium.* The symptoms could develop in a matter of hours in the latter case because the infection is present and it is changed to a pathogenic infection in two to four hours.

When a male and a female are exclusively mated, only a temporary setback is fertility is to be expected. All susceptible

bacterial strains in the organs of both animals will be invaded by the 'phage and lysed or become lysogenic, symbiotic carriers of the dominant 'phage and will thus possess transduced pathogenicity. The strains which are resistant to the 'phage will not be affected. The pathogenic and transduced pathogenic strains will have been homogenized by 'phage infection and in due course immunity to them will develop in the infected hosts. This will terminate pathogenesis in the exclusively mated community of this male and female, but both will be carriers of a potentially pathogenic infection.

If a member of the exclusively mated community is mated to an extraneous male or female carrying avirulent strains of the same bacterial species which are susceptible to the dominant 'phage possessing virulence transducing properties, an infectious condition may be produced in a few hours. Unless time for stabilizing and immunization is allowed, the infection can be transmitted to other individuals by contact **or by transfer of filtrates containing 'phage but no bacteria** because a high 'phage titre may prevail in the blood or in serious or mucous fluids from the organs.

The highly speculative situation discussed in the above paragraphs is merely one example of the theoretical possibilities of bacterial mutation. It brings to mind the many aspects of transformation, gene location, chromosome alterations and loss mutation at present undergoing intensive investigation in many parts of the world. This may have a practical value in connection with the diseased conditions known as Contagious Infertility ("Epivag") and infections of *Vibrio foetus, Corynebacterium pyogenes, Streptococcus* and *Micrococcus*.

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INTUSSUSCEPTION IN THE DOG WITH A CASE OF RECURRENT, PROLONGED BOWEL INVAGINATION.

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SUMMARY

(1) Intussusception in the dog is discussed and some of the literature is briefly reviewed.

(2) A case is reported where a dog had intussusception twice in rapid succession. In the first instance it had the condition for about 14 days before successful bowel anastomosis, and in the second instance for about 7 days until relieved surgically.

(3) A suggestion is advanced as to why bowel gangrene did not supervene.

Intussusception in the dog is not a very common condition in the author's experience. It usually occurs in puppies under 6 months of age and is invariably associated with hyperperistalsis and diarrhoea. What might be regarded as a form of intusussception rectal prolapse, occurs more often and has a somewhat wider age distribution. As the bowel contents of the carnivore are much more liable to produce septic infection than those of the omnivore or herbivore, it is generally and quite rightly regarded that any condition involving obstruction to the flow of blood or chyle in the small intestine, leads to much earlier and more serious sequelae in the meat eaters than in the others. It will be shown that this rule is not without exception.

Review of the Literature:

Representative opinions of various authors in different countries were taken. Lacroix (1952) mentions the relationship between enteritis and intussusception in the dog. In this his opinion is strengthened by Kirk (1939) and Jakob (1924), while the following authors specifically mention hyperperistalsis: Fox and Fincher (1956) in cattle, Hutyra, Marek and Manninger (1938) and Webster (1935) in large domestic animals.

Kirk (1939) stresses the frequent association between rectal prolapse and intussusception, while Jakob (1924) makes a similar observation and reports multiple simultaneous intussusceptions in the dog. Hutyra et al. (1938) report the highest incidence in young dogs and regard the site as usually the small intestine, less often the ileo-caecal valve.

Brumley (1943) advises laparotomy posterior to the umbilicus in the dog and regards the prognosis as unfavourable in all cases not operated upon early. Kirk (1939) emphasizes the necessity for early diagnosis and Jacob (1924) regards bowel resection as

essential. In cattle Udall (1943) states the course to be six to eight days, with recovery possible up to the fourth day if the case is operated upon. Fox and Fincher (1956) agree in that they say that surgery up to the fourth or fifth day may be successful. Finally Hutyra et al. (1938) are of the opinion that in the horse with caecal intussusception the course is six to nine days and if it is caeco-colic six weeks or even months.

It is thus clear that, with the exception of the last mentioned instance, all the authors regard early intervention as conditional for a successful outcome. While this assertion must remain true in the great majority of cases, it can be challenged occasionally.

Subject:

The subject was a five months old Alsatian puppy dog.

Anamnesis:

The pup had been under treatment elsewhere without success for about a week for enteritis. When seen by the author he was in a state of dehydration and rather weak. As he did not respond to treatment at home, he was hospitalized for more complete examination and therapy. After seven days he was discharged, having been treated for coccidiosis. His state was still weak, but he appeared to be on the road to recovery. Fifteen days afterwards he was readmitted, suffering from severe tonsillitis. The stools were very scanty. The owner stated that he appeared to progress for a day or two after discharge and then refused all solids, with the result that he was in extremely poor condition. He was observed to vomit occasionally.

Diagnosis:

A tender sausage shaped mass could be palpated in the epigastrium. Intussusception was diagnosed. The existence of infected tonsils was confirmed.

Treatment:

Penicillin and B complex vitamins were injected and glucose saline infusions were given. Operation was advised and agreed to.

Operation:

After the usual preparation Sagatal (M & B) anaesthesia was induced and a 6 cm incision made along the linea alba from the umbilicus forward. This site was regarded as the best as the ileo-caecal valve lies 2-5 cm in front of the navel and just to the right, so that all the small intestine could be brought under vision, including the less mobile portions. The intussusception was found in the ileum, the ring of the intussuscipiens being about 15 cm from the ileocaecal valve. The length of the intussusceptum was also about 15 cm with the result that the end was nearly in contact with the valve. The intussusception felt hard. Reduction of the invagination proved slow and difficult and was accomplished by gentle traction on the intact bowel and massage and pressure in an antiperistaltic direction. Each part of the intussuscipiens coming to light was very hard, rigid and friable

and numerous fissures down to the submucosa developed during the straightening process. Fortunately the mucosa remained intact. Even though the raw areas were likely to cause adhesions, it was felt that the condition of the patient was too precarious to risk the additional shock and operating time entailed by an enterectomy. A side to side anastomosis, short circuiting the affected bowel and employing a cutting suture, was therefore carried out. Later reference to the cutting suture will be made.

The usual anti-shock measures were employed and streptomycin was injected in case oozing should take place through the cracks in the bowel wall. The dog rallied surprisingly well and was discharged on the 10th post-operative day, having gained a great deal of weight.

Fourteen days afterwards the case was readmitted with the history that he had been doing well until one week before, when he refused food and occasionally passed mucoid faecal material.

An examination showed that the dog was emaciated to a fantastic extent, it being remarkable that is could walk. Another sausage shaped swelling was palpated in the epigastrium and intussusception again diagnosed. Operation was advised but the prognosis given as extremely serious.

The original wound was reopened and the intussusception found in the middle part of the jejunum. The anastomosis previously carried out was found to be functioning normally and the adhesions much less than anticipated. The same manoeuvres were carried out as previously. As the bowel was not as hard and friable as before, the straightening process was easier and fewer fissures developed. Side to side bowel anastomosis without resection was again done, the use of the cutting suture being repeated.

The case again rallied remarkably well so that it could be discharged from hospital as early as the seventh day. In spite of all its vicissitudes it has developed into a fine dog.

Remarks:

From the history it appears fairly definite that the second intussusception lasted seven days before operation. Judging from the degree of alteration of the wall of the relieved intussuscipiens, the first intussusception must have lasted about 14 days. What there is of the rather vague history pertaining to the first episode, supports this statement.

The changes in the bowel wall can only be explained in terms of a marked attenuation of the blood supply, which was sufficient, however, to prevent gangrene.

Attention is directed to the gratifying results attendant on the use of the cutting suture in aseptic side to side bowel anastomosis. This method is recommended, but will not be described here as it has adequately been referred to elsewhere e.g. Markowitz (1937) in reverse Eck fistula, Hofmeyr (1953) in cholecystoduedenostomy in the dog and Hofmeyr (1955) in cholecystoduodenostomy in the cow.

Conclusions:

This case is remarkable in that it has twice survived intussusception which lasted longer than many authorities would regard

as possible in the dog. At the first occurence the patient was in poor condition and it was worse the second time. Now, in the emaciated subject the bowel wall is naturally thinner than when the animal is in good condition. As the outside diameter of the bowel will remain largely the same, it follows that the lumen will be wider in the thin animal. Here, too, the mesentery will be devoid of fat and more delicate. Thus the more emaciated the animal the easier it would be for the bowel to invaginate into itself and the less the pressure would be on the blood vessels of the incarcerated part.

It is therefore tentatively concluded that survival time after intussusception is unfavourably influenced by good bodily condition in the case, or that the emaciated case is in less immediate danger of bowel gangrene in these circumstances than a fat one. This may be a guide to surgeons when determining the prognosis, a further consideration being that in the fat case enterectomy may be obligatory and in the thin one avoidable.

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THE USE OF THE BRUCELLA MILK RING TEST (M.R.T.) IN PRACTICE

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Kenya.

Specific diseases of the genital tract of cattle in Kenya known to take their toll are Brucellosis, Trichomonas foetus infection, 'Epivag' (Infectious epididymitis and cervico-vaginitis),[1,2] post-parturient metritis and vaginitis, and ampullae and prostatic abscesses caused by Corynebacterium pyogenes. Diseases such as Rift Valley Fever and Foot and Mouth disease leave behind them a trail of abortions and infertility. It was felt that the control of all these infections was impossible under existing conditions of poor fencing, illicit movement of stock, sales without certification and difficulty of diagnosis. The control of Contagious Abortion, however, for which there are good diagnostic aids, e.g.

(a) blood-serum agglutination test;

(b) cultural examination and direct smear examination;

(c) biological test;

(d) Milk Ring test and an efficient vaccine, was considered feasible and a step forward in the control of the severe annual losses from these diseases.

In the control of any disease a rapid and efficient diagnosis is essential; for Brucellosis the Milk Ring test seemed to fulfil these requirements.

Over the last three years, apart from initial surveys and group tests, 2078 individual animals from 93 farms in this district have been tested by the M.R.T. Of these 432 were positive from 31 farms i.e. 20% of infected animals and 33% infected farms. As there are approximately 600 farms in the district these figures give a reasonable indication of the incidence of Contagious Abortion. A Veterinary Department survey of samples from creameries in 1956 gave an overall infection rate of 19%.

Materials and Methods:

Whenever a farmer reported an abortion he was asked to submit a sample of milk from the affected cow not less than seven days following the abortion (a squirt from each teat into a clean tube taken at morning milking). If the foetus or membranes were available smears were made and stained with Hansen's technique[3] and examined for the presence of Brucella, Vibrio and Trichomonas organisms. On reception the milk sample would be well shaken and set out in 1 ml quantities in agglutination tubes, approx. 8 mm. diameter, as follows:- undiluted; 1/10; 1/100 in negative creamy milk, plus a negative control. A positive control was included when available. We are at present making arrangements to obtain

a supply of dried positive control milk for routine use. To each tube 1 drop (approx. 0.05 ml) of haematoxylin-stained Brucella antigen was added and the tubes shaken to ensure thorough mixing. The tubes were incubated in a hot-air incubator at 37°C for a minimum of one hour, usually three to four hours. If the incubator was not functioning the tubes were stood at room temperature ±64°F for four hours. A purple cream layer and white milk in the undiluted and 1/10 tubes was regarded as positive. Positive undiluted and negative 1/10 was regarded as doubtful and re-tested one week later.

In all herds where a positive was obtained for the first time a serum sample was sent to the Veterinary Laboratories at Kabete for confirmation. Two tests only have differed; one was a sample of colostral milk which gave a positive M.R.T. and negative serum agglutination. We have been unable to follow up this case. The other was an aborter from an infected herd (one of us — P.W.T. — contracted Brucellosis from an animal in this particular herd) that had aborted two months prior to the test, the M.R.T. was negative where a positive was expected as smears from the foetal membranes and stomach at the time of abortion had shown the presence of numerous Brucella-like organisms. Following the M.R.T. a serum agglutination test was positive and a second M.R.T. was again negative.

Whenever a positive reaction was encountered the herd, if over 50, would be grouped into tens; if under 50, into fives, and screened, using bulk milks undiluted. Any group giving a positive reaction was then individually tested and thus all infected animals identified for isolation at calving and/or culling. The value of this initial screening in time, trouble and money to the farmer cannot be sufficiently stressed.

The differential test as described by van Drimmelen[4] for identifying reactors from vaccinates has been of great use in infected herds that have been using S. 19 vaccine as a routine for a few years. In a number of these established herds with fairly high-grade and pedigree stock the test was used to certify animals for sale and carriers for culling.

The haematoxylin-stained antigen obtained from Onderstepoort has given the best results. The Tetrazolium-stained antigen (samples from Onderstepoort and Kabete) was found to be less stable and frequently confused the test by staining the milk. In a number of tests the cream layer was only slightly coloured, but between the cream and the milk was a fine, very dark purple ring, which on microscopical examination was composed of antigen and a few small fat globules. This phenomenon is mentioned by Oganowski[5] as being of common occurence with goats' milk and is apparently related to the size of the fat globules. Of interest in a number of strongly positive milks (titres up to 1/200) from different breeds was the rapidity of creaming which, both at room temperature and incubated, was almost complete in 7 to 10 minutes.

When serum samples were sent to Kabete for initial confirmation a split sample was tested by the rapid slide agglutination method using the milk test antigen. Dilutions were, serum 1/10,

1/50, 1/100: agglutination at 1/50 in less than 2 minutes was regarded as positive. All these parallel tests agreed.

One of us (P.W.T.) diagnosed Brucellosis in himself by means of this rapid serum agglutination test. Initial titre 1/200, ten days later 1/5000 and a month later a serum sample was sent to the Medical Laboratories at Nairobi and gave a titre of $\pm$1/30,000. Of interest to fellow sufferers is that treatment with Aureomycin and Sulphamezathine orally has, apparently, proved effective, with no relapses after two years.

The highest serum titre encountered was 1/8000 in an infected cow five months after abortion and the highest M.R.T. titre 1/700, four months after abortion, not from the same animal.

Discussion:

From our results and reports from elsewhere it appears that the M.R.T. is equally as good as the serum agglutination test for initial diagnosis and a very much simpler and speedier process. The technique is simple and the apparatus, other than an incubator, or water-bath which are not necessities, available to every practitioner and field veterinary officer. After an initial diagnosis confirmed by a serological test the screening of herds and regular testing by the M.R.T. method is an invaluable aid in controlling and ridding the cattle population of Brucellosis.

The history of the disease in the herds tested so far has been that of occasional abortions, except for three herds in which there were abortion storms. In one herd the disease was suspected, in the absence of recorded abortions, upon seeing a cow with a carpal synovitis the size of a football.

One of our main difficulties in the control of contagious abortion is that farmers do not realise and appreciate the significance of infection. An example is a herd of high-grade and pedigree stock, using imported bulls, where the disease was diagnosed two and a half years ago; the farmer has made no effort to have the herd tested, or vaccinated, in spite of continual warnings and urgings. Again here, to date, there have been only the odd three or four abortions each year in 90 to 100 animals; however there are a number of animals in the herd that breed irregularly and retain the foetal membranes.

Also there is as yet no implementation of any public health ordinance requiring the supply of clean milk to townships in Kenya. As a result of this, Brucellosis is not unknown amongst the human population.

It is of peculiar interest that a country where the basic economy rests on agriculture and stock the role of the veterinarian in stock management, production and public health related to stock, is largely overlooked. A Dairy Board was recently set up in Kenya, under the aegis of the Ministry of Agriculture, to study the whole matter of production, supply and distribution of dairy products; no reference was made to the Department of Veterinary Services, nor was any veterinarian invited to sit upon, or advise the Board. This ignorance of the wide field covered by the profession, as opposed to the purely clinical side foremost in the lay mind is,

in many cases, a stumbling block in the way of disease control and better production.

In all herds, except two, where contagious abortion has been diagnosed, vigorous steps have been taken by screening tests for reactors, culling and vaccination, to control and eradicate the disease. Until such time, however, as the country realises the significance of Brucellosis, both to stock and the public health, and a scheme can be started to test throughout each district, the disease will remain a problem. To our minds the development of the M.R.T. has been of the utmost significance in the fight against Brucellosis and it is hoped that it will be more widely used than is presently apparent.

Summary:

Over the past three years 2078 animals from 93 farms have been tested by the Milk Ring test. Of these, 20% of these were positive and showed 33% of the farms tested to be infected.

The differential test of van Drimmelen for identifying reactors from vaccinates was found useful in vaccinated herds and the haematoxylin-stained antigen from Onderstepoort was found to give more reliable results than the Tetrazolium-stained antigen from Onderstepoort and Kabete. This milk-test antigen was also found useful for serum samples tested by the Rapid-Slide Agglutination method.

The writers believe that the M.R.T. has been of the utmost significance in the fight against Contagious Abortion in this district and could be more widely used.

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THE SPAYING OF HEIFERS

F. B. W. DUCASSE

Dargle, Natal.

The spaying of heifers is becoming increasingly popular amongst farmers in this area, and the aim of this article is to record some practical details which simplify the operation for the veterinarian.

The author has performed many hundreds of spayings over the past three years and contends that the operation is both simple and safe. Experimentation on heifers of various ages from four to 27 months has convinced him that the ideal age for spaying beef heifers is seven to eight months i.e. about one month before they are weaned. They are more easily handled at this age, and as they are still suckling, they appear to recover from the operation far more rapidly than older heifers. Apart from this, the majority of beef animals in this area are weaned in late May or early June, and hence the spaying is done in May when the practitioner can rely on fine weather and there is little or no danger of blow-fly strike.

The very first essential for success, in the author's opinion, is a special crush-pen. This must be substantially constructed (*not* creosoted poles) and the internal width should be 18 inches. It should be long enough to hold ten heifers at once, and the side poles must be fairly close together for about two and a half feet from the ground, otherwise the heifers are continually getting their legs between the poles, resulting in an excessive waste of time rectifying the position. As these young animals are very prone to jump, the side poles must continue for another two feet above this i.e. one pole at three and a half feet and another at four and a half feet from the ground. These top two poles on the left side of the crush should be "slide-through" ones which can easily be slipped out once the animals are in the crush, allowing ease of access to the left flank of the animal. The front of the crush should be preferably closed with a substantial gate, and it is a great advantage to have a "V" stanchion in the centre of the gate to secure the animals head. Two upright poles should be embedded close together on either side of the crush-pen, about four feet from the front, to allow poles to be pushed through from side to side, to prevent the front animal from moving backwards, or the other animals from pushing forwards, and interfering with the operation.

It is preferable to lock the heifers up, with their dams, in a small enclosure the evening before the operation — the heifers then, being semi-starved, are not so difficult to handle, and a half-empty rumen facilitates the operation.

The first ten heifers are brought into the crush, and the front five are then clipped and shaved over the left flank (a native is shown how to do this). Commencing from the front, these five animals are anaesthetised locally over a vertical area about four inches in length and about one inch wide behind the last rib, and commencing as high as possible. Approximately 20cc of 5% Planocaine (M & B) per heifer are used, and by the time the last animal has been anaesthetised the first is ready for spaying.

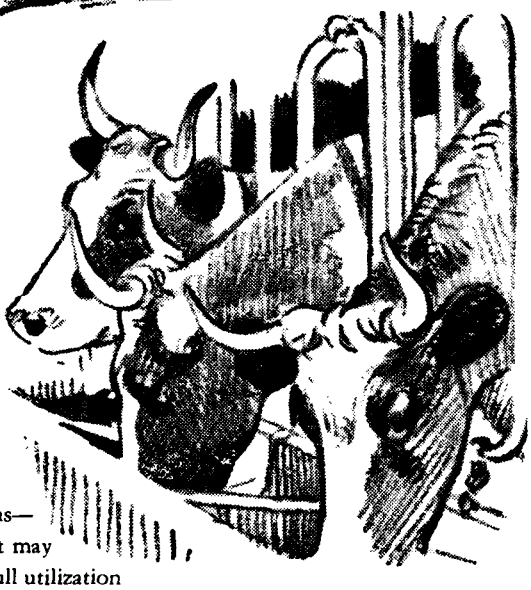
No restraint whatsoever is found necessary during the operation, particularly if the head is secured in a stanchion in the gate, except in the case of very wild heifers, when holding of the nose usually suffices (and here it is found that the fingers are infinitely preferable to a nose-holder as the animals often struggle with the latter, and their bellowing distends their abdomens and renders the operation far more difficult). The shaved area is well cleansed with methylated spirits, as are the operators hands and arms: laparotomy is then performed making an incision about four inches in length, just sufficient to admit the operator's arm, which is then in inserted and the first ovary recognised and grasped. A long handled spaying forceps is then introduced, and the ovary severed as close to its substance as possible, the forceps being left on for about half a minute. The operation is repeated on the second ovary. Struggling on the part of the heifer may occur during the handling and severing of the ovary, and it is advisable to have an assistant standing by to control the animal, if necessary, during this stage of the operation. The wound is then sutured, using No. 3 Chromic Gut throughout, the peritoneum and muscles are sutured in one operation, and dusted with an antibiotic dusting powder. Finally the skin incision is closed, using two mattress sutures, and then treated with antibiotic dusting powder. This heifer is then released and next one brought into the front of the crush or secured — and so on until all five have been spayed. During the course of these operations the remaining five heifers will have been shaved by the assistant, and be ready for anaesthetising. By this means, it is very easy to spay four heifers per hour, and, with experience, this can easily be increased to five.

The instruments are kept in a tray of methylated spirits during the operations and sterilized by boiling after every ten spayings (a Primus stove is carried for this purpose).

Generally no after-treatment of the wound is required, but the animals must be left undisturbed in a shady place, with food and water available, for the next two or three days. They usually lie down shortly after the operation and are disinclined to move, unless disturbed, for about 24 hours; thereafter the majority commence to suckle or graze and recovery is uneventful.

Out of approximately two hundred heifers of all ages, which the author has spayed, and been able to check up on subsequently, his losses have been one animal — a half per cent. On post mortem this heifer showed miliary abscessation throughout, and as death took place eight days after the operation, it is felt that this condition had probably been present prior to the operation.

The fees charged for the operation are £3. 3. 0. per hour plus drugs and mileage. The majority of farmers were hesitant at the beginning as it was impossible to estimate the number of animals that could be spayed in an hour, and they were loath to pay more than £1. 10. 0. per animal. However, spaying at the rate of four heifers per hour, plus drugs which amount to 5/- per animal, works out at a total of £1. 1. 0. per animal, plus mileage charges. As a general rule a practitioner can comfortably spay 25 heifers per day, giving him a nett return of £20.0.0 — sufficient, it is felt, to warrant the interest of the majority of private practitioners in the beef producing areas.



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ABSTRACT

TUBERCULOSIS IN DOGS

J. H. MASON

Johannesburg

Olav A. Berg (1956) — Acta Tub. Scand, 32, 351-361.

The frequency as reported in the literature is given as between 0.04% at clinical examination and between 0.03% and 5.6% at autopsy. Among 1750 canine patients seen at the Veterinary College of Norway during the years 1949 to 1953, eight cases of tuberculosis were detected (0.46%).

The human type of *Mycobacterium tuberculosis* was responsible for at least two-thirds of all cases where typing was carried out. Human excretors of the microbe would appear to be the chief source of canine infection.

The dog is relatively resistant to infection as proved by the large number of microbes necessary to produce the disease experimentally and by the relatively few cases seen among dogs living with tuberculous owners.

The disease in dogs is usually chronic and insidious, and significant clinical symptoms are not seen until the disease is far advanced. The primary complex develops in the lungs or intestines. In the lungs the infection develops in a subacute way giving rise to broncho-pneumonia often accompanied by exudative pleurisy. Generalized tuberculosis has been described, although it is less frequent than that of individual organs.

Histologically, the picture is less characteristic than that seen in other animals; giant cells are seldom found.

The history and post-mortem findings in the eight cases seen at the Veterinary College of Norway are given in more detail. All but one animal was five years of age or more, five breeds were represented, five were males and three were females.

The history common to all but one was loss in weight. In four of six animals, neutrophilia and an increased sedimentation rate were observed. In six cases, the intracutaneous tuberculin test was carried out — three positive, one doubtful and two negative.

At post-mortem examination, generalized infection was found in four animals, abdominal lesions in three, thoracic lesions in one. In two cases, *M. tuberculosis* was detected during life (once in expectorate and once in urine).

The human type of microbe was isolated in the seven cases where typing was carried out.

One animal was probably infected from its owner; the source of infection could not be traced in the others.

Forty-two references are given.

OBITUARY

Ll. E. W. BEVAN, M.R.C.V.S.

The recent death of Mr. Llewellyn E. W. Bevan represents the passing of the founder of veterinary research in Southern Rhodesia.

'Llew' Bevan, as he was popularly known, qualified in London in 1904 and came out to Southern Rhodesia in 1905 as a Government Veterinary Surgeon. His enquiring mind soon led to his being diverted from field duties to serve as Government Veterinary Bacteriologist, a post in which, single handed, he commenced his long research career. As a result of his tireless efforts a separate Department of Veterinary Research was created in 1922 of which he was appointed Director, a position which he held until he retired at the age of 55 in 1933.

During his twenty eight years of Government Service he devoted as much of his energy as could be spared from routine diagnostic work to investigating the multiplicity of diseases about which so little was then known. His keen observations as well as critical experiments contributed greatly to our present knowledge of many diseases and the development of diagnostic techniques and vaccines for their control. His early work is extensively quoted in the literature, particularly in connection with horse-sickness, brucellosis, ephemeral fever and trypanosomiasis.

After his retirement in England he returned to Southern Rhodesia for a period of three years as Beit Research Worker in trypanosomiasis, during which he continued his research into a method of conferring immunity. Even in his final retirement at the Cape he maintained a keen and active interest in the welfare of the animal industry in Southern Rhodesia, frequently delighting, as of old, in writing stimulating articles to the farming press.

To his widow and two daughters our sympathies are extended.

D. A. L.

OBITUARY

CLAUDE JAMESON CONNOCK

Supervising Stock Inspector.

The news of the death of Mr. C. J. Connock, at Ermelo on March 21, 1957, after a comparatively short illness, was received with deep regret.

To his wife, son and two daughters, we extend our heartfelt sympathy in their very sad loss.

Claude Jameson Connock was born on January 14, 1896, at Johannesburg.

Here, at Marists Brothers College, he received his education.

Starting work at Premier Mine in March 1913, he soon progressed to the post of mine overseer. In February 1914 he joined the British South African Police and served with that force until October, 1919, when he resigned to rejoin the Premier Diamond Mining Coy. Ltd.

Just at that time, one of the most extensive outbreaks of East Coast Fever, to be recorded in the union, was raging in the Pretoria District, and in November 1920 Mr. Connock was recruited into the service of the Veterinary Division, where he remained until his death.

He was soon elevated to the post of Dipping Supervisor in the Pretoria area and later received promotion to that of Stock Inspector.

On July 28, 1924, he married Miss Leonora Botha of Ermelo.

In 1928 he was selected to fill a newly created post of Check Inspector and in that year was transferred to Dundee. There are now eighteen posts of Check Inspector (Supervising Stock Inspector) in the Division. These extensions were very largely justified through the success which Mr. Connock made of the initial post.

Supervising Stock Inspector Connock set a very high standard of diligence and application to duty for his colleagues and subordinates to follow. He raised the degree of efficiency among Stock Inspectors to a very high level. No single inspector in the Division made a greater contribution to the control of East Coast Fever, than did Mr. Connock. He had an enormous capacity for work and possessed an intimate knowledge of the routine control measures connected with the major epizootics which occur in South Africa.

He was never satisfied until he had traced an outbreak of disease to its original source and in this sphere of the work he was particularly brilliant. He lived for his work and for years his caravan was his home. Nothing was ever too much trouble for him. He served the public fairly, firmly and completely without prejudice, remaining calm even under the most exacting circumstances. He frequently acted for State Veterinarians in an administrative capacity but never failed to appreciate their professional superiority. Only those of us who had the pleasure and honour of serving with him, can appreciate the contribution he made towards the control of animal diseases.

His principles, his steadfastness, his fearlessness and his boundless energy will live in our memories for many years to come. He was a man in every sense of the word. We deeply regret his passing. We honour the work he did and the examples he set.

For us there will never be another "Connock."

A.M.D.

BLOOD GROUPING AND ITS UTILISATION IN CATTLE BREEDING

by

D. R. OSTERHOFF

Onderstepoort

The study of the blood groups of our domestic animals has made much progress during the last 30 years and in the past ten years the bearing of the results of this research on practical breeding has become more significant.

For the sake of simplicity, we shall only deal in this paper with the blood groups of cattle and their practical utilisation. That an animal belongs to a certain blood group means that its red blood corpuscles possess a characteristic antigenic structure. When red blood corpuscles of one animal are inoculated into another the recipient produces antibodies to all antigens which it does not carry at the surface of its own blood corpuscles. Since the reaction is relatively weak, the transfusion must be repeated several times in the course of 3-5 weeks in order to produce a high level of antibodies. Apart from the antibodies produced by these blood transfusions (immunizations) normal (natural) antibodies also exist. Antibody containing sera are usually called antisera and when these, after absorption, have become specific in such a way that they react only to a single blood corpuscle antigen, they are called reagents or test sera.

The reaction taking place between blood corpuscle antigen and antibody is usually an agglutination or a haemolysis. Agglutination means a clumping of red blood corpuscles, which in most cases, can be observed with the naked eye without technical aids. In haemolysis, the red blood corpuscles disintegrate, liberating haemoglobin into the suspending fluid, which becomes distinctly red in colour.

Fig. 1 shows the diagrammatic representation of the production of test sera and the haemolysis reaction. Three different antigens are present in cow No. 1 from which the erythrocytes are inoculated into cow No. 2 which possesses antigen A but no antigens B and C. Antibodies to the latter antigens are formed. To the serum of this cow erythrocytes are added from a cow possessing only the antigens A and C. Antigen C binds specific antibodies. By means of centrifugalization, the erythrocytes are separated from the serum, which then contains only antibodies to B. If to this test serum erythrocytes are added which have antigen B and as complement, fresh rabbit serum is added as well, the erythrocytes will be haemolysed and the typical reaction observed.

In a series of fundamental investigations, Ferguson, Stormont and Irwin of the University of Wisconsin, U.S.A., have

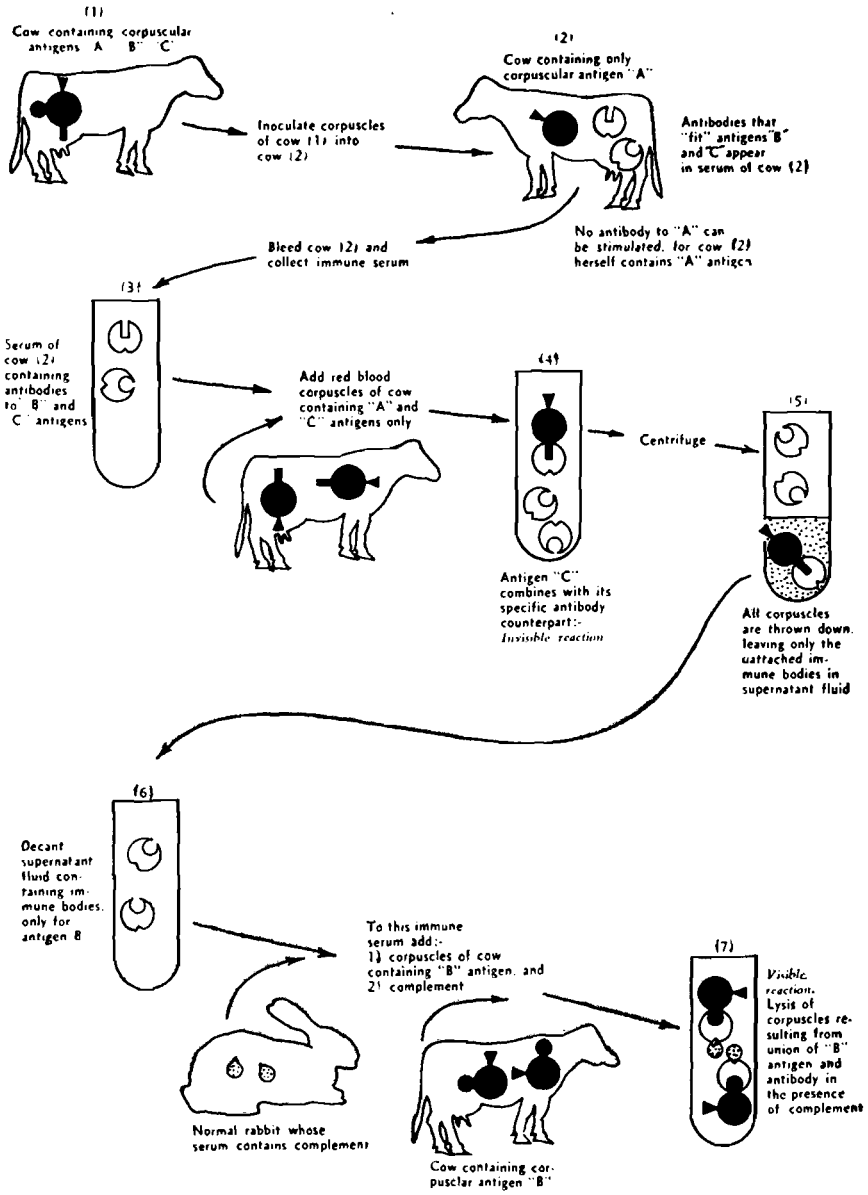
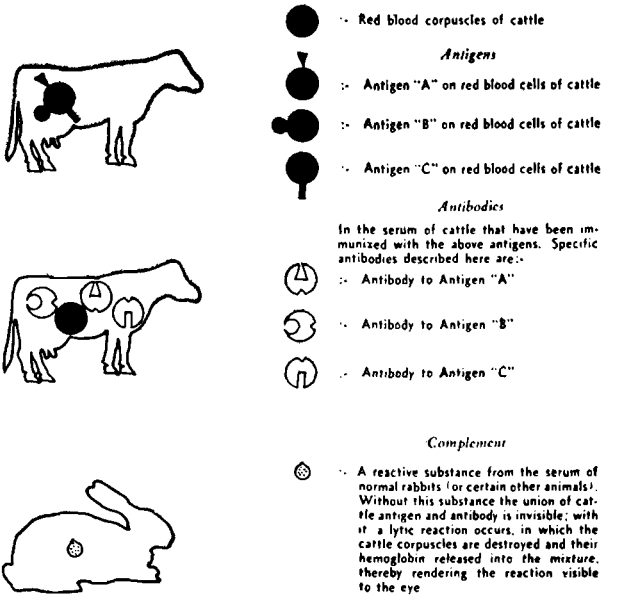


Fig. 1

Diagrammatic representation of the production of immune sera and the establishment of the presence of a specific antigen. (After Stormont and Cumley).

identified no less than 40 unit factors, determining individual differences in the erythrocytes in the blood of cattle.

As in the case of human beings, the blood groups are designated by letters following the alphabet in the order in which they were established, i.e. A, B, C, etc. When more blood groups were



Explanation of Fig. 1.

found the designations A', B', C', etc. were given. It must be pointed out that no relationship exists between A and A', or B and B'; the similar designation is incidental. The J factor is noteworthy in that there is a definite similarity between it and the A. antibodies of humans.

Inheritance of Antigens

In the past it was believed that all factors were inherited independently of one another and that each factor was bound to a specific gene, and was passed with this gene from one generation to the next. This supposition has proved to be incorrect. Stormont was able to prove, by studies of the progeny of a number of bulls, that certain factors are inherited in groups or complexes. So for example no less than 21 factors, which may be present in different combinations belong to the B-complex. The C-complex contains 7 factors while the M, L and H' systems each contain only one factor which may be present or absent. A total of 10 such groups or complexes has been found which can be inherited separately. With a view to representing the inheritance for these antigens in a simple way, the manner of inheritance for three antigens only is demonstrated:

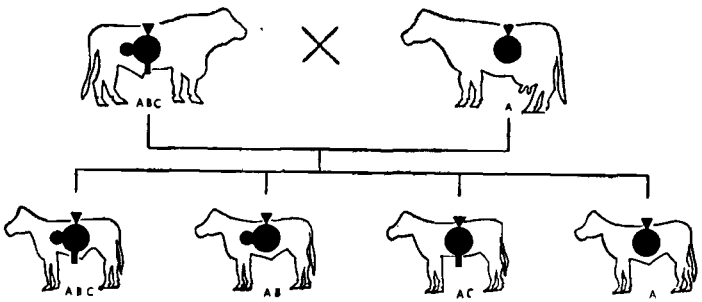


Fig. 2

Inheritance of Antigens.

Fig. 2 illustrates the inheritance of antigens A, B and C in the offspring of a bull having all three antigens, mated to a cow having only one antigen A. The four possible types of offspring from this mating are shown in the lower line. If an antigen appears in the offspring, it must be present in one or both parents.

PRACTICAL APPLICATION OF BLOOD GROUP TESTS WITH CATTLE.

As a result of intensive research it is now possible to exploit the results obtained for the following purposes:-

(1) To prove identity.
(2) To prove parentage.
(3) To prove twins.

Proof of Identity.

Because of the large numer of determinable blood group factors in cattle, the number of possible combinations is considerable. More than ten million genotypical combinations may occur and it is conceivable that the blood test will develop into as reliable an index of individuality as finger prints in man.

Since the animal retains its typical blood combination throughout life, once its blood group structure has been established, it can always be traced by means of a blood group test.

Proof of Parentage.

By means of blood group tests the parentage of a calf can be established. Usually these tests are used to ascertain which of two bulls, sired a particular calf. For this purpose, the blood group factors of the calf, the dam and the two bulls are determined. The factors in the calf, which were not derived from the dam, (since they are absent in the dam), must have been inherited from the sire. The bull which has the remaining blood group factors of the calf, must be the sire.

The basic principle has been observed to be true, in all tests made on animals of known parentage, that an individual possesses a cellular antigen only if one parent or both possess it. An example follows of the use of the blood group test in solving a case in which the dam of the calf was known, but in which either of two bulls could have been the sire.

	Blood Types																			
Possible sire, bull No. 1	A	B		F		J	O				X_2		Z			I'		L'		
Possible sire, bull No. 2	A	B	C_1	F		J	O	R	S	VW	X_2	Y_1	Y_2	Z	A'E'$_3$	H'		J'K'		
Dam	A			F	H	J	O			V	X_2					H'		J'K'	L'	
Calf	A				H		O	R		VW	X_2		Y_2	Z		H'		J'K'	L'	

It will be noted that the calf in question carries cellular characters R, W, Y_2 and Z which were not possessed by the dam, and which must have been inherited from the sire. Both possible sires possess Z, but only bull No. 2 carries R, W and Y_2. Therefore bull No. 1 can be excluded as the sire of the calf, and if either of two bulls is the correct sire, it is No. 2.

At the Swedish blood group laboratory, 105 cases of dubious parentage were investigated during 1956. With all the necessary data available i.e. blood samples of the possible sires, the dam and the calf, 89.3% of these cases were solved. Where the data was incomplete i.e. blood samples were not available of the dam or of one of the possible sires, because these animals had died, only 28.6% could be solved. Some of the incomplete cases were solved by studying the inheritance of the different factors separately.

As artificial insemination becomes more generally employed this branch of heredity research will become progressively more important. The records of the "Section of Infertility and A.I.," at Onderstepoort, show that 40% of all inseminated cows fail to hold and have to be inseminated again. The insemination records also show that only in a few cases is semen from the same bull used for the repeat insemination. Usually, pregnancy is attributed to the last insemination. Extensive blood group tests have shown, however, that this supposition is not justified, because of the marked variation in the length of the gestation period of the cows. The following table shows these variations in the time of pregnancy, according to Bisschop.

Breed	Number of animals	Length of pregnancy, days Min.	Max.	Average
Afrikaner	566	283	299	291
Friesland	573	271	284	278
Red Poll	510	278	293	285
Sussex	566	276	289	282

If an animal is inseminated in two successive periods of oestrus with semen of two different bulls (or is served in the natural way), we have to take into account, as a matter of course, the whole range of variations in both cases. To what extent the two variation curves will overlap depends on the duration of the periods of oestrus of the different breeds. The following table shows the variations in the periods of oestrus for the breeds mentioned (according to Bisschop).

Breed	Duration of Ovarian Cycle, days		
	Min.	Max.	Average
Afrikaner	19.1	21.9	20.2
Friesland	17.6	21.4	19.8
Red Poll	19.1	21.0	19.8
Sussex	18.8	23.0	20.0

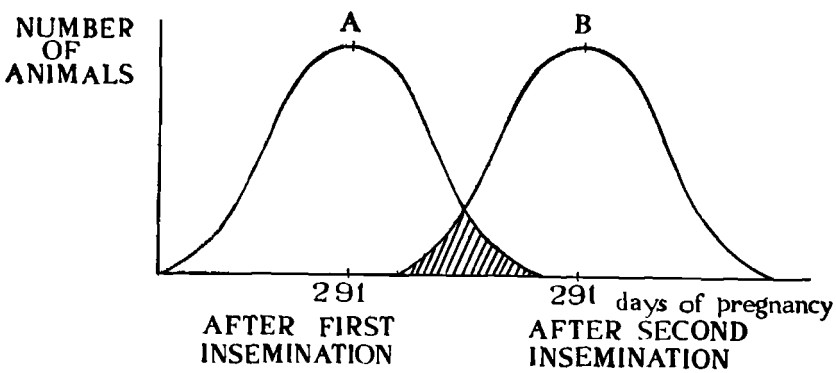


Fig. 3
Variation of the period of pregnancy of cattle.
Diagrammatic representation in the case of the Afrikaner.

The distance between A — B gives the interval between two successive inseminations (or services). The closer B is to A, that is, the sooner the second insemination takes place, the larger will be the shaded surface when the two curves overlap. The size of the shaded surface indicates what percentage of all cases of once repeated insemination is dubious in respect of parentage. One must bear in mind the variation in length of pregnancy after the first as well as after the second insemination. With normal variation in the duration of pregnancy, the relationship between the interval of serving and percentage overlapping is as follows (see fig. 3).

Insemination/service interval in days	Percentage overlapping.
15	16.51
18	9.49
19	7.84
20	6.19
21	4.55
24	2.64

The shorter the interval of insemination, the more we must have recourse to blood group tests. From the normal curve, it is possible to calculate directly the probability of the first or the second insemination having been successful. If, for example, an Afrikaner cow calves four days before the calculated average parturition date for the second insemination, it is certain in 97% of cases that the second insemination was the successful one. If one calves nine or ten days before the calculated average parturition date for the second insemination, it is certain in only 50% of cases that the second insemination was the successful one. If the parturition is 16 days before the calculated average date for the second insemination, it is certain in 98% of cases that the first insemination was the successful one.

These facts have led the Canadian Breeders Association of the Holstein-Friesian breed to incorporate the following rules in their constitution. If an animal is to be registered, a certificate of parentage is required in the following cases:-

(1) When a cow has been served by several bulls or has been inseminated several times within 10 days.

(2) When a cow has been served by different bulls or has been inseminated with semen of different bulls in two successive periods of oestrus, and calves four or more days before the calculated average parturition date in respect of the last service of insemination.

As regards parentage tests, it may be said that blood group tests can be used for ascertaining the most favourable time of insemination. Sires are chosen with the largest differences in blood groups and insemination is effected in an oestrus period at definite intervals. The blood group type of the calf will indicate to which insemination fertilization can be attributed, that is, which time was the most favourable for insemination.

Ascertaining of twins.

More and more use is made in different countries of monozygous twins for experimental purposes because they are fully identical and possess the same hereditary characteristics. Since the phenotypical differences between monozygous and dizygous twins are often difficult to observe, blood group tests are the only reliable tests for monozygosity. If the twins are identical in their external appearance and if they possess a fully identical type of blood, they must be considered to be monozygous twins.

Dizygous twins have a mixture of blood corpuscles, called "Erythrocyte-mosaic," which is due to the fact that before birth, these twins have bloodvessel anastomosis in the uterus, that is, their circulations are in communication with one another. The blood corpuscle-mixture means that if one of the twins possesses blood corpuscles from its own blood producing organs as also blood corpuscles from the corresponding organs of the other twin, the mixture is not necessarily 50-50. By means of fractionated haemolysis tests it has been proved that the proportions vary within large limits.

At the Swedish blood group laboratory, blood group tests of all twins used in experiments in most European countries, have been examined. All of these 200 pairs of twins were considered to be monozygous because of their phenotypical similarities. The tests show that no less than 15% of these pairs were proved to be dizygous twins.

47% of all bovine twins are of different sexes. It has long been known that about 85% of such heifer calves are sterile and about 15% normally fertile. An investigation covering the fertility of such animals could, up to the present, not be carried out before sexual maturity of the animals. As a result of blood tests it is now possible to ascertain at a much earlier date whether such heifers are fertile or infertile. If there is erythrocyte-mosaic it means that the blood circulations of the twins communicated and the male hormones supressed the female hormones because of their much more rapid development, and therefore the female is infertile. If this mosaic is not found, there was no anastomosis and the female animal is fertile.

Problems for the future.

Apart from these practical applications, there are many other problems which may possibly be solved with the aid of blood tests.

In horses and pigs cases have occurred where the differences between the type of blood of the male and that of the female infer early death of the foal or of the piglet. Similar to early foetal mortality of human beings, with this difference that the foal or piglets are born alive and only absorb the antibodies with the milk. Haemolysis of the erythrocytes in the foal or piglet occurs at a later stage but will also ensue. Corresponding cases have not as yet been found in cattle. Explanation for different forms of early foetal absorption, may be found with the aid of blood group tests.

While it is improbable that production characteristics are coupled with blood groups, it appears possible that the lethal characters, debility and predisposition to disease may be so coupled.

It may be that blood group genes are coupled with desirable or undesirable genes and if this is so the type of blood of an animal may serve as an "indication" in breeding for the transmission of these genes from generation to generation.

Furthermore it may become possible to follow the manner of inheritance of such transmissible factors which are associated with specific blood groups. It is also quite possible that blood group studies may solve other problems of heredity.

THE IMMUNIZATION OF GOATS AGAINST HEARTWATER

by

A. D. THOMAS and P. R. MANSVELT

Louis Trichardt

The area north of the Zoutpansberg Mountains with its varied, abundant and nutritious bush, is eminently suited to goat raising. There is a considerable potential market for goat meat and the Angora goat has recently been found to do well in this area, yet the census figures show a marked decline in numbers, viz.:-

1942	—	206,000
1947	—	155,000
1952	—	183,000
1956	—	98,000

An even greater reduction is seen in the sheep population. It is, therefore, apparent that some limiting factor, such as disease, is in operation. Frequent investigation of mortality has revealed Heartwater and Verminosis as the main causes. Verminosis can be controlled by dosing but it is only recently that interest has been taken in the possibility of immunization against Heartwater.

Soon after the establishment of a Heartwater Blood Bank in the area, requests were received for the immunization of a few goat rams imported from the Cape Province. The high susceptibility of the goat to Heartwater was again confirmed and only the use of antibiotics made the process reasonably safe. (For details see Table 1).

It was then decided to determine whether the young kid showed a resistance to Heartwater similar to that shown by the calf and whether immunization at an early age would prove practicable. Experience with young lambs had shown that, although the majority overcame the infection unaided, an appreciable percentage (up to 30%) died. As the goat kids to be used were to be obtained on loan from farmers, it was decided to apply treatment in all cases.

The infective heartwater blood used was that prepared at Onderstepoort and was drawn from the local blood bank. It consists of blood tapped from sheep at the height of the reaction into an equal amount of diluent consisting of:-

Sodium Citrate — 1.0 gm.
Peptone — 2.0 gm.
Lactose — 10.0 gm.
M/25 Phosphate buffer at pH 7,100 ml.*

For treatment Oxytetracycline hydrochloride was used throughout. Soluble tablets were crushed and dissolved in water and the solution was injected intra-muscularly. It was found that the dose should be in the neighbourhood of 3 mg. per lb. live weight and

* For storage the mixture is frozen rapidly and stored in dry-ice.

not less than 2 mg. per lb. To ensure a margin of safety, a minimum dose of 50 mg. to all kids up to 6 weeks old is recommended.

The available animals were divided into three age groups as follows:-

Group 1 — 4 weeks and under.
Group 2 — 5 to 6 weeks old.
Group 3 — 10 to 12 weeks old.

The experimental animals were infected by the intravenous injection of heartwater blood and temperatured daily thereafter. In some cases a "blocking" treatment was administered on the 7th to the 10th day. All animals were treated as indicated by the febrile reaction. The immunity was challenged from 26 days later.

Results. (See Table 1).

Group 1 (4 *weeks and under*).

Three kids (Nos. 4, 5 and 6) were used in a preliminary trial to determine the incubation period and the response to treatment. Their febrile reactions were very similar despite the variation in the infective doses given. On the 10th day there was an initial rise in temperature which subsided on treatment. This was followed three to six days later by a second rise to 105-106°F. This "twin peak" reaction was also seen in Nos. 22, 23, 24 and 26. In the case of the first three kids (4, 5 and 6) treatment was given a second and even a third time on the second rise in temperature. Nos. 22, 23 and 26 were not treated, nevertheless the second febrile reaction subsided of its own accord.

Of the remaining 21 kids, 6 were given suppressive treatment on the 7th, 9th or 10th day or on combinations of these days. Only one of these animals (No. 9) showed a febrile reaction but received no further treatment and recovered. The remainder all showed reactions but recovered with treatment. It will be noted from the table that the incubation period ranged from 10 to 19 days despite the fact that the infective dose (2 c.c.) was kept constant.

On challenge, one kid (No. 12) reacted on the 13th day but recovered after treatment. No. 27 also showed a febrile reaction on the 59th day but it is doubtful whether this was due to heartwater.

Group 2 (5 *to* 6 *weeks old*).

Only 5 kids were included in this group and none was given suppressive treatment. It will be noted that the reaction was easily controlled and there was no reaction to challenge.

Group 3 (10 *to* 12 *weeks old*).

The eleven animals included in this group were all given suppressive treatment on the 10th day, yet all (except two, Nos. 34 and 42) showed severe reactions which could only be brought under control with difficulty. All except No. 34 showed a solid immunity or challenge, indicating that this animal did not become

infected with the initial injection. These results indicate that goats of this age group show a susceptibility to heartwater similar to that of the adult.

Adults.

The reactions of 3 goat rams are included in the table for the sake of comparison. It will be noted that Nos. 1 and 3 required repeated treatment. No. 2 had been in the area some days before being presented for immunization when it showed a temperature of 104°F rising to 106°F. It was treated for presumed heartwater and later infected. The mildness of the reaction indicated that it was already partially immune.

TABLE 1

Number	Sex	Weight lbs.	Infective Dose c.c.	Suppressive Treatment Day	Incubation Period	Reaction	Days Treated	Total oxytetracycline used mg.	Day Challenged	Reaction
GROUP 1 — 4 Weeks and Under										
4	M	10	5	—	10	—	10, 16	100	—	—
5	F	15	4	—	10	—	10, 13	75	—	—
6	F	15	7	—	10	—	10, 13, 14	125	—	—
7	M	15	2	—	17	—	19	50	52	Nil
8	F	15	2	7, 10	—	—	—	50	52	Nil
9	M	15	2	10	27	—	—	25	52	Nil
10	F	15	2	7	—	—	—	25	52	Nil
11	F	15	2	7	—	—	—	25	52	Nil
12	M	15	2	7, 9, 10	—	—	—	125	52	Treated recovered
13	F	15	2	10	19	—	19	50	52	Nil
14	F	15	2	—	13	—	13	50	52	Nil
15	M	15	2	—	13	—	13	50	52	Nil
16	F	15	2	7	—	—	—	50	52	Nil
17	F	15	2	—	12	—	12	50	55	Nil
18	F	15	2	—	10	—	10, 12	100	55	Nil
19	M	15	2	—	14	—	14	50	55	Nil
20	M	15	2	—	8	—	8	50	55	Nil
21	M	15	2	—	14	—	14	50	55	Nil
22	M	15	2	—	10	—	10	50	55	Nil
23	M	15	2	—	10	—	10	50	55	Nil
24	M	15	2	—	10	—	10, 14	100	55	Nil
25	F	15	2	—	13	—	13	50	55	Nil
26	M	15	2	—	10	—	10	50	55	Nil
27	M	15	2	—	14	—	14	50	55	Re-covered
GROUP 2 — 5 to 6 Weeks Old										
29	M	28	3	—	13	—	13	50	33	Nil
30	M	20	1	—	12	—	12	50	33	Nil
31	M	14	1	—	13	—	13	50	33	Nil
32	M	15	2	—	12	—	12	50	33	Nil
33	F	21	2	—	12	—	12, 13	100	33	Nil

Number	Sex	Weight lbs.	Infective Dose c.c.	Suppressive Treatment Day	Incubation Period	Reaction	Days Treated	Total oxytetracycline used mg.	Day Challenged	Reaction
GROUP 3 — 10 to 12 Weeks Old										
34	M	50	3	10	—	—	—	50	26	Reacted treated rec.
35	F	27	4	10	14	—	14, 15	200	26	Nil
36	M	29	3	10	11	—	11, 12	150	26	Nil
37	F	30	4	10	10	—	11, 13, 14	200	26	Nil
38	F	34	3	10	11	—	11, 12, 13, 14	300	26	Nil
39	M	48	3	10	17	—	17, 18	150	26	Nil
40	F	33	4	10	11	—	13, 14	150	26	Nil
41	M	34	5	10	10	—	10, 12, 14, 15	200	26	Nil
42	F	28	3	10	—	—	—	250	26	Nil
43	F	42	3	10	11	—	13, 14	250	26	Nil
44	M	48	3	10	11	—	12, 13, 14	200	26	Nil
ADULTS										
1	M	80	10	—	12	—	12, 13, 14, 15, 16	900[1]	—	—
2	M	70	10	—	14	—	14, 15	200	—	—
3	M	70	10	—	12	—	12, 13, 14, 15	750[2]	—	—

1. Also received 600 mg. chlortetracycline.
2. Also received 450 mg. tetracycline.

Field Trials.

As shown in Table 2, 140 kids have been immunized on farms in the area and the results are extremely promising.

Discussion.

The incubation period following the infective dose varied from 8 to 19 days. This variation could not be correlated with the amount of blood injected and, therefore, with the number of infecting doses administered but is probably dependent on varying individual susceptibility.

The distribution of the length of the incubation periods in these trials was as follows:-

8 days	—	1
10 ,,	—	10
11 ,,	—	5
12 ,,	—	6
13 ,,	—	5
14 ,,	—	5
17 ,,	—	2
19 ,,	—	1

TABLE 2

Farm and Date		*Animals and age*		*Days treated and dose*	*Results*	*Remarks*
Emery	2.7.56	4 kids	4 wks.	10th 50T.	All alive.	
		6 lambs	4 wks.	10th 50T.	,, ,,	
	12.7.56	5 kids	2 wks.	10th 50T.	,, ,,	
	10.8.56	2 kids	4 wks.	10th 50T.	,, ,,	
		4 kids	8 wks.	10th 75T.	,, ,,	
		5 lambs	4-6 wks.	10th 50T.	,, ,,	
Alicedale	2.7.56	13 kids	4 wks.	10th 50T.	,, ,,	
Hardevlakte	10.8.56	19 kids	4 wks.	10th 50T.	,, ,,	Except one died shock.
Atenburg	29.4.56	20 lams	8 wks.	10th 50T.	,, ,,	
Elandspruit	19.6.56	10 lambs	6 wks.	10th 50T.	,, ,,	
De Droom	5.4.56	8 lambs	6-8 wks.	10th 50T.	,, ,,	
Van der Byl	1.10.56	44 Angora kids	under 6 wks.	10th 50T.	All alive of 8 kids not immunized one has since died and another been treated for H.W. and recovered.	

It would, therefore, appear that the 10th day is the best time to apply suppressive treatment as this will catch the early reactors on the rise of the fever curve while at the same time it is late enough to allow of the residual effects of the dose controlling the more delayed reactions.

The injection of heterologous (sheep) heartwater infected blood frequently causes shock in cattle especially when a second, or subsequent dose is given. In these goats the injection of sheep blood caused only mild degrees of shock. In one case in which death occurred a few hours after injection, the cause may have been shock or embolism.

Two goats (Nos. 12 and 34) failed to become infected or react after the infective dose. This phenomenon is also sometimes seen in cattle and cannot as yet be explained.

Summary and Conclusions.

(1) The goat is highly susceptible to heartwater and in the Northern Transvaal Bushveld losses from this disease are considerable.

(2) The natural resistance to heartwater demonstrated in young calves is also present in young kids. It is probably not strong

enough in the latter to achieve unaided as high a percentage of survival to artificial infection as in calves.

(3) Artificial infection during this natural resistant phase, combined with appropriate treatment does offer a practical and economic means of conferring an immunity.

(4) Reduced to its simplest form the process entails:-
(a) Infection with heartwater virus by the intravenous route.
(b) A single dose treatment with oxytetracycline administered intramuscularly on the 10th day after infection.

(5) Since the resistant phase extends at least to the age of six weeks, batches of newborn kids can be accumulated up to that age and be done together. This saves time and the trouble of doing smaller numbers at frequent intervals.

(6) Although actual experiments with lambs have not been done, field trials carried out present enough evidence on which to venture the opinion that immunization of lambs on the same lines as in kids would work equally well and be just as beneficial to the sheep industry in this area.

SOME OBSERVATIONS ON THE STORMONT TEST

M. C. LAMBRECHTS

Pretoria

Kerr, Lamont and McGirr (1946) in the course of experiments on desensitization observed that six to nine days following a single intradermal injection of tuberculin, in an infected bovine, the original injection site exhibited a certain degree of enhanced specific sensitivity. It was observed that the maximum reaction to tuberculin in "sensitized areas" occurred in the first 24 hours. Plum (1937) during experiments where cattle were tested at short intervals noted that the maximum reaction after successive tests occurred earlier than 72 hours — usually at 48 hours. Buxton and Glover (1939) established that the area of enhanced sensitivity of the dermis in cattle, following an intradermal injection of tuberculin, depended on the state of allergy of the individual and involved an area with a radius of 2-4" after one week. Between the second and fourth weeks the area adjacent to the original injection site had become desensitized in some instances but not in all animals tested. Full sensitivity was regained by about the sixth week.

Kerr, Lamont and McGirr, for practical reasons, chose seven days as the period between the initial and 'boost' injections and stated that the optimum time for the development of enhanced sensitivity had not been worked out. They further determined that an increase in the skin measurement of 5 m.m. following the 'boost' injection constitutes a positive reaction irrespective of the nature of the reaction.

The purpose of the investigations to be described in this article was to determine the optimum interval between injections for the development of enhanced sensitivity — using 'human' P.P.D. tuberculin. A second consideration was to determine whether the test, as described by the authors, was reliable under South African conditions.

Experimental.

The opportunity to conduct these investigations was offered by the owner of a herd naturally infected with tuberculosis. A large number of cows, infected with the disease, were kept under conditions of isolation and some of these were used for the purpose of the investigations under discussion. It is realized that a comparable state of allergy can never be obtained in animals naturally infected with the disease. The state of allergy in any one animal is, however, in any case a variable factor. It was therefore decided

that the results of the investigation may be of interest. Thirteen mature cows were selected. Ten months previously the reactions. elicited by them were as stated below — using a 'human' P.P.D. with a strength of 2 mgm. tuberculoprotein per c.c.

No. of Animal	Normal Skin Fold	72 Hours
1	4	54
2	6	14
3	6	15
4	6	21
5	4	11
6	6	23
7	6	33
8	6	32
9	4	15
10	6	11
11	6	16
12	6	12
13	5	9

Nine injection sites were prepared on the necks of cows 10-13 by means of clipping with a No. 000 hair clipper. Sites were spaced more than six inches apart. Sites on the left were designated L.1 to L.4 and on the right R.1 to R.5. O.1 ml. tuberculin was injected initially into all sites intradermally after measuring the normal skin fold in all instances. On successive days a second injection of 0.1 ml. tuberculin was made into the second, third, etc. sites in the same place as the previous injection. Careful measurements of the skin fold in all sites were taken prior to each successive injection. As it was felt that multiple injections of tuberculin might suppress skin reactivity, in cows 10-13, cows Nos. 1-9 were used to control this aspect. Single injections were made into sites prepared on the necks of these animals. Second injections were made into the same sites in cows Nos. 2, 3, etc., on successive days. Accurate measurements of the skin fold were made as before.

Observations were continued until 48 hours following the final injections.

The findings are recorded in tables I and II.

The cows used in the experiment were macroscopically positive for tuberculosis on slaughter. Unfortunately no further detailed examination of the animals was possible.

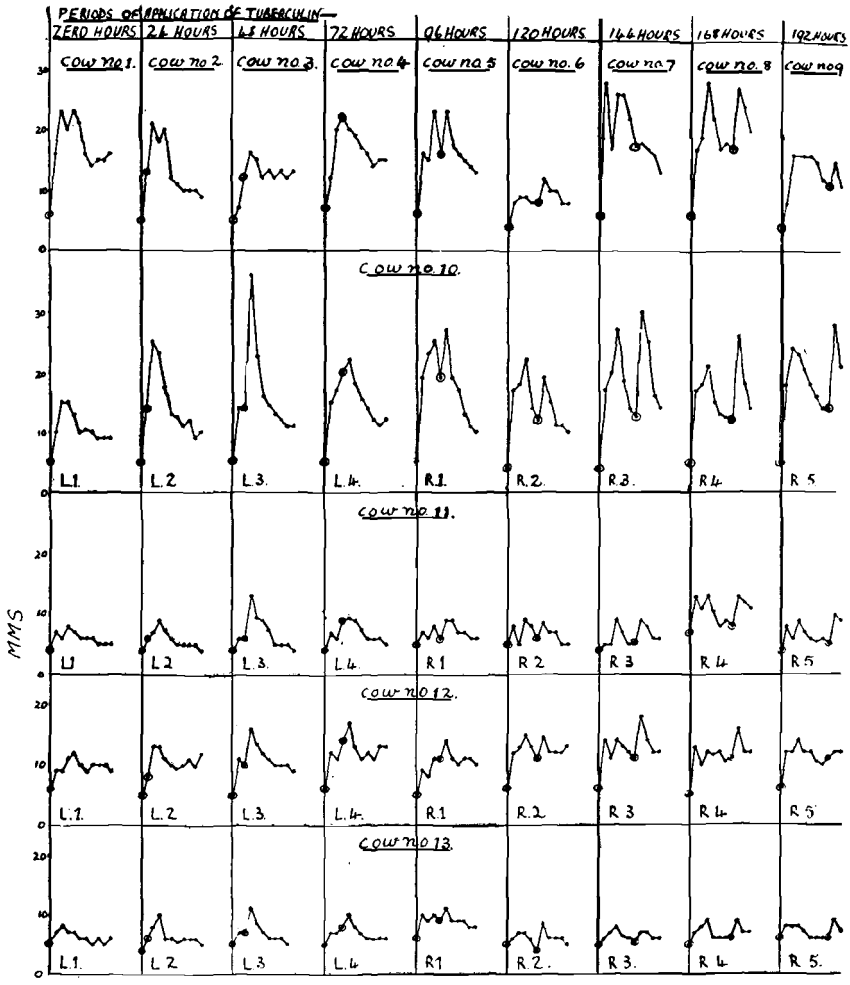
TABLE 1

(1) "X" denotes times when injections were made.
(2) Reactions which correspond to the double intradermal test are underlined.
(3) The 7th to the 8th day "boost" reactions are ringed.

Cow No.	Injection site	Skin Measurements										
		Normal	24 hrs.	48 hrs.	72 hrs.	96 hrs.	120 hrs.	144 hrs.	168 hrs.	192 hrs.	216 hrs.	240 hrs.
1		6X	16	23	20	23	21	16	14	15	15	16
2		5X	13X	21	18	20	12	11	10	10	10	9
3		5X	7	12X	16	15	12	13	12	13	12	13
4		7X	12	20	22X	20	19	17	16	14	15	15
5		6X	16	15	23	16X	23	17.5	16	15	14	13
6		4X	8	9	9	8	8X	12	10	10	8	8
7		6X	28	17	26	22	22	17.5X	18	17	16	13
8		6X	17	19	28	22	17	18	17X	27	24	20
9		4X	8	16	—	16	16	15	12	11X	15	11
10	L.1	5X	10	15	15	13	10	10.5	10	9	9	9
	L.2	5X	14X	25	23	17	13	12	11	12	9	10
	L.3	5X	14	14X	36	22.5	16	14.5	13	12	10	11
	L.4	5X	15	17	20X	22	18	15.5	14	12	11	12
	R.1	5X	19	23	25	18.5X	27	18.5	16	12	10	9
	R.2	4X	17	18	22	14	12X	19	15	11	11	10
	R.3	4X	17	20	27	18.5	14	12.5X	30	25	16	14
	R.4	5X	17	18	21	15	13	12.5	12X	26	18	14
	R.5	5X	18	24	23	20.5	18	16	14	14X	28	21
11	L.1	4X	7	6	8	7	6	6	6	5	5	5
	L.2	4X	6X	7	9	7.5	6	5	5	5	5	4
	L.3	4X	6	6X	13	9.5	9	8	7	6	6	4
	L.4	4X	7	6	9X	9.5	9	7.5	6	6	6	5
	R.1	5X	7	6	8	6X	9	9	7	7	6	6
	R.2	5X	8	5	9	8	6X	8.5	7	7	5	5
	R.3	4X	5	5	9	7	5	5.5X	9	8	6	6
	R.4	7X	13	11	13	10.5	8	8.5	8X	13	12	11
	R.5	4X	8	6	9	7.5	7	6.5	7	6X	10	9
12	L.1	6X	9	9	11	12	10	9	10	10	10	9
	L.2	5X	8X	13	13	11	10	9.5	10	11	10	12
	L.3	5X	11	10X	16	13.5	12	11	10	10	10	9
	L.4	6X	12	11	14X	17	13	11	12	11	13	13
	R.1	5X	9	8	11	11X	14	11	10	11	11	10
	R.2	5X	11	12	14	12	10X	13.5	11	11	11	12
	R.3	5X	13	10	13	12	11	10X	17	13	11	11
	R.4	5X	13	10	12	11.5	12	10.5	11X	16	12	12
	R.5	6X	12	12	14	12	12	10.5	10	11X	12	12
13	L.1	5X	7	8	7	7	6	6	5	6	5	6
	L.2	4X	6X	8	10	6	6	5.5	6	6	6	5
	L.3	5X	7	7X	11	8.5	7	6	6	6	5	5
	L.4	5X	7	7	8X	10	8	7	6	6	6	6
	R.1	6X	10	9	10	9X	11	9	9	9	8	8
	R.2	5X	6	7	7	6	4X	8.5	6	6	6	5
	R.3	5X	6	7	8	6.5	6	5.5X	7	7	6	6
	R.4	5X	7	8	9	6	6	6	6X	9	7	7
	R.5	6X	8	8	8	7	6	6	6	6X	9	7

TABLE II

(1) O denotes injection of tuberculin.
(2) L.1 to L.4 denote injection sites on the left neck.
(3) R.1 to R.5 denote injection sites on the right neck.



Findings and Discussion.

The increase in the skin measurement after the 'boost' injection on the seventh day was generally the best of all intervals more than four days. The considerable reactions found in cows Nos. 10 and 12 with a six-day interval were apparently isolated instances as was the eight-day interval reaction in animal No. 10. The most notable reactions were recorded where a second injection was given after a period of 48 hours. When the "single intradermal," "double intradermal" and "stormont" (seven-day inter-

val) values are compared in cows 10-13 the following figures are found:-

Cow No.	Single Intradermal	Double Intradermal	Stormont
10	10	31	14
11	4	9	5
12	5	11	5
13	2	6	3

The Stormont test was positive in all animals tested except No. 13. This animal showed low reactivity when initially tested and was probably in a state of semi-desensitization.

The multiple injections of tuberculin appeared to have suppressed reactivity somewhat in cows 11 to 13 — especially in the latter. All second injections of tuberculin into the initial site had some 'boosting effect' except that those applied after 72 hours had comparatively little effect. The maximum reaction was also generally obtained after this interval and with a more or less sudden decline immediately afterwards. Priestly (1946) indicated that the Stormont test was statistically, significantly more reliable than either the single or double intradermal tests. Why the longer interval 'boost effect' should be more significant and reliable from a test point of view than the shorter interval 'boost' as applied in the double intradermal test, is not clear. Buxton and Glover (1939) refer to the work of De Potter (1926) who demonstrated hypersensitivity in test sites of tuberculous subjects 2-5 weeks later. He further found that such hypersensitivity could be demonstrated also by using non-tuberculin-containing substances such as peptone water, glycerine veal broth, etc.

They also quote the findings of Dienes and Schonheit who proved that the cells in a tuberculous animal are not only sensitive to tuberculin but also to reagents of an entirely different nature.

Buxton and Glover in sensitivity experiments demonstrated that pseudo-positive reactions to concentrated glycerine broth could be obtained in animals with a high level of allergy provided the broth injections were made in close proximity to an injection of tuberculin.

All these findings suggest that the 'boost' effect of a second injection of tuberculin into the same site as a previous one may not be entirely specific in so far that enhanced sensitivity could also be demonstrated with non-tuberculin-containing substances in the tuberculous subject. The exact nature of the phenomenon of enhanced sensitivity remains therefore somewhat obscure.

The Stormont test was used on a number of animals showing obscure reactions to tuberculin in a 'clean' herd which subsequently proved to be infected with so-called skin lesions. Two of the animals which reacted positively to the test were slaughtered with negative results and four others later became negative. The authors

also found that the test did not differentiate non-specific sensitization due to 'skin lesion' infection. The test was used also to a limited extent in some other instances. It is not possible, however, to draw definite conclusions as to the value of the Stormont test as a routine method of testing.

SUMMARY

Summary.

Limited observations were made on the Stormont test in a number of cows naturally infected with tuberculosis. The aim was to establish the optimum interval for the boost effect of the second injection of tuberculin and also the reliability of the test under South African conditions.

It was found that the seventh-eighth day 'boost' appeared the most significant of the 'longer interval' values. This interval was also chosen by the originators of the test.

During the investigations some observations were also made on some other aspects of the tuberculin reaction.

It is realised that the investigation was very limited in scope and that the findings are subject to the influence of various factors such as variable state of allergy in the different animals and also variable sensitivity of the skin in the different locations in which injections were made.

The findings are nevertheless recorded as a matter of interest.

ACKNOWLEDGEMENTS

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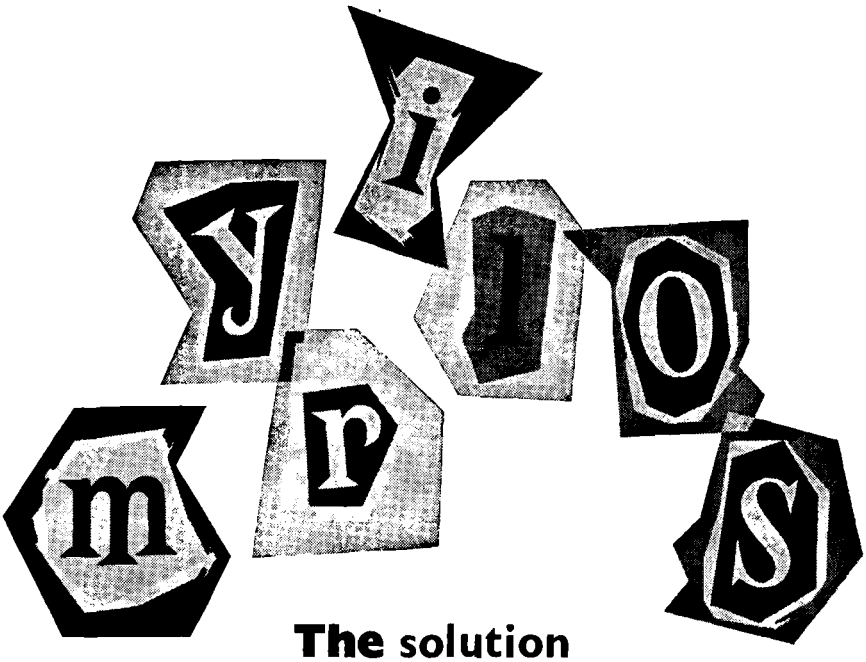

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OPTICAL INSTRUMENTS (PTY.) LTD.,

Sole S.A. & Rhodesian Zeiss Representatives

The recent unique developments by CARL ZEISS in the microscopic geld are best illustrated by two automatic Photo Microscopes, the "Ultraphot II" and the "Photomicroscope". Numerous other new accessories add to the versatility of our range of microscopes.

Other exhibits show an extract from our outstanding range of general laboratory equipment, amongst others Schott Laboratory Glassware, "Heraeus" Ovens, centrifuges, a new "Jung" Rotary Microtome, etc.

Do call at our stand and consult us for expert advice.

PARKE-DAVIS

Introduced to Veterinary Surgeons in Southern Africa for the first time at the last Congress as a quick and efficient treatment for foot-rot, Parke, Davis Laboratories (Pty.) Limited, the Southern African subsidiary of Parke, Davis & Co., are featuring Chloromycetin Tincture 10% Veterinary as one of their main exhibits. In addition to its effectiveness in foot-rot, Chloromycetin Tincture 10% Veterinary is successful in the treatment of joint ill, foul of the foot, orf, calf diphtheria, prevention of infection following surgery, foot lesions, wounds in dogs and cats and photosensitisation.

Other products featured include the Intramuscular, Kapseal and Ophthalmic presentations of Chloromycetin for a wide range of infections in large and small animals; Benadryl Parenteral for bloat, lymhangitis and serum sickness; Abidec Drops for vitamin deficiencies in small animals and Epanutin for hysteria and convulsions.

As a matter of interest almost all the products mentioned above, including Chloromycetin itself, are manufactured in Port Elizabeth.

PFIZER

The Terramycin range of Veterinary Dosage Forms on display at the Pfizer Laboratories South Africa (Pty.) Limited exhibit need no introduction to South African veterinarians. The provision of Terramycin in a wide range of forms to meet all requirements of administration and dispensing in veterinary practice emphasises the versatility of this truly broad-spectrum antibiotic with its firmly established record of successful therapy in veterinary medicine.

A feature of this year's Pfizer exhibit is the long-awaited arrival of Deltacortril Intramuscular — Veterinary. Deltacortril is Pfizer's brand name for prednisolone, the most potent of all the safe corticosteriods for systemic use — four to five times as effective as hydrocortisone. A wide range of indications in small and large animals work make this product an important addition to the armamentarium of every veterinarian. A folder outlining the facts and advantages you will want to know about Deltacortril Intramuscular — Veterinary will be available to you at the Pfizer stand, where you may also wish to discuss its use in more detail with one of the Pfizer men.

When you call at the Pfizer exhibit please also ask about Combiotic Aqueous Suspension. It is another new "veterinarians only" Pfizer product especially formulated for its effectiveness and economy in treating gram-negative, gram-positive and mixed infections.

Two new members of the "METI" range of Veterinary Specialities are being featured by Scherag (Pty.) Limited at the 1957 Congress. These are METICILLIN and METIBIOTIC, two important additions to the veterinarian's armamentarium.

It is recognized that "During stress, the corticoid requirements of all mammals are far above normal.[1]" Also "the sudden demands may at times be enormous, compared to ordinary physiology, and some adrenals are not adequate to cope with the situation.[2]" From experimental evidence and from clinical experience it is evident that infections produce a demand for extra quantities of cortical hormone[3] and this is the reason for the combination of corticoid and antibiotic in METICILLIN.

In the opinion of Koger, "Meticillin is one of the most effective single agents in the treatment of practically all febrile and surgical conditions in small animals.[4]" In large animals METICILLIN is valuable in pulmonary conditions, accident cases, traumatic gastrics, and other states involving stress and infection. Dose: Horses and Cattle, 10 cc.; Dogs, 1-2 cc.; Cats, 0.5 cc. (METICILLIN 10 cc. multiple dose vial, 10 mg. Meticorten and 300,000 units procaine penicillin G per cc.).

METIBIOTIC Mastitis Infusion combines Meticorten acetate with optimum concentrations of two antibiotics, penicillin and streptomycin. METIBIOTIC acts directly to check inflammation and to reduce swelling and oedema. This action facilitates more effective penetration of the antibiotics which are dispersed by the nonaqueous self-emulsifying vehicle throughout the inflamed udder. There is an early return of normal lactation, usually within 24 to 36 hours. Boxes of 12 tubes.

1. Selye, H.: Science 122 : 625 (Oct.) 1955.

2. Tintera, J. W. and Goldman, H. B.: N.Y. State J. Med. 56 : 872 (Mar.) 1956.

3. Paschkis, K. E.: Clinical Endocrinology, Hoeber-Harper, New York. 1954, p. 301.

4. Koger, R. B.: Vet. Med. 52 : 188 (April) 1957.

CHAS. F. THACKRAY (S.A.) (PTY.) LIMITED will be exhibiting a comprehensive range of their Surgical Instruments, Sterilizers and other items of Veterinary interest; including Davis & Geck Catgut & Sutures, Zeal Clinical & Veterinary Thermometers, and Sterling Rubber Gloves. A new feature of Davis & Geck Catgut, is the new Surgilar Pack, in which the Sutures are packed in plastic envelopes instead of glass tubes, thus obviating the problem of broken glass and also effecting a considerable saving of storage space. We shall be pleased to welcome all members of the Congress to our Exhibit.

ERRATA

Article on "Paralysis of the branches of the Nervous Vagus — N. recurrens, N. pharyrgens and N. laryngeus cranialis — as an etiological factor in whistling and roaring in horses. J.S.A.V.M.A. V. 28, 1.

Omitted from Table I :

16(s)	3.125 g6	6.25 g5	9.375

Footnote Table II — delete R.

Pg. 69 No. 8, four from 47.

DRUGS LISTED IN THE SIXTH SCHEDULE OF THE MEDICAL DENTAL AND PHARMACY ACT 1928 (ACT NO. 13 OF 1928) AS AMENDED AND REGISTERED AS REMEDIES UNDER THE FERTILIZERS, FARM-FEEDS, SEEDS AND REMEDIES ACT. NO. 36 OF 1947.

MAY & BAKER (S.A.) (PTY.) LTD.

Avistol — Sulphathiazole — Bacterial Colds in Poultry.
Compron — Sulphanilamide/Sulphapyridine — Post-Partum Infections.
E. M. Bazin — Sulphaquinoxaline — Caecal Coccidiosis.
M. & B. 693 — Sulphapyridine — Systemic Bacterial Infections.
M. & B. 693 Soluble — Sulphapyridine Sodium — Foot Rot.
Penicillin Intramammary Injections — Penicillin — Mastitis.
Protegan with Penicillin — Penicillin — Mastitis.
Solcon — Streptomycin — Vibriosis.
Solupyridine — Sulphapyridine — Systemic Bacterial Infections.
Strinacin — Triple Sulphonamide/Streptomycin Combination — Calf Scours etc.
Sulphanilamide — Sulphanilamide — Systemic Bacterial Infections.
Sulphadiazine — Sulphadiazine — Systemic Bacterial Infections.
Thalazole — Phthalyl-Sulphathiazole — Calf Scours etc.
Thiazamide with Proflavine — Sulphathiazole — Wound Infections. Puffer Pack.
Trinamide — Triple Sulphonamide Combination — Systemic Bacterial Infections.
Vegadin — Sulphadimidine Sodium — Systemic Bacterial Infections.

PFIZER LABORATORIES S.A. (PTY.) LTD.

Terramycin* Animal Formula for Mastitis with Polymyxin B Sulphate.
Terramycin* Animal Formula Soluble Powder.
Terramycin* Animal Formula Soluble Powder.
Terramycin* Animal Formula Tablets — 500 mgm. scored.
Terramycin* Eye Pellets with Polymyxin B Sulphate and Tetracaine.
Terramycin* Eye Pellets with Polymyxin B Sulphate and Tetracaine.
Terramycin* Intramuscular — 100 mgm.
Terramycin* Intramuscular — 1.0 gm.
Terramycin* Ointment with Polymyxin B Sulphate.
Terramycin* Suspension in Oil — 100 c.c.
Terramycin* Suspension in Oil — 500 c.c.

* Trade Mark of Chas. Pfizer & Co. Inc.

MILBORROW & CO. (PTY.) LTD.

Milborrow's Antiseptic Dusting Powder.
" Canker-Cillin.
" Eye Powder.
" Mikrozine brand Phenothiazine.
" Penco-Intramammary Ointment.
" Pen-Feed.
" Pen-Strept-Amide Intramammary Ointment.
" Pen-Strept-Amide Intra-uterine Jelly.
" Pen-Strept-Amide Pessaries.
" Pen-Strept-Amide Wound powder.
" Streptaguanidine.

PARKE, DAVIS LABORATORIES (PTY.) LTD.

Cholormycetin Tincture 10% Veterinary.

I.C.I. SOUTH AFRICA (PHARMACEUTICALS) LTD.

"Avloprocil" Oily — Procaine Penicillin in Oil.
"Avloprocil" A. S. — Procaine Penicillin in prepared aqueous suspension.
"Avdet" Teat Bougies — Penicillin Teat Bougies.
"Phenovis" Tablets 1 gm. & 5 gm. } Phenothiazine
"Phenovis" Dispersible Powder } Preparations.
"Sulphamezathine" Tablets 0.5 gm. }
"Sulphamezathine" Tablets 5.0 gm. } Sulphadimidine
"Sulphamezathine" Sodium Solution 16% } Preparations.
"Sulphamezathine" Sodium Solution 33⅓% }
"Udolac" Udder Paste with Penicillin.

AGRICURA LABORATORIA BPK.

Mastitol — 'n Suspensie van Sulfanilamied en penisillien, teen mastitis.

Streptocillin — 'n Mengsel van prokaien penisillien en streptomisien teen mastitis.

Agricillin — 'n Mengsel van penisillien, streptimosien en Chlortetracycline teen mastitis.

Agricura Oogdruppels — Chlortetracycline in 'n oliebasis.

Agricura Oogsalf — Penisillien en streptomisien salf teen oogontstekings.

Aristamid — Sulphasomidine-pille en 33⅓% oplossing teen bakteriese besmettings en sommige virusse (?) bv. sweetsiekte.

Antilaks — pille wat Furazolidone en Sulphasomidine bevat vir die behandeling van kalwerparatifus e.a. vorme van dermbesmetting by kalwers, ook Koksidiose.

Steekpile — bevat o.a. Sulphasomidine, sulfanilamied, stilboestrol.

Koksidex — oplossing van Sulphaquinoxalin teen koksidiose by hoenders.

Fenpoeier — 98% benatbare fenotiasien.
Fenpoeier P — 99-100% onbenatbare fenotiasien vir perde.
Fenpille — bevat 5 gram fenotiasien.

Mikrofen — besonder fyn fenotiasien met 'n hoër doeltreffendheid en dus 'n laer dosis.

Flavoline Wondpoeier — bevat o.a. sulfanilamied, flavine, boorsuur.

Wondsalf — bevat sulphasomadine, fenielkwiknitraat.

Wondolie — emulsie van vitamien A-olie met sulfanilamied en fenielkwiknitraat.

There may be more, but the Firms concerned did not respond to the invitation extended to them.

DIRECTOR OF VETERINARY SERVICES.

THE SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION COUNCIL MATTERS

Minutes of the Council Meeting held on Thursday, 21st February, 1956, at the Meat Board Building, Pretoria.

Present: Dr. P. S. Snyman (President) Drs. R. Clark, G. D. Sutton, M. de Lange, C. F. B. Hofmeyr, R. du Toit, E. M. Robinson, H. P. Steyn, S. W. J. van Rensburg, H. H. Curson, L. v.d. Heever, S. van Heerden (Hon. Secretary).

Present by Invitation: Drs. W. Malherbe, A. M. Diesel and H. Graf.

Apologies: Drs. P. J. du Toit, M. C. Robinson, M. C. Lambrechts.

I. *Matters arising from the Minutes:-*

(a) *Cooperative Employment of Veterinarians.*

The President asked the Convenor of the Sub-committee of Inquiry to report. Dr. Diesel reported that owing to difficulties in having a further report typed in time, he was unable to make copies available to Council members.

Dr. Steyn then moved that this matter stand over until the next meeting — copies of the report to accompany the agenda of the next meeting. Agreed.

(b) *Schedule of Fees:-*

Dr. Steyn reported that the Committee had been unable to have the draft Schedule of Fees typed and distributed to Council members before the meeting. It was agreed that this be done and the schedule be considered at the next meeting of Council.

(c) *S.P.C.A. Memo and Prevention of Cruelty to Animals Bill:-*

The President explained and outlined the situation obtaining at the present time. Dr. V. Shearer, M.P. had been advised to draw up a bill and as a result of this, the Minister of Justice had agreed to appoint a select committee to consider the bill and to accept memos and evidence on the matter.

The S.A.V.M.A. had asked, and had been granted facilities to present a memo and oral evidence to the Select Committee. In consequence, the collation of comments of members and branches on the memo prepared by the S.P.C.A.s had become unnecessary. The General Purposes Committee had, however, prepared a memo on the bill, bearing in mind, however, the comments of members and branches.

Dr. Clark, Convenor of the General Purposes Committee proceeded to read the memorandum piecemeal. Amendments agreed to by Council were incorporated. It was further agreed that a sufficient number of copies of the amended memo be sent by airmail to the Clerk of the Houses of Parliament, and that Dr. Clark be nominated to submit oral evidence to the Select Committee in Cape Town. Dr. Clark would request the assistance of Drs. Flight and Horwitz. Council agreed to pay costs of travel of Dr. Clark, by air, and in addition reasonable out of pocket expenses.

(d) *Internship of Veterinary Graduates — Natal Branch Resolution:-*

The President asked Dr. Graf, Dean of the Veterinary Faculty, to address Council on this matter.

Dr. Graf said that, as Faculty had appointed a sub-committee to go into the matter of prolonging the veterinary course of training, it would be opportune and expedient to submit the resolution to Faculty for consideration.

Dr. Steyn supported the Dean, and this was agreed to by the meeting.

2. *Faculty Posts:-*

The President sought leave to bring this matter up for discussion now, as the Dean of the Faculty (Dr. Graf) was anxious to leave for another appointment.

Circular No. C.57/4 of 20th February was laid before Council and noted.

The President then read a letter, dated the 14th of February, from the Rector of Pretoria University. After further discussion it was agreed that the Rector be advised that Council was unable to amend its views in regard to this matter.

Council further agreed that the Action Committee remain constituted, with the mandate previously agreed to.

3(a) *Redrafting of Constitution of S.A.V.M.A.:-*

Dr. Diesel reported that a meeting had been held with Mr. Weggers, a companies consultant, and that Mr. Weggers was now redrafting the Constitution.

Mr. Weggers had indicated to the Hon. Secretary that he would have a draft ready for consideration of the sub-committee before the next meeting of Council.

3(b) *Report of Fees Adjudication Committee of the Australian Veterinary Association.*

Dr. Malherbe expressed the view that it might serve a very good purpose to have such a committee of Council. Dr. Clark felt that Dr. Steyn's committee which is drawing up the Schedule of Fees, should consider this report and submit a recommendation to Council on the advisability of having such a committee.

Such committee could act as a "go-between" between a complainant and the veterinarian.

This was agreed to by Council.

3(c) (1) *Sale of drugs not registered (and listed in 6th Schedule) under Act. 36/47.*

Dr. Clark reported on discussions held between deputed members of Council and representatives of Pharmaceutical Society. Arising from these discussions letters were addressed to:-

(1) Secretary for Agriculture,

(2) Secretary for Health, and

(3) Secretary for Health, re sale of strychnine.

Copies of these letters were read.

(2) *Professional Provident Society of S.A.*

Letter of the 10th January read and agreed to publish suggested preamble and prospectus, in Journal for the information of members.

(4) *International Veterinary Congress.*

The President outlined the position of the Permanent Committee and reminded members that Dr. Alexander was the representative of the S.A V.M.A. on the permanent Committee. Copies of the revised statutes of the Permanent Committee and the revised byelaws of the International Veterinary Congresses had been circulated to some Council members and to members of the General Purposes Committee for perusal. It was agreed to approve these.

The President further advised that Dr. Alexander as member of the Permanent Committee received copies of reports of Specialist Organisations which were affiliated to the International Veterinary Congress, and that Dr. v.d. Heever, Hon. Secretary of the Veterinary Public Health Group would be able to obtain copies of such reports from Dr. Alexander. The President reiterated the view that Council did not favour the establishment of such specialist groups in South Africa, but that members of the S.A.V.M.A. could join the International organisations as individuals. Council agreed with this view.

A letter from the Permanent Committee announcing the formation of an "International Association of Tropical Veterinary Medicine," accompanied by a copy of the draft constitution of this Association was read. It was agreed to acknowledge receipt of this intimation and to have a notice published in the Journal for information of members.

(5) *Annual General Meeting,* 1957.

The President expressed the view that more time should be allowed for discussion of papers delivered at the Congress; also that a theme or themes should be decided upon and that papers on these subjects be put up.

Dr. Diesel said that papers should not be read, but spoken to. This was supported by Drs. van Rensburg and Steyn. The Hon. Secretary suggested that a special Congress Sub-committee be appointed to arrange and co-ordinate

all activities of the Congress. It was agreed to consider this along with other details of the Congress at the next Council meeting.

The dates for the Congress had been agreed upon as 14th, 15th, 16th August, 1957.

(6) *General.*

(a) Radio talks — resumé of proceedings at Congress. Dr. Hofmeyr thought that the S.A.B.C. would probably allow these talks to be given every year.

(b) Dr. Steyn asked that branches be requested to submit suggestions on fees for ovine, porcine and avian work for consideration and inclusion in the Schedule of Fees.

OUTBREAKS OF SCHEDULED DISEASES IN THE UNION OF SOUTH AFRICA DURING THE PERIOD 1/3/1957 TO 31/5/1957

Disease	Cape	Natal	O.F.S.	Transvaal
Anthrax	(1) Barkly West (3) East London (1) Fort Beaufort (1) Herbert (2) Kimberley (2) Komgha (1) Peddie	(1) Vryheid	(1) Vrede (1) Boshof (1) Bloemfontein	(1) Christiana (2) Lichtenburg
B.W.D.	—	(1) Camperdown	(1) Heilbron	—
Epididymitis and Vaginitis	—	—	—	(1) Barberton (2) Potchefstroom (1) Pretoria (4) Ventersdorp (1) Rustenburg
Fowl Typhoid	(2) Bellville (1) De Aar (1) East London (1) Komgha (1) Lady Grey (3) Wynberg (1) Mossel Bay	(1) New Hanover	—	(1) Klerksdorp
Fowl Cholera	—	—	—	(1) Brits
Johnes Disease	(1) Bedford	—	—	(1) Ermelo
Lumpy Skin Disease	Fairly widespread over the Union except in the Western and South-Western districts wher isolated cases have been reported.			
Mange (Equine)	(2) St. Marks (5) Umtata	— —	(1) Harrismith	— —
Mange (Goat	(1) Butterworth (1) Glen Grey (1) King Williams Town (3) Peddie (1) Umtata (1) Victoria East (2) Willowvale	— — — — — — —	— — — — — — —	— — — — — — (1) Belfast
Mange (Porcine)	—	—	(1) Bloemfontein	(1) Delareyville
Rabies	(1) Albany	—	(2) Hoopstad (1) Kroonstad (1) Winburg (1) Wesselsbron	(1) Ermelo (1) Nelspruit (2) Zoutpansberg
Scab (sheep)	(3) Mafeking	—	—	(2) Brakpan
Trypanosomiasis Dourine	(1) Kuruman (2) Vryburg	—	—	(4) Potchefstroom (1) Schweizer Reneke (1) Waterberg
Trypanosomiasis Nagana	—	(1) Ingwavuma	—	—
Tuberculosis (Avian)	(1) Caledon	—	—	
Tuberculosis (Bovine)	(3) Beaufort West (1) De Aar (1) Fort Beaufort (1) Port Elizabeth	(1) Eshowe (1) Paulpietersburg (1) Pinetown (1) Vryheid	(2) Kroonstad (1) Senekal (1) Ventersburg (2) Viljoenskroon (1) Winburg	(1) Belfast (2) Bethal (1) Ermelo (3) Klerksdorp (1) Nigel (2) Potgietersrus (1) Pretoria (1) Randfontein (3) Rustenburg (2) Standerton (1) Vereeniging (1) Witbank

PRESIDENTSREDE

52ste JAARLIKSE KONGRES VAN DIE S.A. VETERINÊR-MEDIESE VERENIGING, 13 AUGUSTUS 1957

P. S. SNYMAN

Pretoria

Hierdie rede sal miskien totaal afwyk van die wat by so'n kongress tuis hoort; sekere gebeure van die afgelope twaalf maande genoodsaak my daartoe. As President van hierdie Vereniging is dit my plig om lede en groepe van lede te beskerm teen aanvalle en onverdiende kritiek wat uitgeoefen is. Ek sal nie alleen terug raps nie, maar sal ook gebruik moet maak van die seekoeivelsambok.

Aan die einde van verlede jaar was daar aansoeke ingewag om sekere vakante poste in die Fakulteit van Veeartsenykunde. Die salaris-skaal aangebied was u almal bewus van en is kommentaar oorbodig. 'n Paar maande later is dieselfde poste heradverteer met ietwat beter diens voorwaardes. Indien hierdie advertensies in die buiteland sou gesirkuleer het, sou ons Fakulteit as 'n derde of vierde-range Fakulteit bestempel geword het — dit is geensins die geval nie. As gevolg van die resiprositeit met die "Royal College of Veterinary Surgeons" geniet die graad van die Universiteit van Pretoria erkenning oral waar die S.A. Veterinêr-Mediese Vereniging erken word.

Sedert die stigting van die Fakulteit in 1920, was die uitbreiding maar baie gering. Dit is dan ook te verwagte deurdat die Fakulteit deur 'n Staatsdepartement beheer word. Myns insiens behoort die Fakulteit reeds lankal uit gebrei te gewees het, deur die skepping van minstens twee addisionele departemente, te wete Giënokologie en Virologie, die belangrikheid waarvan al meer en meer op die voorgrond tree. Ons hoop dat hierdie sake binnekort in order gebring sal word.

'n Mens kan ook nie altyd verwag dat slegs die Staat en die Universiteit vir fondse vir uitbreiding moet sorg nie. Daar is gemeenskappe wat in besonder die voordele uit 'n hoër opvoeding trek. Ek keer later weer terug op die onderwerp Skenkings en Begiftinge.

As bewys van die aanvraag na meer Veeartsenykundige dienste, het gedurende die jaar verskeie advertensies in nuus blaaie verskyn om Veeartse te lok om hulle in sekere distrikte te vestig. Hieroor wil ek geen kommentaar lewer nie, aangesien daar 'n Spesiale komitee benoem is om hierdie saak te behartig. Ek wil net in breë trekke die agtergrond van hierdie advertensies skets.

Die vernaamste sentra waar sametrekking van die mees waardevolle veediere bestaan is in die omgewing van die Witwatersrand, die middel lande van Natal, die Westelike Provinsie en sekere distrikte in die Oostelike Provinsie. Gevolglik vind ons dat die meeste Veeartse hulle in hierdie streke gevestig het. In die ander dele van die land is dit maar in die laaste jare dat boeregemeenskappe hulle meer toelê op die teling van Stoet of hoëgraadse vee. Daar was bepaald in die Westelike Transvaal se graanstreke, meer goeie trekkers as bulle. Die boere gemeenskap in die streke, maak slegs van die dienste van 'n Veearts gebruik in gevalle van kliniese behandeling. Die gedagte om die kudde onder toesig van 'n Veearts te plaas het nog nie by hulle posgevat nie, en word die waarde daarvan nog nie besef nie. Sodra hierdie sienswyse by die boere gemeenskappe posvat, sal dit nie nodig wees om advertensies te plaas nie, maar sal Veeartsenydiens in die streke outomaties uitbrei.

Aan die begin van die somer toe die Knopvelsiekte vinnig aan die versprei was weens die gunstige klimaats toestande vir die verspreiding van die siekte, het Landbou-distriks-Unies dikwels die Afdeling Veeartsenydiens van afsydigheid en selfs nalatigheid beskuldig teenoor die belange van die Veenywerheid. Minder as 'n maand gelede was die Afdeling Veeartsenydiens nog pertinent beskuldig van nalatigheid omdat een van die virussiektes van die Slenkdal en Wesselsbron tiepe sy verskyning in die Karoo gemaak het.

Dit is nou hier waar ek die seekoeivelsambok moet uit-trek. In een van ons vernaamste landboublaaie het die Inleidings artikel die Afdeling beskuldig van afsydigheid en pligs versuim. Instede van eers uit te vind wat die werklike posisie is, is sommer ingeval en saamgesing met die paddakoor. Mens sou kwalik verwag dat sulke onsin uit die pen van 'n verantwoordelike Redakteur kon vloei.

'n Paar weke daarna, toe die Redaksie miskien beter ingelig was, het daar net in 'n klein hoekie van 'n kolom, iets verskyn oor die werklike toedrag van sake. Heden en tot vandag toe sal ek in daardie selfde weekblad nog iets sien van 'n verskoning of iets dergeliks. Ek nooi van ganse harte al die Verslaggewers hier teenwoordig uit om die referate oor virussiektes by te woon. Dan kan u self oordeel of die kritiek op die Afdeling Veeartsenydiens geregverdig was of nie.

Ek het reeds in die begin van my rede melding gemaak van die begiftings of skenkings. In die meeste bedrywe, veral op die gebied van die industrie, word daagliks skenkings gemaak aan navorsingsinrigtings, ter bevordering van ondersoek in een of ander vertakking van die betrokke industrie. Behalwe vir 'n paar beurse aan individue om oorsee in 'n bepaalde rigting te studeer en 'n geringe bydrae om navorsing uit te voer, word weinig deur die boerderybedryf gedoen in vergelyking met ander industrieë. Ons is natuurlik dankbaar vir die bydraes, maar voel tog dat baie meer in die rigting gedoen kan word, en waarvan die boerderygemeenskap mettertyd die vrugte sal pluk. So word b.v. in die geval van

Steriliteit onder ons veediere, waar die verliese enorm en onberekenbaar is, slegs 'n paar duisend pond per jaar voorsien. 'n Bedrag van vyftig of sestigduisend pond per jaar sou baie meer in verhouding wees met die verliese wat gely word.

Ons landboublaaie behoort die boerderygemeenskap in te lig en voortdurend van die menigvuldige veesiekteprobleme op hoogte te hou.

Ek doen dus 'n beroep op die Redakteurs van ons landboublaaie om die boeregemeenskap in hierdie rigting te lei.

Alhoewel ons Vereniging nog jonk is en die getalle in die professie nie groot nie, is ek tog oortuig daarvan dat, nieteenstaande die aanvalle en kritiek na ons geslinger, ons tog sterk en standvastig genoeg is om dit alles te trotseer en die hoof te bied.

Ons beleid is kerngesond, ons wetenskap staan op 'n baie hoë vlak.

Jl. S.A.V.M.A. 28 (3), 1957

TUBERCULOSIS IN SOUTH AFRICA

by
Dr. B. A. DORMER,
Durban

History.

Of the original inhabitants of South Africa, the Bushmen, very little trace remains today and we have no knowledge of what diseases plagued them.

The first White settlers brought with them the odd case of tuberculosis, the first ever recorded being Paulo da Gama, brother of the famous Vasco. These persons passed on the disease to their close associates including servants, but there was little spread beyond these small foci.

Cornelius de Jong, the Commander of a Dutch man of war, in a book published on his travels in the Cape in 1781-97 mentions "consumption," and Percival and Barrow during the first occupation of the Cape by the English, also comment on this disease.

The Bantu invasion from the North, into what is now the Union, took place about the same time as the first Europeans landed in the South. There may have been some tuberculosis among these people, but the ruthless killing and abandonment of the sick would soon have dealt effectively with this.

In the early 19th century Moody stated that Pulmonary Consumption was uncommon among the Dutch and English, but common amongst Hottentots.

Persons with consumption, usually well off financially, started to flock to the Cape Colony when they became aware of the pleasant dry climate of such places as Cradock, Beaufort West and other Karoo towns. In the latter days of the last century the ideal treatment of tuberculosis consisted of living in a dry sunny climate, especially at a moderate altitude, and a carefully regulated life in such a climate, accompanied by plenty of good food and a peaceful existence in a world untorn by wars and thermonuclear weapons, tended to lead to the arrest of tuberculosis lesions and to natural healing by fibrosis.

These wealthy settlers gave the disease to their contacts in the towns and on the farms they bought, and considerably larger tuberculosis foci than those created round Cape Town in the early days resulted.

One or two items of interest regarding this invasion are given from evidence in the 1912 Tuberculosis Commission report in which a member comments:

"I had one case of a young fellow whom I had to dinner one Sunday in a very advanced stage of the disease, and this is what he told me — that the doctor told him that if he were here (Beaufort

West) for three months he would be allright. He had dinner at my place on a Sunday and the following Wednesday he died on my stoep, knocking at the door, with a tremendous haemorrhage."

The Chairman of the same Commission remembered seeing at Beaufort West the consumptives at the chief hotel, and of whom there were a number, sitting all day on the stoep, expectorating into the adjacent water furrow, which was the only source of water supply to many dwellings and to the extensive Coloured location just below.

The discovery of diamonds and gold in South Africa (1867) led to the arrival of hundreds of miners from Europe and also to the arrival of thousands of primitive Bantu from their isolated homes. The miners who contracted tuberculosis, especially the black ones, went home to die amongst their own folk, but before they achieved this end they spread infection all round them. Luckily in those days persons infected in the rural areas tended to stabilise their lesions, so that even at this stage not a great deal of harm was done. However, enough active disease arose to make responsible people aware of the growing menace of tuberculosis throughout the country.

In 1895 registration of births and deaths came into force in the Cape Colony and this gave a good idea of the mortality from tuberculosis.

In 1901 a Conference of the Principal Medical Officers of Health of all British South African Colonies and Territories exposed the great increase of tuberculosis amongst non-Europeans. In 1903 a South African Medical Congress passed a resolution to form a National Anti-Tuberculosis Association in South Africa, a body which did not come into being until 1948.

In 1905 the South African Native Affairs Commission pointed out the increase of tuberculosis amongst the natives. In 1906 the Municipal Associations resolved to approach the Governments of the various colonies to appoint a Commission to enquire into the matter. In 1910 Theal, the historian, pointed out that tuberculosis was seriously affecting the Bantu.

In 1910 the South African Medical Congress resolved "that the Government be requested to appoint a Commission to investigate the amount of tuberculosis amongst the white and coloured races of the Union."

All this led to the appointment of the South African Commission on Tuberculosis in 1912. The report of this Commission led in turn to improved housing, hygienic and dietetic measures on the mines, to the genesis of Nelspoort Sanatorium in the Karoo (1923) and to the appointment of a tuberculosis officer attached to the Union Health Department (1922-23).

This officer surveyed the field in 1923 and found plenty of tuberculosis disease, but the reassuring picture of a falling death rate. The whole question of tuberculosis in the Bantu was again studied when a "Tuberculosis Research Committee" was formed

in 1925 as a joint Government and Chamber of Mines effort. This Committee issued its findings in 1929 and showed clearly what a hold tuberculosis had on the Bantu people; for instance, more than half the total persons tested showed a positive tuberculin reaction. Still, the report was not "alarmist" because it was felt that the Bantu would soon develop an immunity to tuberculosis in the same way as the people of Europe were supposed to have done. This theory of racial susceptibility in primitive people was, to use modern parlance, sold to the Committee by Professor Lyle Cummings, who was its expert adviser.

This report really ends the first phase of tuberculosis in this country. The second opens with the beginning of South Africa's industrial revolution, accelerated beyond all imagination by the second World War.

The rural Bantu, most of them positive tuberculin reactors, poured by the hundred thousand into urban areas to seek work, at a time when housing was at a standstill because the country's total resources were mobilised for war. The result was a vast killing epidemic of tuberculosis. The environmental stresses- malnourishment, crowding and lack of hygienic amenities, could not have been more classically arranged to demonstrate that it is not racial susceptibility but these stresses that cause a major epidemic of tuberculosis.

If a miracle had not happened a large part of the South African human population would have been exterminated. That miracle was the advent of Streptomycin, Para-Aminosalicylic Acid and Isonicotinic Acid Hydrazide. By using these substances it was realised that there was a chance of doing something for the active cases, while society caught up with its environmental programme of proper housing, pure water supplies and adequate sewerage disposal, designed to prevent more cases from emerging.

THE BOVINE ORGANISM

A Survey of Bovine Tuberculosis in South Africa with Special Reference to Human Infection.

In his book "Animal Diseases in South Africa" Henning declared available information on bovine tuberculosis in South Africa, to be extremely scanty.

In 1892, the eminent veterinarian Hutcheon stated that there was no evidence of tuberculosis among cattle in any part of the Cape Province.

In 1898, Crowhurst reported a positive case of tuberculosis in a cow at Newlands.

In 1903, Hutcheon stated that although native cattle were comparatively free from tuberculosis a large number of better-class cattle in the western districts were affected.

In 1928, Paine and Martinaglia recorded tuberculosis in wild buck living under natural conditions in the Eastern Province of the Cape. They typed the strains and concluded: "The strains isolated so far, from Kudu and Duiker, may be summed up as highly pathogenic for rabbits and guinea pigs. The few fowls tested were refractory. The isolated organisms are dysgonic and non-glycerophilic, especially in the first generation. We have little doubt, from the above finding that the organisms studied are strains of B. tuberculosis bovis."

In 1930, Irvine-Smith, Director of the Johannesburg Livestock Market Department, reported four cases of tuberculosis of the udder in dairy cows. This was diagnosed clinically and confirmed microscopically on milk examination. Since then, many cases of open tuberculosis and tuberculous mastitis have been reported by veterinarians in public health services.

For many years the annual reports of the large abattoirs in the Union revealed a very low incidence of tuberculosis in cattle slaughtered. This, however, is hardly a true reflection of the position, as many owners of old dairy cows sell to cattle dealers or send the animals to uncontrolled peri-urban abattoirs and private slaughter establishments, where the disease is not recorded or even recognised.

In 1930 (Green) extensive tuberculin testing was carried out by the Union Government in co-operation with the City Council of Durban. When about 40% of the cattle reacted, the campaign was discontinued and the Durban Public Health Department embarked on a progressive pasteurization scheme.

It would appear that in the past tuberculosis in cattle has been associated mainly with well-bred dairy cattle but in 1953 Robinson reported a severe outbreak of tuberculosis in ranch cattle.

In 1956 (Lambrechts M.C. et al) acting on certain abattoir findings, disclosed a high degree of infection after the tuberculin test had been carried out on 1705 ranch cattle.

Very little is known about tuberculosis in native cattle but we know that very few cattle owned by Bantu living in Natal are tuberculin tested. In the Native Reserves cattle are kept mainly for lobola and to be slaughtered for feasts.

There is no information of tuberculosis in native cattle in Zululand but Major Daly, Sub-Director of Veterinary Services for Natal, informed us that tuberculosis had been detected at the Durban Abattoir, in a number of cattle, mainly oxen, sent by European farmers, from a specified area of Zululand.

This briefly is our present knowledge of tuberculosis in cattle in South Africa. What then is the position of bovine tuberculosis in man? Until recently we had little knowledge, despite many attempts at typing by resourceful workers.

Pirie in 1937 examined 100 mine workers and 98 cases of bone glandular and meningeal tuberculosis in the Union. No bovine cases were encountered.

Dormer and Friedlander in 1940-42, examined over 1,000 cases of tuberculosis bone, joint and lymph nodes; they found three atypical strains of M. tuberculosis. These were further investigated by Dr. E. M. Robinson of Onderstepoort and reported as atypical human strains.

In the large cities of the Union not all the milk sold is pasteurized. Horwitz, 1950-51, reported viable tubercle bacilli in 2.7% of herd samples supplying milk to Cape Town. Robinson and Pullinger estimated the percentage of infected milk samples supplied to Johannesburg was, at one stage, as high as 2.4%.

Since 1939, only 18 cases of tuberculosis of bovine origin in man have been described. These are enumerated as follows:-

Harrington and Emerson in 1939 examined 100 cases of human tuberculosis in Port Elizabeth. Of these, one bovine strain was recovered from the stool of a child.

Du Toit and Buchanan in 1942, isolated and identified a bovine strain from the cerebrospinal fluid of a young European female.

In 1953, out of 200 cases of human tuberculosis meningitis examined in the Western Province, Coetzee recovered strains of bovine origin from 2 cases.

In 1954, Buchanan, working on the Witwatersrand, examined 266 selected non-pulmonary cases of tuberculosis, culturally and biologically. He found that 13 were infected with the bovine strain.

In 1957, Martinaglia, Hobbs and Blaine recorded the first case in South Africa, of pulmonary tuberculosis in man due to a bovine strain. It was obtained from a European patient at the King George V Hospital.

The patient informed us that he had been drinking raw milk supplied by a dairy near his home outside the Durban Municipal area. The Government Veterinary Department was notified and tuberculin testing of the herd followed. This showed a severe herd infection confirmed during autopsy at the Durban Abattoir.

In South Africa, little is known of the role played by wild animals and man infected with bovine or human tuberculosis in spreading the disease to cattle. Fourie 1952, reported the unexpected occurrence of tuberculosis in a clean and tested herd at Onderstepoort, the animals showed lesions of bovine type.

Two Europeans and a native attendant who had been in close contact were sent to hospital for examination. They were underweight and complained of coughing but tuberculosis could not be established by X-Ray examination.

In another case, screening showed that the native attendant had pulmonary tuberculosis but his sputum proved culturally and biologically negative. It was felt that the positive reactors where no lesions were found on autopsy, must have been sensitized by this contact suffering from human tuberculosis.

It is interesting to note that de Kock described the first re-

corded case in which tuberculosis of the skin in man, was contracted from an infected dog, through a wound; it was proved to be of the human type.

Reasons for Paucity of Bovine infections reported in Human Beings.

The first thing to realise is that at a young age a large number of non-European children are infected by human bacilli, as is shown by tuberculin testing. The curve which follows gives some idea of the infection rate in each race. It is almost impossible to superinfect a child which already has human infection, with the bovine disease, so that a large number of children thus escape it.

The amount of infection in bovines is greatest in dairy herds and least in rural or ranch cattle. Dairy herds usually supply large towns and the greater proportion of this milk is pasteurized.

The amount of milk consumed by non-European children is very little indeed and dosage of bovine bacilli is therefore likely to be small.

Religious habits and tribal customs have a large influence. Indians, for example, boil all milk and the majority of Bantu drink soured milk. Boiling kills the bacilli and so does souring, so that of the small portion of milk consumed by non-Europeans the greatest portion is rendered safe.

However, the question of bovine infection in humans becomes more and more important as we achieve control of human disease, because as the number of susceptibles becomes larger with increasing control something will have to be done to eradicate tuberculosis in bovine herds in order to prevent infection of humans with bovine bacilli.

Prevention.

The most important factor is mitigation of environmental stresses. The better people are fed, the better they are housed, the better their water supply and hygiene generally, the less tuberculosis emerges.

Throughout the world tuberculosis is most prevalent in the poor and primitive and least prevalent in the well off and civilised. If certain environmental stresses operating in any community produce a certain number of tuberculosis cases each year, this number will be constant, in spite of treatment in hospital, until those stresses are removed.

B.C.G.

B.C.G. appears to confer some degree of raised resistance in humans and can be used in countries where numerous cases exist, as a biological method of prevention. It is, however, only a very small factor in the total picture.

I.N.H. Prophylaxis.

The use of I.N.H. in prophylaxis is new but appears to be of great value.

Three experiments of importance in human tuberculosis are in being at King George V Hospital.

1. All babies born of tuberculosis mothers in King George V Hospital were transferred immediately, in the past, to another hospital, to save them from being infected by their mothers. This is merely the human side of successful animal control.

However, the mortality of babies thus transferred was 100%, mostly from gastro-enteritis and pheumonia.

Two years ago it was decided to keep all babies at King George V Hospital, giving them 5 mg. per kilo per day of I.N.H. as a prophylactic agent, and since that date some 43 babies have been born and breasted by their tuberculous mothers in a tuberculosis ward. The mortality has been nil and only 2 children have developed a positive Mantoux reaction and those two did not receive their I.N.H. prophylaxis owing to the stupidity of the mother.

This is fairly clear proof of the value of I.N.H. as a prophylactic agent in children.

2. All staff in intimate contact with patients (domestic, nursing, laboratory) have received I.N.H. 5 mg. per kilo per day for 2 years. No member of the staff in this period has developed tuberculosis.

3. A community of Indians living in a modern housing estate is tuberculin tested and X-rayed each 6 months. All cases are treated with I.N.H., all contacts receive I.N.H. 5 mg. per kilo per day as a prophylactic agent. It may be possible in this way to determine the use of I.N.H. in a human herd.

Diagnosis.

Tuberculin testing divides humans into 2 groups — positive and negative.

X-Ray of the positive gives 3 groups:

1. Normal
2. Calcification (Ghon's focus)
3. Disease — Primary
   Adult.

Usually:

1. Represents high host resistance and complete resolution of the infecting focus.
2. Represents a medium degree of host resistance with the possibility of breakdown at any time if either host resistance diminishes or environmental stresses increase. This is the group from which most of our cases ensue.
3. Represents the lowest degree of host resistance.

Our present system of attack is to give (1) three months of I.N.H. 5 mg. per kilo per day, to kill possible live organisms and leave resistance unimpaired.

Give (2) 6 months of I.N.H. 5 mg. per kilo per day to kill live organisms and leave resolution.

Give (3) full course of treatment.

Treatment.

I.N.H. forms the mainstay of Tuberculosis treatment today, other drugs are adjuvants.

The dose should be as large as can be tolerated by the individual, usually in the region of 10-15 mg. per kilo per day and should be prolonged, if necessary for the whole of the patient's life.

Other drugs do two things — delay the emergence of resistance and enhance the effect of the I.N.H.

The question of resistance is in the melting pot. It seems as if the higher the degree of resistance to I.N.H. the less lethal the organism. The aim of treatment is to get resolution if possible and if this is not possible, as is usually the case where there is much pulmonary destruction, to use excision or collapse to get negative sputum. However, a certain number of chronic infectious cases will always be encountered and it is these who have to be made as little dangerous as possible to others. Here prolonged, high dosage therapy with I.N.H. is best.

Cost of Control in Humans.

The following table gives the cost of tuberculosis control in the Union:-

1947-48	Total Health Vote	£4,108,500
	Tuberculosis Allocation 16.8% of the total Health Vote	689,332
1952-53	Total Health Vote	7,144,000
	Tuberculosis Allocation 19.6% of the total Health Vote	1,402,715
1955-56	Total Health Vote	10,000,000
	Tuberculosis Allocation 30% of the total Health Vote	3,000,000

Lessons of Human Experience which may be of Value in the Veterinary Picture.

1st. I.N.H. prophylaxis.

Can we protect calves with I.N.H.?

If human experience is of any value, we can.

2nd. Is treatment of cattle with I.N.H. of any value?

We have two experiments planned or already in being to test this out in cattle.

One experiment is the study of a dairy herd of 125 head to test the effect of I.N.H. treatment of positive reacting cattle and I.N.H. prophylaxis in negative reactors.

The second experiment is more ambitious as it is to be a study of calves living under rural conditions, to test the value of I.N.H. treatment and prophylaxis in cattle living in this sort of environment.

It is proposed to use 4 groups of calves divided as follows:

(a) Controls.

(b) Infected with virulent bovine organisms.

(c) Infected with virulent bovine organisms and given I.N.H. therapy.

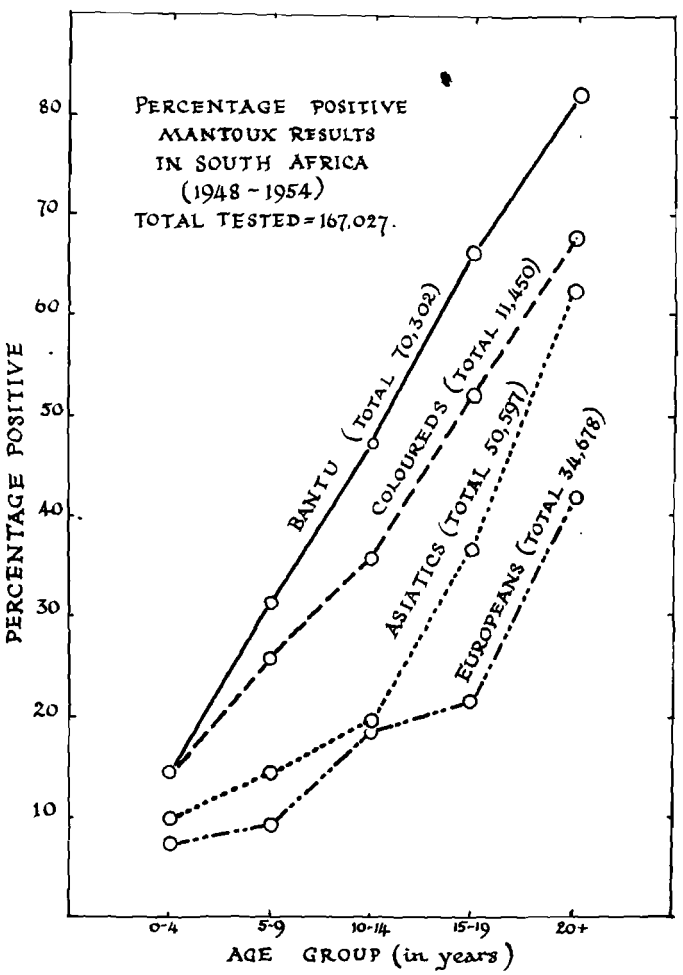
(d) Given I.N.H. prophylaxis.

Summary.

A short account is given of the overall picture of tuberculosis in South Africa.

The possible use of human experience in bovine disease is discussed.

Experiments are described to test the validity of I.N.H. treatment and prophylaxis in bovines.



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INTRATHORACIC SURGERY IN THE DOG

C. F. B. HOFMEYR

Pretoria.

The thoracic cage remained inviolate to the surgeon until not so very long ago. Large scale invasion of this fortress in man occurred only during the past two decades or so and has been closely associated with the evolution of an adequate technique of positive pressure respiration.

In this field our profession lagged a great deal behind the sister profession because our leading surgical patient, the dog, in contrast to the human being, cannot survive a unilateral open thorax at operation in the absence of an adequate technique for positive pressure respiration. This does not mean, of course, that the surgical hazard is not greatly increased in man in the absence of such technique.

The late appearance of thoracic surgery on the horizon of veterinary surgery, has given it an undeserved prestige. With an adequate but not elaborate instrumentarium surgical invasion of the thorax can be practised by all veterinary clinicians except those who have an aversion to the knife. No special skill is required other than the application of general surgical principles.

A brief review of the history of thoracic surgery in man may be of interest.

Fifty years ago and for the subsequent three decades the chest had no special appeal to surgeons apart from drainage of empyaemas and epithoracic operations. In his "Surgery of the Chest" published at the end of the last century Paget stated: "There are signs that we have reached the stage in this position of our art beyond which on our present lines we cannot advance much further." Of operations on the heart he wrote: "Surgery of the heart has probably reached the limits set by nature to all surgery." At that time there were some operations on tuberculous lungs, but these were regarded as daring and lucky.

The first World War with its numbers of open chest wounds was responsible for the greatest advances in this field and can be regarded as the beginning of the modern era of thoracic operations. However, between the beginning of this century and 1914 some notable pioneering work was done. The American, Meyer, with a positive-negative pressure apparatus and the famous German surgeon, Sauerbruch, with a negative pressure cabinet tried to overcome the disabilities imposed by pneumothorax during open chest surgery. Sauerbruch's apparatus enclosed the whole of patient, surgeons and assistants except their heads and weighed 10,000 lbs. It is not difficult to see why such an apparatus did not find favour with the veterinarians of the day.

In 1909 Meltzer and Auer, and Elsberg introduced a practical method of positive pressure anaesthesia by endotracheal intubation. At that time there was no enthusiasm for such developments. D. Tate in 1907 raised the following objections: "It reverses normal respiratory rhythm. Sudden distension of the lung causes interstitial emphysema and sub-pleural haemorrhages etc." We know of course that the latter observation is only associated with over inflation. In 1918 a leading surgeon, Bevan, stated: "Endo-tracheal anaesthesia has little place in practical surgery." This astonishing statement is perhaps better understood when one remembers that the numbers of strong young men with open chest wounds sustained in the war could have given the impression that protection from an open pneumothorax was unnecessary. Bell and Graham were members of the Empyaema Commission, World War 1. They studied this problem at the Johns Hopkins Medical School. Their observations are also of prime importance to the veterinarian and include the following: "A unilateral open pneumothorax exercises a deleterious influence on both lungs, the degree depending largely on the fixation of the mediastinum. As this is not constant, it cannot be regarded as an effective barrier in the presence of an open chest on either side. The ability of any individual to stand such an opening is largely dependent on his vital capacity."

As we are not discussing cardiac surgery now, no reference will be made to developments in this field.

With regard to oesophageal surgery, all the cases of oesophagogastrostomy attempted in man during the first one and a half decades of this century died.

Achalazia was usually treated by dilatation. Heller described an operation in 1913 and, although it is now a standard technique, it was largely overlooked at the time. Franz Torek did a successful resection of the thoracic oesophagus during the same year. Only in 1924 was this triumph repeated.

In 1918 Meltzer, delivering the presidential address at the newly formed American Association for Thoracic Surgery, discussed his method of endotracheal anaesthesia. He emphasized that, when the individual receives sufficient artificial respiration, the heart may be handled with impunity. However, with insufficient ventilation irregular beats and eventually, ventricular fibrillation and death might ensue. The two decades to 1940 showed steady development of thoracic surgery association with tremendous improvement of anaesthetic techniques and gradual understanding of shock, fluid replacement and blood transfusion. These advances were mostly concerned with pulmonary surgery.

In view of the sensitivity of the dog to air in the pleural cavity, canine intrathoracic surgery, for practical purposes, is of very recent development and awaited the advent of intravenous anaesthesia and the development of an acceptable technique of positive pressure respiration e.g. as can be applied by Magill's endotracheal catheter.

It is not intended to discuss all surgical conditions of the

interior of the chest in the dog. I hope only to refer to operations within my personal experience and having particular reference to the oesophagus as well as diaphragmatic hernia.

Foreign Body in the Thoracic Oesophagus.

Although oesophagoscopic examination is very helpful, two skiagraphs (lateral and ventro-dorsal) are usually adequate in case of a radio-opague body; otherwise the additional use of a barium meal is indicated. Any impacted foreign body has to be removed by oesophagotomy, the wound being closed by Connell sutures. Large rounded foreign bodies may be lodged against the arch of the aorta, but the cardia is a common site for all types. Incision of the gullet is avoided if the body can be eased through into the stomach.

Oesophageal Dilatation as a result of Persistence of a right Primitive Aortic Arch.

In veterinary literature only about 18 cases have been reported, four of which survived operation, none of them making a complete recovery.

My first case was a very weak six weeks old golden Spaniel pup investigated fully endoscopically and radiologically. It showed gross dilation of the oesophagus with a constriction above the heart. It was not recognised as probably being a case of right aortic arch, the ligamentum arteriosum pressing on the oesophagus. A thoracic surgeon who was consulted counselled the right costal approach. As the chest was opened the condition was recognised. As the pup was an almost forlorn surgical hope, what chance it might have had would certainly have vanished if the left side of the chest had been opened. It was therefore decided to short circuit the stenosis by performing an oesophago-oesophagostomy between the dilatation and the oesphagus of normal diameter nearer the cardia. This could only be done after the Vena azygos had been ligated. It was not a good operation as the anastomosis was under unavoidable tension. The pup surprisingly survived for 12 hours and then gave up the struggle.

The other case was seen very recently. All the clinical and radiological features corresponded to that of the first one. It even was the same breed and about the same age. A typical symptom was eating food, then walking backwards and regurgitating everything. It was regarded as a very poor surgical risk. Supportive preoperative therapy was given including prednisolone (a cortisone derivative). However, it did not appear as if the prognosis was improved. Under 'Sagatal' anaesthesia and after endotracheal intubation the left chest was opened at the ninth intercostal space, exposing the oesophageal hiatus. The diaphragm was then dissected from two thirds of the cardia leaving the far side intact. This brought the parietal wall of the stomach into view. It was drawn into the chest cavity until sufficient of the stomach wall was herniated to enable it to reach the dilatation. As was anticipated the dilatation could not safely be stretched to reach the surgical wound

after securing the stomach wall by means of interrupted sutures to the incised part of the diaphragm in order to avoid a tendency for it to slip back. To prevent post-operative herniation of other abdominal organs, a silk guy suture was passed through the apex of the herniated stomach pouch. A further thoracotomy wound was then made in the fifth intercostal space immediately above the dilatation. Using the guy suture to pull on the stomach, its wall was now delivered out of this last surgical wound.

Using an aseptic technique of anastomosis and the cutting suture, a union was now effected between the dilatation and the stomach wall, thus completing the oesophago-gastrostomy. The wound in the fifth space was then closed in the usual manner, and also the wound in the ninth space. The puppy stood the operation surprisingly well, as it resumed breathing soon after. Oxygen, parenteral fluids and other supportive therapy were given and the hope that springs eternal began to rise, but the animal suddenly died about seven hours post-operatively.

It is felt that provided the subject is a better surgical risk this operation ought to give satisfactory results. Some of you here may like to try it when the opportunity offers.

Achalasia (*cardiospasm*). i.e. any condition in which there is obstruction to the passage of food from the oesophagus to the stomach in the absence of an organic stricture. This condition is well recognised in man and copious medical literature is available. It is almost unknown in veterinary literature. Devine stated that in man the condition might be confused with gastric affections and may remain unrecognised. Johnson and Kirby, like Devine, prefer non-surgical therapy. Franklin studied the relevant operations extensively and Knight and Adamson produced achalasia in cats by bilateral vagal section, preventing it by simultaneous sympathectomy. Hill considered the cells of the plexus of Auerbach as vagal nerves. Degeneration of these cells has been described by Rake in cardiospasm. Etzel regarded B_1 deficiency of as aetiological significance in this generation.

The appetite of the dog with achalasia is undiminished, but the food is returned during or just after a meal, or fluid is brought up at intervals between meals. Since the food and fluid are held up at the cardia, the greatly distended oesophagus acting as a reservoir, its ejection takes place apparently without effort or resistance. It is interesting to mention that a human case recorded at the beginning of the seventeenth century always ate with a sponge attached to a smooth stick by his side. After having filled his gullet the sponge was used to dilate the cardia.

As this condition causes a dog to be extremely objectionable in the house, operation is indicated. I have not had sufficient experience of achalasia to be able to attempt to lay down a rule, but cardioplasty appears to be the operation of choice. After exposing the cardia by thoracotomy at the 8th or 9th intercostal space a crescent of diaphragm is dissected from the oesophagus at the

oesophageal hiatus in the manner previously described, the visible part of the parietal wall of the stomach is brought through and a 5 cm. incision is made along the length of the terminal oesophagus and on to the stomach wall. This incision is made either into the lumen of the organs mentioned or, if Heller's method is used, only an extramucous cardiomyotomy is done i.e. the operation is similar to the one for relief of pyloric stenosis in the infant. In both methods the wound is sutured transversely, a small part of the stomach being deliberately herniated and sutured to the incision in the diaphragm in the manner described. The operation is completed as in any thoracotomy.

Only a few cases have been done so that it is as yet impossible to say which method is best.

Hernia of the Diaphragm. This is the most common condition in the dog requiring intra-thoracic surgery. As is generally known, the diagnosis is based on auscultation, percussion and radiographic as well as general clinical examination.

The dog with diaphragmatic hernia is usually in a state of shock of greater or less severity. Major surgery is thus best postponed until the patient has rallied.

There are two conditions, however, which might require urgent surgical intervention. Firstly, if the thorax contains the stomach it is possible that both the gastric inlet and outlet might be obstructed leading to tympanites of the stomach and gradually increasing pressure on the heart and lungs causing asphyxia. All cases with diaphragmatic hernia should be carefully percussed to help in the determination of the presence of a hollow herniated organ, as they are potential victims of asphyxia. A fairly rapidly increasing dyspnoea is often due to tympanites and should be treated by the immediate puncture of the organ with a serum needle to release the gas. Thoracotomy should be done as soon as possible afterwards.

The other condition that may precipitate an emergency thoracotomy is hydrothorax due to partial strangulation of, in particular, some hepatic lobes. In view of the presence of abdominal organs in the thoracic cavity it is not possible to determine a fluid level by percussion. In the face of gradually mounting dyspnoea and the absence of an obvious cause, hydrothorax should be suspected. Draining the fluid away is difficult or impossible because it is interspersed between the herniated organs. In such a case holding the patient supine for radiography might cause death. The longer operation is postponed, the graver the prognosis.

For hernia of the diaphragm, operation is usually performed at the 8th or, perhaps better, the 9th intercostal space, on the affected side. In the case operated upon long enough after the accident to have developed adhesions, great care must be exercised especially where the liver is adherent, as separation of the adhesions may easily cause fatal hepatic haemorrhage.

The wound in the diaphragm is closed by continuous catgut sutures after the hernial ring has been secured by artery forceps

and the edges have been freshened if necessary. There are, however, some points requiring special consideration.

(1) If the tear is situated purely in the fleshy part of the diaphragm the suture line may tear out as muscle does not hold sutures well. This leads to recurrence of the hernia. In this case it is best, after closing the muscle wound, to anchor sutures in the tendinous part of the diaphragm, criss-cross the suture, and have these tension sutures carry sufficient strain to relieve it.

(2) When the diaphragm has been torn from the costal attachment, it is sewn on to the thoracotomy wound.

(3) Occasionally the ventral end of the tear runs at right angles to the rest of the wound and to the operation wound. Suturing of that part of the tear in the usual way is impossible as it is out of reach, and yet it is imperative as some herniation might again take place. With one finger inside the chest to act as guide a long curved, roundbodied needle threaded with No. 3 or 4 catgut is passed against the border of the rib in front of the tear, through the chest wall, through the wound edges, against the border of the rib behind the tear, and out again through the skin. An incision is then made through the skin between the points of the emergence of the catgut. It is then tied very tightly so that the catgut lies along the skin incision. This is then closed with interrupted silk satures, burying the catgut. As many such sutures can be put in as may be required. The tight tying is done to encourage adhesion between that part of the diaphragm and the thoracic wall. As tight tying causes some slight necrosis of the enclosed tissces, it follows that, unless the sutures are right against the ribs at either end, small openings will occur allowing ingress of air into the thoracic cavity with possible fatal results. Burying the catgut suture prevents this.

LITERATURE

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SOME UNUSUAL CASES

U. von BACKSTRÖM

Krugersdorp

The boredom of routine in private practice is fortunately relieved periodically by the unusual case. To be continuously on the qui vive for such cases is one of the prime requisites for a practitioner, and the satisfaction resulting from the correct diagnosis and successful treatment of the rare case is one of the greatest rewards of a practising veterinarian.

On the other hand the case may be diagnosed only at post mortem. It is best to be philosophical about these, and important not to become depressed by one's lack of acumen or experience for this is likely to be a constantly recurring phenomenon.

Unfortunately most practitioners never make time to record these types of cases, with the result that every beginner has to learn for himself. Every published history of an unusual case can be of immense benefit to other veterinarians.

It is suggested that some reward, prize or medal be offered for the most interesting or informative published report of this kind, to encourage especially practitioners and others not employed in research.

To set the ball rolling here follows a brief descriptions of some unusual cases.

(1) SEPTICAEMIC TUBERCULOSIS — DOG (1952).

Subject: 6 Year Old Collie.

Clinical: Persistent high temperature of 106 to 107°F for five or six days. Pale mucous membranes. No appetite or thirst. Rapid bounding pulse resembling that of biliary fever. Rapid weakening.

Tentative Diagnosis: Septicaemia.

Penicillin from the first day, and Streptomycin-Penicillin therapy for the last four days produced no recession of symptoms. The total course of the disease was six days.

Bloodsmears were negative until the final day when Giemsa-stained smears showed massive distribution of intercellular material. In an attempt to clarify this, various stains were tried, including Ziehl-Nielsen, which showed millions of M. tuberculosis. The dog succumbed the same night.

Autopsy. Septicaemic changes. Diffuse distribution in all organs of miliary tubercular nodules, so small in size as to be almost invisible to the naked eye. No primary lesion discovered.

Discussion. Might more massive Streptomycin therapy have produced results? It is very doubtful. The dog was receiving 1 gm

per day in two divided doses. Could this case have been diagnosed earlier by some other method?

(2) Liver Torsion — Pig. (1957).

Subject: 5 Year Old Large White sow.

Clinical: The sow had farrowed normally 10 days before, and the litter of eight was flourishing. The previous evening she had refused her food but allowed the young ones to suckle. In the morning she looked depressed, was weak in the hindquarters, and her eyes, mouth and ears seemed very pale. When the owner tried to examine her at 7 a.m. she made attempts to get to her feet, grunted and protested, then lay down panting with exhaustion. She evinced no signs of colic at any stage. At 8 a.m. she was found dead.

Autopsy. I was called in at 10 a.m. to do an autopsy. All mucous membranes and the epidermis were very pale, except for a few small mauve patches on the mammae and a mauve tongue.

The abdominal cavity contained about three quarts of watery dark-red fluid. On the right side of the ventral and visceral side of the liver was a half-inch thick layer of dark-red fibrin. The left lateral lobe had twisted anti-clockwise through 180° and had come to lie on the right central lobe. The colour of this twisted lobe was very much darker, and the size very much increased in contrast to the rest of the liver. The left lateral lobe had ruptured at the centre of its attachment near to the hepatic vein.

Discussion. The rapidity with which the complex developed i.e. approximately 12 hours from the first symptom to death, would have made a correct diagnosis improbable. It is presumed that internal haemorrhage was the immediate cause of death. If bile leakage with little haemorrhage had occurred, the more prolonged course might have enabled one to make a diagnosis.

A pig-breeder of at least 15 years experience informs me that he has not infrequently found a ruptured liver in a pig that had suddenly died. He could not say, however, whether there had been any torsion.

(3) Strangulated Undescended Testical. With Ureteric and Kidney involvement — DOG. (1953).

Subject: 3 Year old cross-bred West Highland Dog.

Anamnesis: Over a period of weeks the appetite had gradually decreased, and within the last 24 hours vomiting had started. The dog resented being touched. About 4 hours before I was called in, as he was mounting the stairs, he suddenly yelled, and sat down. After this it was impossible to approach him or touch him at all, as the least movement seemed to bring on agonising pains.

Clinical. After capture, examination showed that the dog's hind-legs were drawn up towards the abdomen which was very tense. Palpation over the kidney-region and the abdomen produced acute agony. To facilitate examination pentobarbital sodium was ad-

ministered intravenously. Renewed abdominal palpation revealed a small hen's-egg sized swelling in the upper posterior abdomen. The bladder was too small to be palpable. The dog was a unilateral cryptorchid. The pulse was rapid and hard, the temperature normal. Other findings were too vague to be of special note. A radiograph showed a black shadow in the region of the bladder-fundus. As (1) the swelling was in a fairly fixed position (2) Rectal palpation was negative and (3) the shadow could not be identified, the radiograph was submitted to a radiologist-friend. He made a tentative diagnosis of a dense bladder-tumour (perhaps external) or an unusual cystic calculus.

Exploratory laparotomy showed a normal bladder with no solid content. On the left side, near the fundus but not attached to it was a tumour-like body, reddish-black in appearance, with a tense shiny surface. On tracing its attachment which was in the nature of a slender cord, it was found to be wound around the left ureter. The ureter anterior to this point was enlarged to about five times its normal diameter, and was extremely thin-walled and very dark in appearance. The kidney was in a very advanced state of hydronephros, with very little kidney-tissue remaining.

It was concluded and later confirmed that the dark "tumour" was the left testicle, the blood-supply of which had become strangulated. A rupture of the ureteral ligament just anterior to the bladder had allowed the testicle to herniate, causing pressure on the ureter and gradual hydronephros. Subsequently the testicle had swung through 360° and passed through the tear again, thereby causing acute strangulation of the spermatic cord. Unilateral castration and nephrectomy was done. The dog made an uneventful recovery, and when last heard of, three years afterwards, was keeping perfectly healthy.

Discussion. Neither the radiologist, much less I, can explain why the shadow of the strangulated testicle should have shown up as dark instead of light.

The gradual onset of the original symptoms of anorexia is explained by the developing hydronephros and the sudden acute colic by the onset of strangulation of the spermatic blood vessels. If the necessity to treat had not been so urgent as to force one into an immediate laparotomy, it might have been possible to reach a correct diagnosis by a gradual process of elimination.

It may be noted that in the short-bodied dog palpation of the abdomen is sometimes very difficult, if not impossible.

(4) Atypical Lead Poisoning — Bull. Specific Therapy with Calcium Disodium Versenate. (1956).

Subject: 3 Year old Imported Ayrshire Bull.

Anamnesis: The Bull was in very good condition, and had been stall-fed for three weeks prior to the day he fell ill. He was exercised in a large pen attached to the loose-box. All feeding was strictly supervised, and the same food was also issued to two

adjoining bulls similarly housed with separate exercise pens, who never fell ill.

Clinical: I was first called in on Saturday night, March 3rd, 1956. The habitus of the bull was fairly normal. He had stopped eating, drinking and chewing the cud the previous day. Temperature was 107°F. Motion slightly mucoid and voided in small quantities only. Bloodsmears were negative. Thorough examination revealed only a unilateral (right side) pleuritis, and an early bronchitis (auscultation), but no nasal discharge. 1.5 mega units of penicillin were given, with a promise to call again the following day.

4.3.56: The bull was lying down on his chest, and was inclined to roll onto his side, so that he had to be watched continuously and be propped up to prevent bloat, which had already occurred during the previous night. He was groaning deeply and continuously, being obviously in severe pain. When he attempted to rise, the hindquarters seemed weak. There was marked unilateral lachrymation and slight salivation. The motion was very mucoid with small haemorrhage patches. There was complete ruminal atony. The bladder was paralysed and contained a very large quantity of urine; manual pressure per rectum allowed evacuation of three-quarters of its contents, at which stage sphincter control was again exercised by the bull.

The temperature which had fallen during the night and the early morning to 102°F, was up again to 106°F. The pleuritis was still marked, but the bronchitis seemed to be improved, although it was difficult to pick up the bronchial râles due to the deep groaning.

The mucous membranes were mauve in colour.

As on the previous day, the cardiac rate was normal (50) and the respiratory rate only very slightly increased. The marked colic caused very deep respirations with groaning which probably accounts for the absence of increased respiratory rate to be expected with pleuritis and/or lead poisoning.

A tentative diagnosis of poisoning was made, with the two possibilities of caustic or lead, with stronger suspicion on the latter. This diagnosis of lead poisoning was based mainly on the lachrymation, salivation, severe colic, bronchitis, paralysed bladder and weakened hindquarters. It must be stressed that none of the classical symptoms of blindness, aggressiveness or other nervous symptoms were manifested, nor was the motion diagnostic.

Two ounces of sodium thiosulphate plus two ounces of chloral hydrate plus eight ounces of magnesium sulphate were administered. The colic largely abated within 20 minutes, and had disappeared by the next morning. Four ounces of magnesium sulphate plus one ounce hypo three times daily were administered thereafter for one day, and four ounces magnesium sulphate twice daily for another five days.

In addition 2.5 gm. Aureomycin in 500 cc. Calcium-borogluconate with Magnesium and Phosphorus (Burroughs-Wellcome) were given intravenously to reduce the temperature, get the pleuro-

bronchitis under control, and to neutralise the free lead in circulation, and to control the pain and to act as a general stimulant, since there were signs of imminent cardiac failure. (Procaine Penicillin in oil 1½ M.U. twice daily was continued for four days).

5.3.56: The bull got up in the morning and drank a little water. Afterwards he lay down again, but showed no further signs of colic, and was much brighter. The colour of the mucous membranes was better. The temperature was down to 103°F.

After some considerable difficulty a supply was obtained of Calcium Disodium Versenate (Calcium Disodium — Ethylenediamine Tetra-Acetate), also called Versene or *Edta*, a product of Riker Laboratories. 24 Gm. in 120 cc. solution (24 Phials each of 5 cc.) were administered slowly intravenously. There were no side-reactions. The bull was up within 8 hours, started eating within 12 hours, and after 24 hours was apparently quite normal. There was no relapse.

Discussion. Faeces samples sent to Onderstepoort proved to be strongly positive for lead. This result was obtained only a few days after the bull had recovered, as the faeces were collected from the stall on 5.3.56 only. The faeces were a mixture of pre- and post- treatment lots.

The source of the lead was eventually traced to the roof-paint of the stables. Due to faulty drainage all the water from the very extensive roof collected in a pool in his exercise pen. This had been going on for many years, so that, apparently, a considerable lead concentration had built up in this pool. Although normally dry or only slightly muddy, recent heavy rains had caused a collection of water, and it is presumed that intermittent strong sunlight had concentrated the lead in the water by evaporation.

Since rain water also washed through an old storeroom, there was a possibility that there might have been white lead stored in this room, to be washed through by stormwater into the pool in the bull's pen.

It now appeared that at least two bulls, and probably a third, had died mysteriously in the same pen in the last ten years. As far as could be remembered the symptoms were vaguely similar.

SUMMARY

An atypical case of lead poisoning is described in a bull. Calcium Disodium Versenate was used as an antidote, and seems to be absolutely specific in the acute case. Supportive symptomatic therapy was beneficial.

ACKNOWLEDGEMENT

Thanks to Dr. Adelaar of Onderstepoort for the loan of the Calcium Versenate, and to the Director of Veterinary Services for the Analysis.

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(5) PERACUTE ANAPLASMOSIS — INVOLVING 337 COWS.

History: The outbreak involved a herd of 337 Grade Fries cows at the establishment of a cattle-dealer. The majority of these had come from the Cape Province, and a few from the Northern Free-state within the previous three to six months. The outbreak occurred in May, 1949, and before I had been called in, 19 cows had died within 20 days. Subsequently and before the outbreak was under control another 16 died from the same cause i.e. Peracute Gall-sickness.

Five different veterinarians had been called in during the first 20 days, and the diagnosis in every case had been poisoning, with the heaviest suspicion on lead. I feel that my colleagues were not to blame for missing this diagnosis (except for the fact that they were remiss in not making bloodsmears), for the symptomatology was almost diagnostic of lead poisoning in its most classical form.

Between the 8th and 28th of May 1949, I visited this farm ten times (every second day), and had, therefore, many opportunities of checking on the facts.

Clinical: The first case, and about 70% of the subsequent 120 cows, showed the following symptomatology:-

Temperature 105° to 107°F. Extreme depression until disturbed or handled, when hyperaesthesia supervened, with signs of aggressiveness, "blind" rushes, moving in circles, restlessness, muscular tremors, and rapid respiratory and cardiac rate.

The faeces were usually normal or slightly firmer and darker than usual. No clinical anaemia.

Some cases were prostrate within 12 to 24 hours and death frequently occurred within 24 to 36 or at the most, 48 hours of the onset of symptoms. In some instances the affected animal would be on its feet one minute, then suddenly bellow and drop with the dramatic suddenness of an anthrax case.

Because the farm had been extensively explored for poisons by the owners, manager and previous visiting veterinarians, I decided after a short investigation to eliminate this possibility from my mind, and start from the beginning again, and go through all the routine procedures.

The first of these i.e. bloodsmears, immediately gave the solution.

The bloodsmears showed the most massive Anaplasma marginale infection I have ever seen. About 60% of the erythrocytes contained parasites, and many corpuscles contained as many as ten (10) A. marginale. Others commonly had 4, 6 and 8 anaplasms to a cell.

The only blood change was a very marked anisocytosis. Some of the erythrocytes (not necessarily infected ones only) must have been three to four times (or possibly more) as big as the normal r.b.c. None of these larger r.b.c's. showed any sign of a nucleus, or residue of a nucleus. There was no punctate basophilia.

Autopsies: Of these I did five, but they were uniformly disappointing. Except for the usual changes associated with a hyperthermia, the disease was of too short a duration to leave any post-mortem lesions. In one case there was a suspicion of oedema of the meninges of the brain, but this was not definite.

Treatment: At every visit there was a sickline of usually about 20 case of peracute anaplasmosis. The manager became so expert in diagnosing the cases that afterwards we must have caught them all within 12 hours of the first symptoms showing.

Extensive use was made of a proprietary gallsickness antitoxin and a full dose (from 12-15 cc) was injected subcutaneously every 24 hours. In addition from 2-4 cc Carbachol (M&B) was administered subcutaneously every 12 hours. Since constipation was slight at this early stage, no other purgatives were necessary. 104 of the 120 cases so treated responded within 48 hours and were saved. 16 cases died (mainly more advanced ones, caught at a later stage, in the early part of the outbreak, but also some particularly peracute ones).

Preventive Inoculation: On the third day after I was called I managed, through the good graces of the Director of Veterinary Services, Onderstepoort, to get 77 doses of gallsickness vaccine, with which I inoculated the same number of cows. Eight days later another 204 were inoculated.

Sporadic cases still occurred 20 days after I was called initially but the manager coped with these without professional assistance.

Subsequently all new cattle introduced from the Cape Province or Free State were immediately inoculated.

Discussion: (1) Differential diagnosis included peracute lead poisoning and Heartwater. The bloodsmears as previously mentioned, excluded these possibilities.

(2) The nervous symptoms can be accounted for by either the marked hyperthermia, acute oedema of the brain, circulatory disturbance in the C.N.S., or other causes relating to the massive A-marginale infection and not clearly understood, since this protozoon is still very much of a mystery itself.

(3) I have come to the conclusion that the antitoxin used is of definite benefit in the very early case of Anaplasmosis. Subsequent use in imported Herefords that were seen very early in the course of the disease, responded with no other treatment. It must be stressed, however, that it can only be used in this way in cases where no systemic complications like constipation have developed.

(4) Notwithstanding the fact that the usual temperature reaction to gallsickness inoculation does not occur before the 21st day and usually only by the 5th or 6th week, it is felt that the inoculation was advisable and that an immunity started developing before the 3rd week even, otherwise it would be difficult to explain the definite abatement of the outbreak from ten days after the inoculation.

(5) It may be mentioned that of the herd of 337 cows over 150 had chronic metritis. Whether this had a debilitating influence and contributed to the unusually peracute form of the outbreak, is a moot point. Unfortunately no record was kept of the percentage incidence of cases in metritic and non-metritic cows.

(6) A large number of abortions and more than the usual number of retained placentas accompanied and followed on the outbreak.

Summary: A description is given of an outbreak of peracute gallsickness in a large herd of dairy cows. The symptoms were very similar to those of acute lead poisoning. The value of a field microscope with on-the-spot examination is stressed.

KIDNEY CYST — COW (1956)

Subject: 7 Year Old Grade Fries Cow.

History: The Cow had had four calves, the last one 15 months before her death. All four calves had been born alive.

About 10 months before death the owner had noticed that the abdomen of the cow was enlarging and naturally presumed that she was in calf. Later he thought it must be at least twins or triplets as she continued swelling up. The appetite continued to be quite good, and she always had all her cow-meal and lucerne. Two days before I was called in she went down, and from then her condition deteriorated rapidly, although she ate and chewed the cud right to the end.

Clinical: The cow was lying in the splayed position, on her chest. Temperature 98°F. Eyes sunken, mucous membranes normal, breathing very rapid and shallow. The heart could not be auscultated, but the jugular pulse gave a count of 40 per minute. The cow measured 5'6" from side to side over the widest part of her abdomen. Rectal palpation revealed a normal non-pregnant uterus, thus excluding the first suspicion of hydrops amnii. The two next most possible conditions, ascites or chronic peritonitis, necessitated an abdominal puncture which was made on the right side about 12" from the linea alba with a horse trochar and canula. A clear light-yellow thin fluid poured out. This had a very slight smell of urine. Bladder catheterisation and palpation revealed this organ to be normal with normal content, thus excluding ruptured or enlarged bladder. Vagus paralysis with abnormal enlargement of one of the stomachs was also automatically excluded by the

result of the paracentesis. As the cow was in extremis it was decided to shoot her and make a post-mortem diagnosis.

Post Mortem: After incising the cow along the ventral midline, the peritoneum was found adherent to a cyst from just behind the xiphoid cartilage to 12" behind the umbilicus. Two lateral incisions from the linea alba to the lumbar processes on either side, showed that the cyst was adherent for about 18 to 24" on either side of the midline. The duodenum and pancreas were inseparable from it. There were also multiple adhesions between the cyst wall and the liver and intestines, as also between the liver and diaphragm. None of the pelvic organs was adherent. The spleen was normal and non-adherent.

The liver and intestines were all considerably atrophied. The four stomachs were reduced to the size of that of about a 9 month old calf, and were lying antero-dorsally on the cyst. The intestines were all lying dorso-posteriorly on the cyst.

The carcase was cachectic and the mesentery non-existent to the eye, as it was inextricably mixed up with the multiple adhesions and reduced by pressure.

The cyst was incised (to make room to work in), and the wall had an average thickness of $\frac{1}{2}$ to $\frac{3}{4}$ inches, consisting of a very dense fibrous stroma, resembling tendinous tissue. It contained an estimated 40 to 50 gallons of fluid (which would weigh 400 to 500 lbs., as also about 10 lbs. of off-white fibrin). The origin and attachment of the cyst was traced to the right sublumbar region, where the right kidney should have been. Of this latter organ not the faintest trace was left. The right ureter varied in outside diameter from $\frac{1}{2}$ to $1\frac{1}{2}$ cm. and was still patent (estimated inside diameter at its narrowest point 1 mm). Bloodvessels to and from the cyst were enormously enlarged.

Discussion: The cow weighed an estimated 400 lbs. after removal of the cyst with contents, which means that more than half her total weight when alive was due to the cyst wall and contents.

It is surmised that the fluid content of the cyst consisted largely of serum, with a little urine only. The fibrin slowly separated out. The kidney probably ceased functioning altogether some time before death, and from then on the cyst-enlargement was due to plasma-accumulation due to circulatory disturbances.

The left kidney was normal in size and in every other respect. There were no uraemic symptoms or lesions whatsoever. The entire function had been taken over by this kidney.

The abscess of heart-sounds is difficult to explain, for the heart was hypertrophic, and both lungs atrophic. At the time of examination it is possible that cardial decompensation was setting in, which might explain the phenomenon.

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VETERINARY GERONTOLOGY — A SHORT SURVEY

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ABSTRACT: A brief review of the definition and scope of Gerontology in Veterinary Science and a single instance to illustrate the scope of geriatric medicine is presented. A plea for wider observations on the ageing canine is made.

Gerontology is the science of ageing in its broadest sense. The process of ageing involves every single aspect of the living organism which begins with conception and ends with death; thus infancy, growth, maturation, degeneration and senility are all stages of the same process.

Gerontology is concerned with ageing as a biological phenomenon, as well as with the consequences of ageing, and involves not only the individual or a group but also the species, for various species differ markedly in patterns of ageing.

Geriatrics is that branch of medical science concerned with physiological and pathological problems of individuals after reaching maturity. It recognizes that ageing brings specific changes in structure, functional activity, nutrition, response to infection, stress, and the like.

Geriatric medicine concerns itself with the treatment of the aged but is by no means limited to the futile staving off of death in the senile. On the contrary the largest contribution of geriatrics lies in the anticipation, recognition and correction of normal processes of ageing, which if left untreated, may prematurely lead to the malfunction of one or more organs resulting in a spiralling catastrophy for the whole organism.

It is unfortunately all too true in veterinary practice that the older patients are denied the benefits of modern therapy and clinical procedure on the assumption that the animal is too old in any case.

The veterinarian can derive intense satisfaction and the lasting goodwill of the owner of an old much loved pet if he will only recognise the fact that much can be done to make the years of later maturity relatively free of pain and incapacities. Therapy is rarely dramatically curative, but intelligent control of ageing often halts and sometimes completely reverses conditions leading to early senility and death.

Old animals tolerate surgery and severe illness surprisingly well if proper attention is paid to nutrition and conditions of anaemia, dehydration, protein loss, and similar easily corrected

states. It is true that old age has its problems of degeneration and decay, but a sensitive infection in an old dog reacts as promptly to the correct antibiotics as in the vigorous youngster. The operative procedure for relieving a bowel obstruction in the aged remains exactly the same as for taking an obstructive bottle-top out of the over-enthusiastic scavenging pup.

The difference lies essentially in the recognition of different response. The pup will probably oblige with the classic set of symptoms of vomiting, abdominal pain, tenseness, etc. The onset in the aged animal will be far less dramatic and unless acutely aware of the fact that he is dealing with an organism of lowered homeostatic efficiency, and senile atrophic adrenals with an inadequate stress response, the clinician will be faced with an undiagnosed toxaemia and total collapse.

The young animal can be operated on with success with little pre-operative preparation, but in the aged animal administration of cortisone and a blood transfusion as well as a drip infusion during the operation will make all the difference between successful surgical procedure and a last ditch stand just to satisfy the owner that everything was done for his animal.

As a branch of veterinary science gerontology hardly exists. In 1918 Goodpasture published a survey which must be one of the first attempts in veterinary medicine to evaluate many of the changes taking place during senescence. Unfortunately this challenge was not accepted by the veterinary profession at that time. Although Charcot published his Lectures on Senile and Chronic Diseases as far back as 1881, gerontology only became a well recognised branch of human medicine during the last two decades.

In 1952 the School of Veterinary Medicine at the University of California started longevity studies in kennelled Beagles under contract to the Atomic Energy Commission. This project is primarily for the study of the "Effect of Radiation on work capacity and Longevity in the Dog". Much valuable data on normal physiological values in the dog can be expected from this experiment.

Two chapters published in Canine Medicine under the authorships of Bourne and Bloom constitute the most thoughtful and comprehensive publications in veterinary science in the Western world at the moment. Both these publications, however, draw our attention to the vast opportunities which exist for gathering information about the changing picture of senescence in the animal kingdom. At present experimental and pathological laboratories constitute our main source of physiological information. These data however, can only be correctly interpreted if compared with normals for the particular age group to which the individual belongs.

If the major causes of death in old dogs are compared with those in the same human group, one may anticipate that a better understanding of ageing in the dog must eventually make in its

turn a valuable contribution to human medicine.

The dog is specially mentioned because, of all the animals, the canine lives in closest proximity to its master and is subjected to the entire varied environmental and dietetic pattern of the human race. In South Africa for instance, the primary cause of death in the European after maturity is of cardio-vascular origin. The dogs belonging to the European die primarily of chronic nephritis.

In the Bantu cardio-vascular diseases are rare, and from limited observations the author believes that nephritis is also less common in dogs belonging to Africans. It is quite possible that this factor is only related to the fact that dogs rarely become old when living in a Bantu community, but much wider observation is necessary to clarify this point. The abuse of alcohol, and tobacco, and the not easily definable strain-of-modern-living or psychological factors tend to be confusing when pure tissue degenerations are investigated in the human, but these factors are absent in the ageing dog.

One therefore feels that further investigation into the processes and diseases of the aged canine will eventually benefit both the old dog and his old master.

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SOME VETERINARY NOTES ON CANADA, WITH SPECIAL REFERENCE TO ONTARIO

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Ontario Veterinary College, Canada

Members of the Veterinary profession are proud of their vocation, and spend energy and thought in furthering its aims and welfare, apart from merely earning a living by working in one of the many spheres available to them in veterinary science. In order that we in South Africa may be sure that we are continually progressing in and bettering the profession, it is necessary that periodically we compare our organization, administration, education, and plans for the future with those of veterinary bodies in other parts of the world. For these reasons and in the hope that the following paragraphs may, in addition, be of general interest to his colleagues in South Africa, the writer has submitted this short account of the Ontario Veterinary College, and the veterinary associations, state veterinary services, veterinary organisation and man power of Canada.

Points of interest relating to veterinary education in Ontario are discussed first. The Ontario Veterinary College was founded in Toronto in 1862, and is the oldest of the accredited Veterinary Colleges in North America. It is the only English speaking Veterinary School in Canada. At St. Hyacinth in the province of Quebec there is a French speaking college. Other veterinary schools were founded in Eastern Canada about the turn of the century, and for various reasons, often financial, were forced to close. In 1908 the O.V.C. became affiliated with the University of Toronto, and this association was made closer in 1918 in that University entrance qualifications were adopted, and the course lengthened to four years duration. In 1922 the College was transferred to its present site in the city of Guelph, some 60 miles from Toronto.

The O.V.C. closes the western side of a campus shared with the Ontario Agricultural College and the Macdonald Institute of Home Economics. The word campus may require some explanation, since it is not in common usage in the Union. In the sense in which the word is used here it means a grass square that stretches about 250 yards in each direction, fringed by trees of several varieties spaced irregularly. The buildings and grounds of the Ontario Agricultural College and Macdonald Institute make up the remainder of the campus. The buildings and land of the three colleges belong to the Government of Ontario, and almost all the staff is employed by the government.

The main O.V.C. buildings houses classrooms; anatomy, bac-

teriology, histology, parasitology, pathology, and physiology laboratories; large and small animal clinics; an infirmary for small animals; radiology facilities; offices, departmental museums, and a library. There is also a well appointed student lounge, containing easy chairs, radio, table tennis and card tables. A students' shop is run, where many student requirements may be met and thus a trip "down town" saved. A manager is hired by the students to run this shop. All text books are sold to students at cost. The profit on other items is used to pay the manager.

Diagnostic and other extension work is carried on in a separate building. Other buildings include a large animal infirmary, and structures housing special equipment for work on virus diseases, poultry pathology, and diseases of fur-bearing animals.

At present a $1 million building programme is in progress and is expected to be complete in January, 1958. A further $300,000 will be spent in equipping the buildings. This will bring the physical equipment of the clinical departments to the fore front of the Veterinary Colleges in North America. Meticulous planning of detail and layout has characterised the project. The principal of the college, who is assisted in administration by a bursar/secretary and a registrar, is responsible to the Minister of Agriculture in all but academic matters. In regard to the latter he is responsible to the Senate of the University of Toronto of which he is a member *ex officio*. That the three colleges are under the authority of the Minister of Agriculture in the Government of Ontario to a considerable degree is reckoned by some to be an undesirable state of affairs. It is claimed that it is politically expedient for the government to retain its control over the colleges, but that this is not in the best interests of the college. There is ample evidence, however, to indicate that this close association with a department of the government results in greater financial support than would be the case with any other arrangement. The appropriation for ordinary expenditure (non-capital) for the current fiscal year is $1,097,000.

Of the 60 students who are admitted to the first year annually, about 10 to 15 fail to graduate. The degree earned is that of Doctor of Veterinary Medicine, which is conferred by the University of Toronto. The University also awards a Diploma in Veterinary Public Health, a M.V.Sc., D.V.Sc., and a Ph.D. The course for the degree of D.V.M. is of five years duration and besides the usual subjects, includes courses in Radiology, Veterinary Bibliography, Library Orientation, Diseases of Fur-Bearing animals, Public Speaking, and English Literature.

Short courses are offered to practising veterinarians by the college during the summer vacation. There are two varieties of courses, those lasting one day and those lasting a whole week. The one-day course is aimed at discussing a disease that has broken

out in a particular area, and the practitioners in that area are invited to attend. The week-long course is to bring practitioners up to date with recent developments, knowledge, and techniques relating to a particular field. Subjects covered in recent years in the latter courses include Poultry Pathology, Surgery of Small Animals, Surgery of Large Animals, Bovine Reproductive Diseases and Veterinary Radiology. A nominal fee is payable by those attending the course.

An Annual Report is published by order of the Minister of Agriculture for Ontario. In this the principal and heads of departments review the year's work.

The faculty for instruction, research, and extension comprises some 68 persons, of whom 18 are non-veterinarians. These figures exclude the staff members of the Ontario Agriculture College who give classes to veterinary students in a number of the basic subjects.

Several details of internal arrangements differ from those employed at Onderstepoort. Here in Guelph the clinical work is divided in the first instance into the departments of Large and Small Animals, and each of these has a Medical and a Surgical subdivision, whereas at Onderstepoort the main divisions of the clinical work are Medicine and Surgery, work on large and small animals being carried out by both. The Department of Anatomy is responsible only for the gross anatomy of the domestic animals, whereas a Department of Biology is responsible for teaching developmental and microscopic anatomy. The Ambulatory Clinic called "farm service" is run on an around-the-clock basis, and corresponds to a well organised large animal practice. Three veterinarians form the staff, and work after hours by rotation. Two students are assigned to each of the three cars going on calls. After hours two students are always on duty to answer the telephone and assist in any other way. Suitable sleeping accommodation is provided in the offices of the department.

The library is well stocked and provides ample reading and writing space. The staff includes two trained librarians. Students are encouraged to make the utmost use of the library facilities. During academic sessions the library is open from 8.30 a.m. to 10.00 p.m. for the use of students. Use of library facilities is obligatory for consulting references when students work on assignments. A list of contents of all new journals received by the library is sent to each faculty member. Before the journals are put in the reading room, they are routed to the staff members who have checked them on a list put out by the library.

Several of the departments, in addition to the regular professional staff have a graduate student who is working toward a higher degree. Graduate students act in a demonstrating capacity several hours per day, and in this way earn enough money to live on. In North America students working for a higher degree are generally required to attend several under-graduate and/or

graduate lecture courses, on which they may be examined, in addition to presenting a thesis.

A number of students are employed about the College as "student labour"; these students earn .75 cents an hour. Those who hold jobs under this scheme must have a certain minimum percentage of marks in term and final examinations. Posts available are janitorships, library assistant-ships, and technical assistantships in one or other of the departments. A student may not be required to work more than two hours a day during term time, and may in the five years he spends at college earn $1,000 or $1,200, which amount will pay his class fees and for some text books. On account of the four month summer vacation, a student is usually able to take a summer job at which he can earn sufficient money to pay his hostel, clothing, and pocketmoney for the ensuing year. Students in the junior years usually do unskilled labour during the summer, and those in the higher years may work for practising veterinarians or the Provincial or Federal Governments, in a field or laboratory capacity. Scholarships are awarded annually to nine students who have finished the fourth year. The incumbents are assigned to the various clinical departments in rotation during the summer vacation. In this way a diligent man may finance his education.

The College Royal is an annual event that arouses considerable enthusiasm. Students exhibit drawings, charts, illustrated case histories, and mounted specimens of various format that have a bearing on veterinary science. Many drug houses, meat packing concerns, and other industrial firms provide prizes for the contributors of winning preparation. This is a co-operative campus project, all three Colleges being represented on the board of directors. The main divisions of the show correspond to the option, e.g. Horticultural Div., Veterinary Div., Home Economics Div., etc. Each division is again broken down into classes. Any student on the campus can enter any or all divisions. Veterinary students also participate in the O.A.C. College Royal. The 33rd Annual College Royal was held in 1957, and great credit is due those students who organised the event. This is virtually an Agricultural Show and an 88 page booklet is required to catalogue the many entries in the various classes. The cash value of prizes was around $2,500 in 1957, but the honour and prestige of being a winner is more highly rewarding.

Looking at the broader field of Veterinary activities in Canada the following points may be mentioned. The local veterinary associations are active and enjoy a big membership. An example of these is the Toronto Academy of Veterinary Medicine, formed by the practitioners of that city. They hold monthly meetings for seven months during the winter, when papers are read by members or visiting speakers, local affairs are discussed, and a social hour is enjoyed. One of their schemes provides for a pooling of after-

hour phone calls. The calls are sent to a central point, and the telephone operator is instructed to try to sort the genuine emergencies from the calls that can safely wait till morning, and to report them to the member who is on duty that night. It is interesting to note that nearly all the private practitioners in New Brunswick are provincial employees. Only when a veterinarian is a member of his local association is he eligible for membership of the Canadian Veterinary Medical Association and the American Veterinary Medical Association. This arrangement is reversed in South Africa.

Before any veterinarian may practice in a state in the U.S., or in any province in Canada, he must fulfil certain criteria as set forth in the Acts, General Statutes, Laws, etc., of the state or province. These criteria may include examinations, credentials regarding character, registration, and citizenship clauses.

In 1949 the Canadian Veterinary Medical Association was formed, and this body has amongst its objects the sponsoring of examinations that will enable a successful candidate to practise in any part of Canada, but leaves to the individual province the granting of registration. As a national body with a high proportion of membership, the C.V.M.A. is in a better position to promote the welfare and interests of the profession on a broad front than the provincial associations.

In 1956 the veterinary associations of 64 states, provinces, and countries were affiliated to the A.V.M.A. as constituent associations. This body of nearly 15,000 members gives veterinarians an opportunity to act as a unit in furthering the interests of the profession throughout the whole of North America and several South American countries. The constitution of the A.V.M.A. provides for 17 standing councils and committees, including those on Education, Public Relations, History, Food Hygiene, and Ethics.

Members are guided by the "Principles of Veterinary Medical Ethics" which form a basis for professional etiquette. The president of the association appoints a three-man committee on Ethics, whose duty it is to promote an interest in and secure compliance with the code of ethics. The committee attempts to work in conjunction with the local committee of the constituent association, and may submit formal charges against offending members as provided for in the Administrative By-Laws. The code of ethics employed by the A.V.M.A. differs little from that of the S.A. V.M.A., except that the wording allows of wider interpretation. In paragraph 14, however, a distinct difference is shown in that it says, "It is customary and advisable in certain communities to print advertisements of professional men in local newspapers. But such advertisements should be reasonable in size and display. They should be limited to name, title, addresses, office hours and telephone number".

Recently the Principal of the Ontario Veterinary College, Dr. T. Lloyd Jones, quoted figures arrived at by an advisory

committee,which give an interesting summary of the fields of activity of the veterinary manpower in Canada.

General practice	791	47.1%
Small Animals	96	5.7%
Government of Canada	459	27.5%
Provincial Governments	87	5.2%
Municipal Government (P.H.)	62	3.7%
Teaching	73	4.3%
Other	98	5.8%

The Federal Veterinary Services are headed by a Veterinary Director General, Dr. K. F. Wells. These services comprise three parts: the Health of Animals Division, a division controlling the Meat and Canned Foods Act, and a division of Laboratory Services. The H. of A. Division is concerned with the control of epizootics and organising quarantine, mass rabies vaccination programmes, contagious abortion, tuberculosis control and inspection of certain imports that might harbour infectious organisms. The Health of Animals Division of the Federal Production Service controls contagious diseases outbreaks. There is a central veterinary laboratory at Ottawa and four regional laboratories across the country.

The South African Government Service is not unique in experiencing a shortage of veterinarians. The Federal Service has at present 24 vacant positions in spite of having had its numbers recently increased by many foreign gradutes. It is estimated that during the next five years 276 veterinarians will have to be recruited into the Federal, and Provincial Governments and the Public Health Services. The two veterinary colleges estimate that their total graduates during the period will number 388, of which 30 are from overseas and will be returning to their homelands.

The Meat and Canned Foods Act of 1907 is comprehensive and is published in a pocket-sized booklet for the convenience of those using it. It covers the production of all meats as well as canned fish, shell fish, meats and processed foods which are "placed in any closed can, bottle, package or container". Federal meat inspection is compulsory in establishments where products are exported from one province to another, or out of Canada. Local authorities enforce their own inspection regulations concerning foods to be consumed in the province of origin. At Federal and local levels veterinarians are responsible for red (beef, mutton, etc.) and white (poultry) meat inspection. Other foodstuffs are inspected by agricultural graduates.

What of the future? Increasing specialisation will certainly continue. It is interesting to note that the present tendency for practitioners to engage in a limited field is sending eddies into the O.V.C., where many students have already determined to go into a particular branch of veterinary practice, e.g. small or large animal. Several reasons for this trend are apparent. Practitioners in a

big city e.g. find it more remunerative and a lot less exhausting to limit their practice to small animals. With increasing competition it is difficult to remain abreast of all clinical developments while running a busy practice. For a recent graduate to set up in opposition to his older colleagues, he must have a certain minimum of equipment, and his limited capital will stretch only so far as that necessary to treat one group of animals adequately. In marginal areas, where large and small animals are equally numerous the group practice will favour one partner's doing more and more of one branch of the work.

The future of the O.V.C. is in the writers opinion a natural evolution towards becoming part of an independent university formed about the present campus together with the O.A.C. and Macdonald Institute. Only good can result to all three colleges. From the point of view of the O.V.C. it may be said that for years the veterinary profession has been trying to shake off the lingering aura of "horse doctor" and to place the profession on a par with that of Medicine. Owing to the necessity for having clinical material and adequate space, many veterinary faculties are geographically divorced from the parent university with the result that veterinary students are to some extent denied the privileges that the phrase "university education" implies. Should arts faculties be established at Guelph, the veterinary students would be given a wonderful stimulation to develop a philosophical background to their university education. Students in other faculties would be able to form a better understanding of veterinary science and the high degree of academic standing attained by veterinary students. To quote Beveridge: "Also the mixing of veterinary students with those preparing for various other forms of life will engender a mutual understanding and respect which will be useful later".

The figures quoted by Jones raise the question of where Canada is going to find veterinarians to fill all the posts falling vacant in the next five years and thereafter. Two possibilities exist: the establishment of another veterinary college in Canada, presumably in the West, but this it is not likely to obtain until at least such time as all O.V.C. vacancies are filled annually, or graduates from elsewhere are encouraged to emigrate to Canada. There is a faintly recognisable drift of graduate Canadian veterinarians to the U.S.A., drawn by financial attractions, and a corresponding drift of British and other European graduate veterinarians to Canada, drawn by a greater wage earning potential than exists in Europe.

Cameron (1953) points to Canada's vast land mass and foresees great opportunities for a progressive economy with the veterinarian playing an important part. He expects that research in breeding and nutrition will be as important as that in pathology and pathogens. Looking further afield, Cameron pleads the case of a commonwealth-wide uniform standard of veterinary education.

OPSOMMING

Die beheer, bestuur, geboue en geriewe van die Ontario Veteriary College word beskrywe. Die veeartsenyvereniginge in Noord-Amerika, die Meat & Canned Foods Act, die tekort van veeartse en moontlike toekoms van die veeartsenykunde in Kanada word kortliks bespreek.

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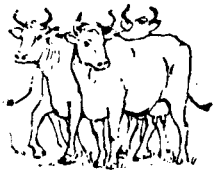

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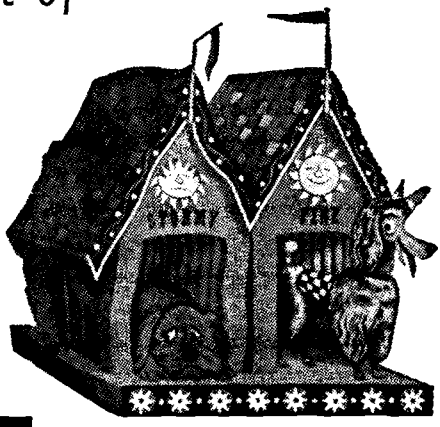

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THE USE OF CHLORPROMAZINE ('LARGACTIL' — MAYBAKER) IN PRIVATE PRACTICE

L. L. HANSMEYER & P. J. MEYER
Springs

This is not intended to be a scientific paper, but rather a general résumé of the results that have been obtained with 'Largactil' in this practice since June 1955.

For two years now (June 1955 — June 1957) 'Largactil' has been used as a routine in all procedures which required general anaesthesia in dogs and also for all other conditions where sedation to a greater or lesser extent was required, such as removal of diseased Harderian glands, teeth scaling, stitching of minor wounds, immobilisation of fractures and quietening of fractious patients. 'Largactil' was used alone for the latter conditions.

The drug has always been administered by the intravenous route in our practice at a rate of 1 mgm. per lb. weight. In the literature available nearly all authors have stressed the need for dilution of 'Largactil' if it is to be used intravenously, but we have used it in its normal strength of 2.5% w/v; and injected at the rate of 1 minim per second, that is, it can be given intravenously fairly rapidly with no untoward effect.

Within 15-60 seconds the effect is noticeable. The animal relaxes completely and usually goes down on the table. If the dog is roused it will become completely awake, open its eyes, and have the normal intelligent expression, but will show very little inclination to move. If put on its legs it will attempt to walk, some dogs being able to stagger along, others going down immediately. The time taken to recover completely varies considerably according to the route of administration and the age and condition of the patient.

If 'Largactil' is given intravenously the animal is back to normal within two to four hours, if young. In older animals the effect is more prolonged: used intravenously the effects are evident for up to four to ten hours and subcutaneously the effects are evident for twelve to twenty hours. Both with intramuscular and subcutaneous injections the effect is markedly prolonged in old animals — more so than by the intravenous route — even up to 48 hours with subcutaneous administration.

Used in conjunction with one of the barbiturates (in this practice in all cases pentobarbitone sodium) 'Largactil'* produces a state of pre-operative sedation so that animals can be handled more easily. Struggling is minimised and excitement, which often occurs during induction of anaesthesia and which is a significant feature of recovery from the effect of the barbiturates, is entirely absent.

Our usual procedure is to give the dog the required amount of 'Largactil' followed in 3-4 minutes by the estimated amount

* Sagatal (May & Baker).

of 'Sagatal'. In young animals the dose of 'Sagatal' varies from $\frac{2}{3}$ to $\frac{3}{4}$ of a c.c. per 5 lbs. weight after 'Largactil.' In older animals and in patients suffering from a toxaemia, the 'Sagatal' required to induce anaesthesia sufficient for surgical interference becomes markedly reduced, varying from a $\frac{1}{4}$ to $\frac{1}{3}$ c.c. per 5 lbs. weight. From these latter figures it can be deduced what great advantages stem from the reduced amount of barbiturate which is required.

Since June 1955, 527 bitches have been ovarectomised; 38 caesarean sections and 31 panhysterectimies (nearly all aged bitches) have been done using 'Largactil' and 'Sagatal', with one death only. One of the panhysterectomies died of generalised peritonitis ninety-six hours after the operation.

Unfortunately no figures were kept of other cases where 'Largactil' was used by itself or in conjunction with 'Sagatal,' but a conservative figure would not be less than one thousand dogs. The only unexplained mortality in two years was a well grown Boxer pup 12 weeks of age which had its ears cropped and which died while still under anaesthesia.

Apart from its obvious advantages when used as a sedative or for the induction of anaesthesia, the use of 'Largactil' really becomes important and in our estimation indespensable when dealing with old and debilitated patients or with animals suffering from infections or any toxaemic condition. To illustrate this I would like to quote some actual case histories.

(1) Ridgeback bitch, aged 13 years, suffering from acute purulent metritis. This animal had had its tubes tied 7 years previously — a complete ovarectomy not being done for the reason that she was at that time very fat and it was believed that she would become much worse if she were completely sterilised. She was seen again by us in April 1956 — off colour, temperature 104°, tender abdomen, and a profuse bloodstained mucopurulent vaginal discharge. She was treated in the usual way with antibiotics and stilboestrol and by the fourth day the temperature was down to 102.4° and it was decided to operate. At this stage she was still very fat and weighed 113 lbs. She was given a 100 mgm. 'Largactil' followed in four minutes by $5\frac{1}{2}$ c.c. 'Sagatal' which gave a plane of anaesthesia sufficient to carry out the operation. She was also put on to a glucose-saline drip immediately prior to the operation and this was continued until half an hour later. As she was so tremendously fat the operation took longer than usual, about forty minutes, and as she was being lifted from the table she awakened. She remained drowsy but awake all the day and by the evening she could get up without help. At no time during the recovery period were there any signs of excitement or inco-ordinated muscular movements. Within one hour after the operation she could lift her head and show recognition of her owners by wagging her tail.

(2) Fox terrier bitch, aged 12 years, presented for examination

suffering from acute metritis and severe toxaemia — temperature 104° — and she was so weak that she could stand but not walk without falling. She was given the usual treatment but after 24 hours her condition had deteriorated — temperature still 104° — but she could no longer get up. The owner wanted the dog destroyed but the operation was carried out against his wishes. The bitch weighed 15 lbs. and was given 15 mgm. 'Largactil' and ¾ c.c. 'Sagatal.' She was also put onto a glucose-saline drip. She stood the operation very well, also awakening within ½ hour after the operation. She remained drowsy for 36 hours and made an uneventful recovery.

As many metritis cases occur in aged bitches, we consider that 'Largactil' gives such a wide margin of safety when using barbiturates that the hazards of anaesthesia are minimised to such an extent that surgery can be carried out with far more confidence and with hope of complete recovery.

As we have very rarely used 'Largactil' orally for induction of anaesthesia we are unable to quote any experience of this method of administration.

'Largactil' has been used with great success in canine practice for the following conditions.

(a) Control of vomiting — initial dose given intramuscularly and maintained by oral dosage.

(b) 'Largactil' in conjunction with methylamphetamine for all accident cases. One would consider these two drugs to be antagonistic but we have found them excellent for control of shock.

(c) The following observation was made on one dog only so one can draw no conclusions from it but it does open interesting possibilities. A bitch was presented at full term unable to give birth and after vaginal examination she was given pituitrin extract. After nine hours there were still no signs of giving normal birth though she was bearing down strongly and it was decided to do a caesarean operation. She was given the usual dose of 'Largactil' intravenously preparatory to anaesthesia which was not started in the usual four to five minutes. The patient was left for a while because the instruments had not yet been sterilised. About 12 minutes after the administration of the 'Largactil' she gave birth with slight assistance to a very large dead pup. She was then given another injection of pituitrin and gave birth to four more live pups at approximately 15 minute intervals. She evinced no interest in the puppies as they were being born and they had to be helped from the placentas. One wonders therefore if this was purely accidental, or whether it was due to the complete relaxation of the musculature or even possibly to some psychic factor.

The use of 'Largactil' in large animals has been limited since not so much surgical work is carried out on them as in small animals. We believe that general anaesthesia in the adult bovine

should be avoided, if at all possible, because of the danger of inhalation of regurgitated ruminal contents. If assistants are available this is not so important, but the veterinarian is usually by himself, and when busy with an operation has little time to attend to other matters.

In cows we have used 'Largactil' with great success in caesarean sections. The general dosage is 500 mgm. intravenously used in conjunction with epidural anaesthesia, and local at the sight of incision. We have done 14 caesareans by this method in the last 2 years and in 11 cases the animals have lain completely quiet throughout the operation. In the other three cases 'Sagatal' had to be administered varying from 8 to 15 c.c. intravenously, to enable us to complete the operation.

In acute enteritis of adult bovines, specially valuable animals, we have used 'Largactil' in conjunction with the usual remedies and have found that the drug brings about an almost complete cessation of diarrhoea for up to 18 hours, thus giving the bowel a chance to rest and leading to quicker healing.

We have used 'Largactil' for the control of nervous symptoms in heartwater with good results, the drug being administered intravenously. Also for the control of vicious bulls for the purpose of handling, dehorning and paring of overgrown feet.

As far as equine practice is concerned there are not very many horses in this area and so we see very few of these animals. We have used 'Largactil' alone for nine castrations with very satisfying results. These animals were not thoroughbreds. Again the dosage is more or less 500 mgm. intravenously depending upon the weight of the animal. We have found that after the injections we had to work very quickly to put the sidelines on, otherwise the animal went down before we were ready. Before going down all the animals assumed a rather characteristic attitude viz. head held low down, the forelegs planted wide apart, and a tendency to knuckle over on the hind pasterns.

With the actual operation the animals gave a slight twitch when the scrotum was incised and also when the cord was severed. They got up within 5-20 minutes and tended to stagger until they had orientated themselves, but never have we seen any tendency to excitement or frenzy. Within 2-3 minutes after rising they stood drowsily without any inclination to move and remained so for up to two hours.

'Largactil' has also been used in colic with great success to control nervousness, excitement and frenzy. Given intravenously the animal quietens down immediately and if the dose is lowered to slightly below 1 mgm. per lb. the animal will not go down and can still be led around.

As 'Largactil' has been used extensively in this practice for the last 2 years, we feel that we can speak with some measure of authority on this matter. We have been so impressed with 'Largactil' that we feel that it will soon be regarded as an indispensable part of every veterinarian's list of essential drugs.

RELAPSES IN MILK FEVER

D. M. WALTERS

Worcester

Relapses after treatment of milk fever with calcium gluconate is a major problem in any rural veterinary practice.

The necessity of repeating in many cases a long journey to give a second and even a third injection, the digestive disturbances and reduced milk flow which appear to be associated with relapse, and the danger of unobserved relapse with fatal result, made the announcement by the S. E. Massengill Co., of America of a preparation to prevent relapses, most welcome.

The preparation which is marketed under the name 'Parterol' is described as a derivative of ergosterol and is claimed to be able to substitute for the parathyroid hormone, a deficiency of which is still the most commonly accepted cause of milk fever.

Since it has been my experience that the greatest incidence of relapse occurs in cows that have the first attack within twenty-four hours of calving, the use of the drug was limited to such cases.

Overa period of fifteen months, forty-three such cases have been treated with calcium gluconate intravenously and a simultaneous intramuscular injection of 10 milligrams of 'Parterol'.

Three of these animals relapsed in spite of the treatment, a figure which, though higher than that given by American veterinarians, still represents a considerable reduction from the previous records of 22% relapses.

The American figure of 0.0213% relapse rate after 'Parterol' represents the use of the drug in all cases of milk fever, regardless of the period between calving and the first attack.

It is my opinion that 'Parterol' is an extremely useful addition to the equipment of the rural veterinarian.

My thanks to Mesrs. Westdene Products, South African agents for the Massengill Co., for supplying a generous amount of the drug for testing.

REFERENCE

Harris J. R. and Clarkson T. B. (1955) — "Prevention of Relapses in Milk Fever." "VETERINARY MEDICINE." December 1955.

RETIREMENT OF SIR THOMAS DALLING

Sir Thomas Dalling who has recently retired from the position of Senior Veterinarian to the Food and Agricultural Organization of the United Nations, has had a very distinguished career, first as a research worker and subsequently as a teacher and administrator. On a short visit to South Africa in January of this year he gave a very interesting talk on the work of F.A.O. to the staff of the Onderstepoort Laboratory. In his early career he made some outstanding contributions in the fields of veterinary bacteriology, and immunology. He served in the Army Veterinary Corps in the first World War and it was then that he developed a taste for research work. After the war he worked with the late Professor Gaiger at the Glasgow Veterinary College where they were able to isolate the causal organism of lamb dysentery in sheep, an outstanding piece of work on which Dalling was able to elaborate when he was appointed to the Wellcome Laboratories, where he developed a veterinary department and worked for fourteen years. From the Wellcome institution he went to Cambridge as Professor of Animal Pathology where for the next five years he initiated and supervised valuable work on tuberculin, infertility in cattle etc. From Cambridge he went to Weybridge as Director of the Ministry of Agriculture's Veterinary Laboratory and in 1949 was appointed Chief Veterinary Officer of Great Britain, a position which he held jointly with the Weybridge one for a time, an onerous undertaking. On reaching retiring age he was given the appointment with F.A.O. which involved a great amount of travel in many parts of the world in an advisory capacity.

Sir Thomas Dalling received his knighthood in 1951 in recognition of his outstanding services, which also resulted in his being elected a Fellow of the Royal Society of Edinburgh and having three honorary doctorates conferred on him. He was awarded the Victory Medal of the Central Veterinary Society of England, the Dalrymple Champneys Medal, the Sir Thomas Baxter Medal and the John Henry Steel Medal. For two years he was President of the Royal College of Veterinary Surgeons of England.

Sir Thomas has always been interested in the younger generation, and plans — amongst other things — to devote himself to veterinary education on his retirement. His services will also be available to F.A.O. as a Senior Consultant so that his wise advice and active guidance will still be available to further the national and international control of animal diseases.

E.M.R.

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THE PROBLEM OF SKIN IRRITATION IN DOGS

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(Paper presented at the 52nd Annual Congress of the S.A.V.M.A.)

ABSTRACT

A concept is presented that the so-called "nonspecific dermatoses" are brought into being as a result of a trigger mechanism acting on an underlying systemic "proneness". This "proneness" manifests itself as a pruritus which is caused by allergies, organic diseases, nutritional factors, hormonal dysfunction, breeding and heredity, climatic and seasonal factors, focal infections, and errors of management. The trigger or excitant cause is frequently very slight and may be parasitic or mechanical. In the final analysis a complete examination of the dog may be necessary in order to ascertain the true cause of the pruritus.

INTRODUCTION

There is little doubt that one of the most important groups of conditions of dogs in practice is provided by the dermatoses. Any dog is entitled to scratch himself but when it becomes distressing to himself or to his owners it becomes pathological. With summer now not very far off it seems timely to reassess some of our ideas on the subject. Anybody who deals with dogs in his practice is certain to come up against the problem almost daily.

With the availability now of generaly excellent what one might call "wide-spectrum" parasiticides the incidence of parasitic dermatoses has receded into a position almost of insignificance, with the notable exception of follicular or demodectic mange. The question of parasitic skin diseases thus resolves itself into a matter of making a diagnosis by means of suitable specimens from the skin and then applying a suitable medicament to kill the parasites.

Dermatitis due to mycotic or bacterial infections also form a considerable group presenting its own diagnosic and therapeutic problems, but both this and the almost negligible category of virus infections causing pruritus are not particularly the subject of discussion in this paper.

We then have the wider group which has been lumped together under the omnibus term of "nonspecific dermatoses", a term open to quite definite objection as it admits to a lack of understanding of these conditions which is only partly true. It is about these that we really wish to speak today.

CONCEPT OF IRRITABILITY

Any study of skin irritation makes it clear that there must be an underlying *"proneness"* to itching before it becomes a pathological condition. We speak of pruritus as indicating itching *in the absence of organic lesions* but these changes may be microscopic or they may be purely functional, involving nerve endings or more

likely the vascular tissues in and under the skin. My own guess is that the minute endarteries and capillary bed forming the link between the arterial and venous systems are the site of these functional or anatomical changes, whether this be due to chemical or to nervous influences. In frank allergy or anaphylaxis such changes become easily recognizable in exaggerated form as erythema or urticaria.

Be that as it may, the fact remains that any given excitant, say a flea, may walk across a piece of skin of a non-"prone" dog and occasion no more than a casual jab of the paw. In a "prone" dog, however, a reaction is produced out of all proportion to the irritant. The question is then: why is the one dog so (literally) irritable, and the other not at all? This we shall attempt to answer by identifying some of the known aetiological mechanisms.

Briefly then the concept being put forward is that for the development of macroscopic skin pathology of "nonspecific" nature there must be an underlying *"proneness"* plus a trigger mechanism (represented in the above instance by a flea).

Aetiology of Skin Irritability

The demonstrable pathological lesion usually produced is the condition of eczema. This is a form of dermatitis which has been designated as eczema simply because it is a convenient and established clinical entity. There are a number of circumstances or sets of circumstances which may occur singly or in combination with each other and which give rise to eczema proneness. These include:

a. *Allergies*: We have considerable circumstantial evidence that allergy plays an important rôle, in the favourable response to antihistamine treatment and the influence of offending items of diet. We have been impressed with the frequency of the experience that dogs suffering from a longstanding and troublesome form of eczema lose their lesions when removed from their home environment and placed in kennels in the Onderstepoort Small Animal Clinic, regardless of what type of food is fed and in the absence of any type of treatment whatever. One must recognize the presence of possible allergens such as house dust, furnishing materials, floor polishes, blackjacks, insecticides, drugs, dietary constituents (e.g. horse meat), poultry and dairy meals and endogenous atopens such as worm infestations (especially Ascaris) and certain types of bacterial infections of the intestines. Fleas and other ectoparasites have been mentioned as mechanical triggers or excitants, but there is no doubt that they themselves or their excreta or their saliva may become incriminated as potent allergens in the body of the dog.

b. *Organic Disease*: It is a wellknown fact in dermatology that many systemic and organic diseases find a reflection in the condition of the skin. In dogs the usual ones are: failing kidneys, failing liver, chronic pancreatic disease and chronic constipation or chronic catarrh of the intestinal tract. It immediately becomes

obvious that where you are confronted with a "mere" eczema an investigation of the whole internal economy of the dog is called for if a complete diagnosis is to be made and the animal successfully treated.

c. *Nutritional Factors*: Overfeeding and underfeeding are important factors but in both cases an imbalance of constituents plays a more decisive rôle. Vitamins (particularly A) may be lacking as may be fats (unsaturated fatty acids). In the latter case there is dryness of the skin and a tendency to thinness of hair, and pruritus. In this country the major trouble is probably the idea that dogs can live their lives on a diet of refined maize porridge with nothing more than a little milk to supplement it. In the case of the less functional dogs, the pets, a fairly general fault is the feeding of sweets, chocolates, sweet biscuits and sweet tea. Generally speaking dogs that are fed on scraps and left-overs from the table do not do as well as ones that are fed *as dogs*, for the reason that human food is usually too well salted and spiced, thus putting an undue load on the kidneys.

d. *Hormonal Dysfunction*: This is encountered largely in spayed and castrated animals and in entire dogs and bitches with hypogonadism. In these cases pruritus can be intense and the skin is usually dry with a sparse hairy coat and brittleness of claws. The good response of most of these to administration of thyroid extract or of sex hormones suggests a deficiency of such hormones as the direct cause. Undesirable side-effects of androgen or oestrogen therapy could be avoided by the use of physiologically balanced male and female hormones together.* There is also the phenomenon of alopecia and pruritus in dogs with Sertoli cell tumours of the testes, frequently accompanied by feminization. This is dealt with by castration or androgen therapy, and provides another instance of the effect of hormonal deficiencies.

e. *Breeding and Heredity*: Some breeds are certainly more prone to skin irritation and eczema than others. Scottish Terriers, Cairns, Sealyhams and Wirehaired Fox Terriers deserve special mention as susceptible types. Alsatians (German Shepherds) show some proneness but probably this is due to their thick undercoat. Boerbulls in South Africa frequently suffer from eczema but this is due probably more to unbalanced diet, parasitism and neglect generally than to hereditary factors.

f. *Climate and Season*: Our dry and generally warm climate contributes quite considerably to the incidence of skin irritation. We often hear the complaint from owners that their dogs itch and scratch all through the summer months. This can be only partly due to unsuitable food, the higher incidence of fleas and other parasites, the ubiquity of allergens, and hormonal stresses. Heat in itself must be an important factor.

g. *Focal Infections*: These can be most obscure but have

* 'Androgynon' (Schering).

often to be looked for and suitably dealt with. Such pockets of pus are located in anal glands, prostate, uterus (pyometra), roots of the teeth, sinuses, interdigital spaces and other parts of the body.

h. *Errors of Management*: The most usual trouble in this category is the fastidious owner who has an exaggerated idea of cleanliness and whose dog is bathed regularly in disinfectants of various kinds and washed with soap. The soap need not even be particularly irritant but the skin gets dried out and more itchy than ever. This results in the application of insect powders and more frequent washings. The owner ends up in desperation with an eczematous and very miserable dog at the veterinarian's rooms.

Trigger Mechanisms

When eczema-proneness is already present the trigger needed may be slight: one or more fleas, lice, ticks, mites, grass awns, loose hairs, dirt or very mild trauma. The dog scratches the spot involved which becomes irritable, erythematous and even swollen with early oedema, inducing more violent scratching. And the more scratching, the more skin reaction. (It is to be noted that eczema lesions are always in areas that can be reached with the hind legs or the mouth.) This vicious spiral is maintained and built up till after a few hours there may be a red, weeping, exquisitely painful, swollen and denuded area, a patch of eczema.

Treatment

It will be clear from the foregoing that to treat eczema you have first of all a diagnostic problem which embraces the examination of the whole dog. There is hardly ever any tendency to spontaneous cure and almost always there is (a) a vicious cycle to break, and (b) an eczema-proneness to remove.

Selfmutilation by the dog must be stopped immediately. As lesions are often intensely painful resentment must be removed by one of the tranquillizers. Chlorpromazine* in a dosage of 2 mgm per kilo intramuscularly has been found more satisfactory than anaesthesia. This sedation can if necessary be maintained by peroral dosage. Even a short-acting anaesthetic such as thiopentone sodium** causes some small degree of disorientation. Anaesthetic ointments are generally speaking in the early acute stage, of little value as the act of applying them is too painful.

A soft ointment made from zinc oxide and kaolin has given excellent results in our hands and has the great merit of cheapness. Antihistamine creams or lotions of antihistamine and calamine***

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* Largactil (May & Baker).
** Intraval (May & Baker).
*** 'Caladryl' (Parke, Davis) or 'Anthical' (May & Baker).

have also proved successful. More recently adrenal corticoid creams and ointments* have become available and though comparatively expensive are most definitive in their action. A recent introduction is also a cream containing an anticholinergic drug.**

Systemic attack of the acute skin condition has been carried out in three ways:

a. Sedation with suitable tranquillizers.
b. Antihistamine therapy (parenterally or by the mouth).
c. Adrenal corticoids such as prednisone*** or prednisolone**** intramuscularly.

These, while in an ascending order of cost, are also proportionately more effective. The choice of medicament would be actuated by the severity of the condition.

The basic condition of itchiness or pruritus, frequently the only complaint of the owner, is however the more difficult problem. Where it is very distressing and of long standing, particularly where the breed is the major factor, the situation may well need heroic measures. Where irreversible organic changes are present and responsible, the condition becomes correspondingly resistant to successful treatment.

Fundamentally the task is to produce a normal dog living under normal conditions. Allergies have to be identified if possible and allergens eliminated. Chronic organic disease and verminosis should be suitably treated. The diet should receive serious attention, particularly in the direction of elimination of refined carbohydrates of all kinds and substitution of high protein and crude or unrefined cereal products. Potatoes should be divested of the prejudice against them and used as a valuable part of the dog's food. Fat should be provided at a rate of 5-10% of the diet and milk fed ad lib. Raw linseed oil, ½-1 teaspoon per day improves the pliability of the skin and removes the dryness often responsible for the pruritus.

Grooming daily at a regular hour in preference to baths is the most important cleansing procedure. The cutaneous circulation is healthily stimulated and loose hairs removed. The massage promotes the growth of a healthy coat.

It remains true that corticoid therapy, topically or systemically, and in many cases antihistamines used similarly, may stop skin irritation while they are being used. It may also be true that they break the vicious spiral of eczema and allow an established skin lesion to heal. But in many cases they can only be palliatives and one is forced to go back to fundamentals and to try and return to the owner an entirely normal dog which is no longer "prone" to eczema, and no longer itching.

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* 'Magnacort' (Pfizer).
** 'Variton' (Schering).
*** 'Meticorten' (Schering).
**** 'Deltacortril' (Pfizer).

Discussion at Meeting

1. *Dr. H. P. Steyn* (Chairman) in thanking the speaker expressed the view that the subject was a most timely one. He asked whether aspirin had been tried at Onderstepoort as a systemic treatment.

Reply: It had not, but it was considered likely to be useful in selected cases. Aspirin did of course raise the pain threshold and there were people who thought that it might have a mild corticotrophin-like action.

2. *Dr. J. M. M. Brown* asked whether senility should not be listed as an aetiological factor. He also inquired into the use of methyl testosterone, glycyrrhetinic acid, intravenous procaine and colloidal calcium.

Reply: Senility did not in the opinion of the speaker merit separate listing as he regarded it merely as the sum of failure of various organs. Senile hypogonadism was an example of such failure. In such cases sex hormones certainly had a rejuvenating effect. Glycyrrhetinic acid was the active principle of liquorice and had according to medical literature given irregular results in the United Kingdom. At Onderstepoort a few chronic cases had responded well to peroral dosage of extract of liquorice which has a desoxycorticosterone-like action. Intravenous procaine had not been used as it had not been found necessary and was not entirely free from objection. Some cases responded well to 'Calci-ostelin' injections over say 5-7 days.

3. *Dr. D. G. Steyn (Sr.)* very warmly supported the speaker's ideas on the influence of diet on allergy as it conformed with his own experiences over a considerable period.

4. *Dr. S. W. J. van Rensburg* described a case of chronic pruritus and eczema which had made a complete recovery on being fed potatoes as the sole source of carbohydrate. All medicinal treatment had previously failed.

5. *Dr. D. J. Louw* had come across a case or two of children who reacted unfavourably to antihistamines.

Reply: Such cases were known to occur but were doubtless a rarity.

6. *Dr. L. R. Morford* described cases where dogs apparently cured of pruritus immediately started scratching the moment they saw their owner. What about psychosomatic causes?

Reply: In such cases the dog should find a new owner.

7. *Dr. K. van der Walt* stated that he had frequently encountered allergy from milk. He regarded acute eczema as an "alarm reaction" in the terminology of Selye.

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ASUNTOL,* A NEW INSECTICIDAL COMPOUND CAPABLE OF CONTROLLING ALL SOUTH AFRICAN CATTLE TICKS

by

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Pretoria

Adequate control of cattle ticks still constitutes a very difficult problem in Southern Africa in spite of numerous new insecticidal compounds which have been discovered during the last fifteen years. The Blue Tick (*Boophilus decoloratus* Koch) in particular has become a serious problem of recent years as this species has developed a high degree of resistance to practically all dipping materials.

In 1937 a strain of this species with a marked tolerance to arsenical preparations was discovered in the East London area (du Toit et al., 1941). Subsequently it developed a marked resistance to B.H.C. several years later (Whitnall et al., 1949). At the same time this arsenic- B.H.C.- resistant Blue Tick displayed a definite degree of resistance to Toxaphene and was shown to be resistant to Dieldrin and related compounds (Fiedler, 1952).

Resistance to B.H.C. combined with a fairly strong tolerance to Toxaphene was demonstrated practically all over Southern Africa during the following years, and it was proved that this type of resistance was not necessarily always coupled with resistance to arsenic (Bekker, 1953). As all the resistant strains remained fully susceptible to D.D.T., and related compounds, such as Methoxychlor and Rhothane (DDD), D.D.T. preparations became the dips of choice for the control of resistant Blue Ticks.

It was, therefore, a cause of great concern when D.D.T. became less effective on certain farms in the East London district (Blomefield, 1954) and solid resistance to this insecticide was demonstrated subsequently (Whitehead, 1956).

Even prior to the advent of D.D.T. resistance, adequate tick control was difficult owing to the different patterns of specific susceptibility to insecticides displayed by different species. It may be anticipated that the situation is likely to become more difficult as resistance is spreading generally, with the result that conventional dipping materials become obsolete.

Extensive research was, therefore, undertaken during the last two years to meet this rather unpleasant development. Eventually a compound was found amongst the organic phosphates which, at low concentrations, was highly lethal to all species of cattle ticks without any hazards to livestock. This compound, so

* Asuntol — Agridip.

far known as Bayer 21/199, is a chloromethyl-coumarin-diethyl-thiophosphate, now referred to as Asuntol. It is very stable to alkali and does not readily hydrolyse in water.

Its efficiency against South African ticks was established by in-vitro dipping experiments. Fully engorged female ticks were used, freshly collected from cattle at the Pretoria abattoir. This is the most resistant stage, and also allows observations to be made on egg-laying and hatching of larvae. All ticks were immersed for one minute whilst the dipping fluid was stirred constantly, then allowed to dry off in a petri dish, and transferred to clean tubes to be observed under constant climatic conditions. In this way 50 to 100 specimens were used for each single test with Blue Ticks, and 20 to 25 specimens with Bont Ticks (*Amblyomma hebraeum* Koch) and Brown Ear Ticks (*Rhipicephalus appendiculatus* Neumann). Only comparatively small numbers of engorged Red-legged Ticks (*Rhipicephalus evertsi* Neumann) and Bont-legged Ticks (*Hyalomma spp.*) could be obtained and included in the experiments. Each experiment was repeated 3 to 5 times. The average results are given in Table 1.

TABLE 1: The effect of different insecticides on fully engorged females of *Boophilus decoloratus, Amblyomma hebraeum,* and *Rhipicephalus appendiculatus* applied in a dip.

(a) *Boophilus decoloratus*:

Insecticide	Concentration in ppm.	Peercentage females killed	Percentage egg batches deposited	Percentage egg batches hatched
Control	—	11.0	89.0	86.0
Toxaphene emuls.	2500	64.5	35.5	20.6
D.D.T. Emuls.	2000 pp	90.5	9.5	0.7
Lindane Emuls.	200	65.8	34.2	20.9
B.H.C. Emuls.	200 gamma	61.2	38.8	15.3
Asuntol W.P.	25	39.7	60.3	27.6
Asuntol W.P.	37.5	75.2	24.8	2.0
Asuntol W.P.	50	84.8	15.2	4.1
Asuntol W.P.	75	87.8	12.2	1.2
Asuntol W.P.	100	96.8	3.2	0.7
Asuntol W.P.	150	98.4	1.6	Nil
Asuntol W.P.	200	97.6	2.4	Nil
Asuntol W.P.	400	100	Nil	Nil
(b) *Amblyomma hebraeum*:				
Controls	—	9.9	90.1	
Toxaphene Emuls.	5000	28.6	71.5	
Toxaphene Emuls.	2500	29.2	70.7	
B.H.C. Emuls.	200 gamma	39.6	60.5	
Asuntol W.P.	50	10.0	90.0	
Asuntol W.P.	100	38.6	61.4	
Asuntol W.P.	150	77.8	22.2	
Asuntol W.P.	200	75.4	24.6	
Asuntol W.P.	300	91.2	8.0	
Asuntol W.P.	400	97.0	3.0	
Asuntol W.P.	800	100	Nil	

(c) *Rhipicephalus appendiculatus*:

Controls	—	4.8	95.2	91.9
Asuntol W.P.	100	77.2	22.8	8.5
Asuntol W.P.	200	98.5	1.5	Nil
Asuntol W.P.	400	100	Nil	Nil

According to these experiments Asuntol wettable powder compares most favourably with the conventional dipping materials, which were applied as emulsions.

97.1% and over of Blue Ticks, not resistant to B.H.C. and D.D.T., were killed and practically no eggs were deposited at concentrations of 100 ppm. a.i. and upwards. Asuntol at 35.7 ppm. was equivalent in efficacy to 200 ppm. gamma B.H.C., and at 100 ppm. was comparable with 0.2% (2000 ppm.) pp' D.D.T.

91.1% and over of the Bont Ticks were killed by concentrations of 300 ppm. and upwards, as against a mortality of 29% using the present recommended strength of 0.25% Toxaphene. The recommended Toxaphene concentration is only slightly more effective than 100 ppm. Asuntol.

98.5% and over of the Brown Ticks were killed by concentrations of 200 ppm. and upwards. The susceptibility of Red-legged Ticks and Bont-legged Ticks to Asuntol was similar to that of the Brown Bar Ticks.

The residual effect of the compound on the hair of cattle was determined by two tests. Four Friesland cows heavily infested with Blue Ticks were handsprayed with 200 ppm. Asuntol, and scrapings on the neck were taken prior to treatment and again 3, 5, 7 and 10 days thereafter. Four untreated cows served as controls. All animals were grazed together, and the results for the sprayed group were as follows:-

(a) No living *larvae* were encountered on the treated animals during the first three days after application, and up to the seventh day their numbers were small compared with the controls, but many dead larvae were present. On the tenth day equal numbers of both groups were counted.

(b) No newly moulted and still unengorged *nymphs* were found up to the tenth day, only fully engorged specimens in the pre-moulting stage.

(c) Moulting of adults took place during the entire period of observation as the well protected nymphs of the pre-moulting stage were not killed by the treatment. Only newly moulted adults, however, were observed up to the fifth day, and they died within 24 hours when left on the hairs of the scrapings, whereas those of the controls were still fully alive after this period. Engorgement of females was noticed after seven days, and fully engorged specimens made their appearance again on the tenth day.

The test revealed that all larvae crawling onto the animals were killed by 200 ppm. until the fifth day after application and that substantial control was effected up to seven days.

In a second test four cows were handsprayed with 200 ppm.

Asuntol wettable powder for nine consecutive weeks, and skin scrapings of the neck were taken prior to each application. The control group had to be sprayed after 5 weeks as infestation became too heavy. This test demonstrated again that a concentration of 200 ppm. provides a sufficiently long residual effect to prevent development of Blue Ticks beyond the larval stage on heavily infested grazing.

The above mentioned findings were confirmed by two field trials conducted in the bushveld of the Transvaal with over 300 dairy cattle where 200 ppm. Asuntol applied in a spray race and by handspraying at weekly intervals gave complete control of resistant Blue Ticks. A large decrease in numbers was achieved by the first treatment, and nymphs and adult ticks were eliminated entirely by the third application. Equally good results were also obtained with only half this concentration, i.e. 100 ppm. The in-vitro dipping experiments with engorged females have shown that the other species, and especially the Bont Tick, possess a slightly lower degree of susceptibility to this compound than the Blue Tick.

All adult specimens of the other species, however, such as *Amblyomma hebraeum, Rhipicephalus evertsi, Rh. appendiculatus* and *Hyalomma spp.* which were on the animals at the time of treatment were killed in situ by sprays containing 200 ppm. Asuntol and remained attached to the skin. Numerous dried up unengorged as well as engorged females were found on the animals every week. The residual effect of this concentration on the bare and semi-bare parts of the animals' body was not sufficient, however, to affect the adult ticks attaching themselves several days after treatment.

Consequently, higher concentrations were tested against Bont Ticks and those species which engorge very quickly during the hot summer months, and 400 ppm. applied weekly provided excellent control of all cattle ticks and prevented engorgement of females completely.

Similar results were also obtained by a weekly spray with 200 ppm. followed by handdressing of those parts of the body where ticks tend to congregate, with 400 to 500 ppm.

The toxicity of Asuntol to warm-blooded animals is so low, that the compound can be used safely on livestock. The acute oral toxicity to white rats is about that of gamma B.H.C. On the other hand it is less toxic than gamma B.H.C. to calves 1 to 2 weeks old (Radeleff et al., 1955). The great asset of the compound is, however, its relatively low dermal toxicity. Since Asuntol is insoluble in water and only slightly soluble in most of the organic solvents, it does not penetrate easily through the skin like other chlorinated hydrocarbon or phosphorus insecticides. The maximum non-toxic concentration for dairy calves 1 to 2 weeks old was found to be 0.2% of the active ingredient, and the minimum toxic strength 0.5% which surpasses many times that of gamma B.H.C.

The following tests proved the safety of this compounds under local conditions.

Two groups of laboratory white rats were dipped 14 and 16 times, respectively, at weekly intervals for one minute in a concentration of 1,000 ppm. Asuntol without ill effects. These tests are of great significance as rats lick and clean their coats as soon as they become wet. A large amount of the insecticidal deposit on the hair was ingested in this way.

Eleven dairy cows and four to eight calves from one and a half months old were handsprayed with a concentration of 200 ppm. for 37 weeks during two consecutive seasons. The animals were inspected daily and no ill effects were noticed.

A herd of dairy cattle, consisting of 210 Friesland cows, 100 heifers and calves of all ages, and two bulls were machine-sprayed with 200 ppm. for 13 weeks, and the heifers and calves were hand-dressed each week with 500 ppm. The health and milk production of the entire herd remained unchanged.

SUMMARY

Laboratory and field trials have shown that Asuntol is a very suitable and safe agent for the control of all cattle ticks including the resistant Blue Tick (*Boophilus decoloratus*). A concentration of 200 ppm. applied at weekly intervals is sufficient to achieve complete control of Blue Ticks, whereas 400 ppm. provides similar control of all two- and three host species such as *Amblyomma hebraeum, Rhipicephalus appendiculatis, Rh. evertsi* and *Hyalomma spp.* The concentrations applied are also capable of killing in situ all adult ticks on the animal at the time of treatment. The toxicity of Asuntol is too low to endanger the well-being of animals treated regularly.

ACKNOWLEDGEMENT

The authors wish to acknowledge gratefully the assistance of Dr. W. J. Wheeler, who supplied the adult ticks for the laboratory experiments. Further extensive tests, which confirm the above findings, were carried out in the Eastern Cape Province by Drs. L. C. Blomefield and J. de Wet, to whom thanks are due for assistance and advice in this work.

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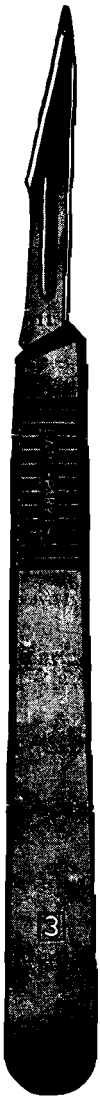

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Jl. S.A.V.M.A. 28 (3), 1957

THE INSTITUTION OF A REGISTRY OF NEOPLASMS IN DOGS IN SOUTH AFRICA

by

GILLES DE KOCK

(Liesbeek Cancer Clinic)

In an address at the meeting of the Cape Western Branch of the S.A.V.M.A. on the 27th May this year, reference was made to the institution of a Registry of neoplasms in dogs in South Africa.

The study of neoplasms in dogs is not only of value in investigation of the problem in these animals, but is undoubtedly of great comparative value and importance to the study of neoplasms in man. There appears to be a need for a more concise and intensive survey so that more light can be shed, not only on the nature and incidence of these tumours in different breeds of dogs, but also on the problem of their histogenesis and the histological diagnosis. Uncertainty still exists in respect of the histological nomenclature, especially in those neoplasms mainly composed of undifferentiated cells.

It would appear that the greatest number of neoplasms of dogs occur between 7 and 14 years of age and it is estimated that the cancer age of the dog is from about 6 years onwards and increasingly noticeable by 10 years.

Since studies on neoplasms in dogs commenced at the Liesbeek Cancer Clinic, Cape Town, at the end of 1956, a number of interesting tumours (± 50) have been investigated. In a few dogs more than one neoplasm was observed in the same dog. Apart from this, a number of cases of hyperplasia, hypertrophy or neoplasia have been observed in such organs as the spleen, prostate, adrenals and testes of aged dogs. Some of these lesions were only observed at post mortem on clinically healthy dogs, killed on account of old age. This should be borne in mind when such a registry is instituted.

The valuable information and material so far submitted by veterinarians of the Cape Peninsula have been utilised for pathological, cytological, biological and biochemical studies at the Liesbeek Cancer Clinic where adequate facilities have been provided for these investigations. In the attached form veterinarians submit the information desired in every post mortem, especially in respect of breed, age, and the more important clinical observations since the tumour was observed. This form may perhaps be utilised as a basis for discussion as regards the information desired in a registry of neoplasms. In the case of specimens of biopsies and the tumours surgically removed, full particulars of the site and nature of the tumour are required.

At the autopsies particular attention is paid to the following:-

(1) Site, nature, size, etc., of the primary tumour and the problem of metastasis is carefully investigated. It has been reported that metastasis in dogs is restricted and mainly confined to the lungs and regional lymph nodes. The liver apparently is not as predominantly affected as in man. In two cases at the Liesbeek Clinic metastasis occurred in the ventricle of the heart.

(2) In the majority of cases a study was made of blood and bone marrow smears and imprint smears from primary and secondary neoplasms. These have been most helpful, especially in cases of leukaemia, lymphosarcoma, and reticulum-cell sarcoma.

Amongst the cases so far studied at the Liesbeek Clinic, sarcomas of the skin of the dog form an interesting but puzzling group and difficulties were encountered in differentiating these from the so-called "transmissible venereal tumour" in dogs. The clinical course of some of these sarcomas was at variance with the histological malignancy. In an undescended testicle an adenoma was observed and cases of chondrosarcoma were encountered in the humerus and mandible.

Adenoma and carcinoma were diagnosed in the liver, kidneys, parotid salivary gland, adrenal cortex and intestine. In one case there was a perianal adenoma and in another a carcinoma of the peri-anal region, with metastasis in the regional lymph nodes.

The following books, besides several references, were of great help in these studies:-

I. E. Cotchin (1956) — Neoplasms of the Domesticated Mammals (A Review). Commonwealth Agricultural Bureau.

II. C. Jackson (1936) — The Incidence of Tumours of Domestic Animals in South Africa. Onderstepoort J., Vol. 6, pp. 1-460.

III. R. M. Mulligan (1949) — Neoplasms of the Dog. The Williams & Wilkens Co., Baltimore.

IV. R. A. Willis (1953) — Pathology of Tumours (Second Edition). Butterworth & Co., Ltd., London.

REPORT ON DOG TUMOUR

FROM: (Name and address of Veterinarian). Ref. No.....

TO:

.....
.....
.....

The Director,
Liesbeek Cancer Research Clinic,
Liesbeek Road,
Rosebank,
Cape Town.

1. BREED OF DOG: .....
2. SEX AND AGE OF DOG: .....
3. WHEN WAS TUMOUR FIRST BY OWNER: .....
4. CLINICAL DIAGNOSIS:
   (a) Any Marks of Injury .....
   (b) Anaemia, Emaciation, Cachexia .....
   (c) Any recognisable Disease or other symptoms .....

5. DATE OF SURGICAL REMOVAL OF TUMOUR .....

6. ANAESTHETIC USED: .....

.....

PARTICULARS OF TUMOUR AND AUTOPSY FINDINGS: .....

(a) Site .....

(b) Size of Tumour .....

(c) Infiltration of adjoining tissues .....

(d) Metastasis (Lymph Nodes, Liver, Lung, etc.) .....

(e) Other P.M. Observations .....

.....

8. SMEARS SUBMITTED:

(a) Blood Smear from: .....

(b) Imprint Smears from: .....

(1) Tumour .....

(2) Other organs (state which) .....

9. DATE AND TIME OF DESPATCH OF LIVE DOG OR SPECIMENTS: .....

SIGNATURE OF VETERINARIAN .....

A CENTURY AGO

Dr. H. H. Curson has recently come across a very interesting advertisement from the Natal Government Gazette of 29/1/1856 which will be of interest to our members. It reads as follows:-

"William Jarman, Veterinary Surgeon, Member of the Royal College of Veterinary Surgeons, Edinburgh, begs most respectfully to intimate to the owners of horses and cattle etc., that he has commenced practice in the above profession and trusts by unremitting attention to cases committed to his care, to obtain a practice, which will be his constant study to merit.

Good stabling for sick and lame horses etc. Residence — Mr. Marshall's Livery and Bait Stables, Church St., Pietermaritzburg, Nov. 26th 1855.

Jarman was the earliest private practitioner in Natal and Dr. Curson wonders what happened to him subsequently.

Editor.

Jl. S.A.V.M.A. 28 (3), 1957

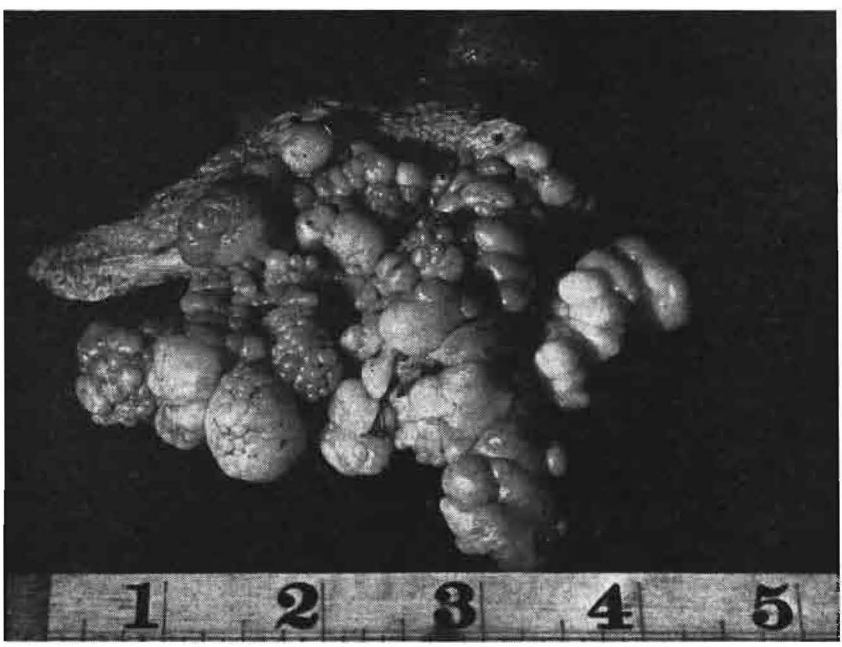
CASE REPORT

A CASE OF RUMINAL FIBROMATA

D. H. G. Irwin*

The case history recently reported by Benson reminds the writer of a similar case encountered a few years ago. The subject was the only poor animal in a group of Friesland heifers. For about three weeks prior to examination she had shown varying degrees of ruminal tympany. On clinical examination no cause for the poor condition and tympany could be found, whereupon a tuberculin test was carried out. This proved to be negative. An exploratory rumenotomy was then undertaken, and a suspected neoplasm was identified near the oesophageal groove. When given a doubtful prognosis the owner requested euthanasia, and at post mortem the accompanying photograph was taken. This gives a good impression of the size and shape of the fibroma-like tumour. Histological and cultural examinations were not undertaken.

Ruminal Fibromata



* Late of Somerset West, at present Ontario Veterinary College, Guelph, Ontario, Canada. Reference: Benson J. A. Vet. Rec., 69, 417, 1957.
Photograph: Rembrandt Studio, Somerset West.

Sitting Pretty

Slow recovery from distemper . . . 'flu that lingers on and on: how often such cases of retarded convalescence can be blamed on a history of faulty feeding. It is for just such cases that MINADEX is made. The protective and formative vitamins A and D are present at high concentration in MINADEX; the traditional tonic glycerophosphates are present too, plus iron for the blood. And because MINADEX is a genuinely *palatable* syrup, it is taken readily by small animals.

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CASE REPORT

THE USE OF CHLORPROMAZINE HYDROCHLORIDE ("LARGACTIL" — MAYBAKER) IN THE ANAESTHESIA OF A BROWN BEAR (URSUS ARCTOS).

A. J. LOUW

Municipal Veterinarian, Bloemfontein

Katrina, a 33 year old brown bear of the Bloemfontein Zoo, developed what appeared to be a raw sore on the inside of her right thigh. The affected area increased in size and after three weeks observation it was so noticeable to the general public that I decided to anaesthetise her for a closer examination and if necessary, surgical treatment.

She was starved for 24 hours and coaxed into a "squeeze box" — a strong portable iron cage of which the one lateral side is movable and any animal inside could be immobilised by being pressed against the opposite side. She weighed approximately 300 lbs. and was given 10 x 25 m.g.m. tablets of 'Largactil' per os. These were crushed, mixed with honey and bread crumbs and readily taken.

After a time lapse of two hours, the maximum allowed for the drug to take effect when administered by the oral route, slight symptoms of sedation were noticed. When aroused she however displayed a very nasty temper and apparently resented the close confinement of the small cage.

She was then "squeezed up" and anaethetised with a pentobarbitone sodium solution ("Sagatal" — Maybaker). This was administered intravenously in a vein of the hindleg corresponding in position to the saphenous vein of the dog. The computed dose was 60 c.c. of the solution (1 gr. per c.c.) but after the slow administration of 10 c.c. the animal was completely relaxed and after a further 5 c.c. a plane of deep surgical anaesthesia was reached.

The lesion on closer examination, proved to be a clearly defined superficial tumour, oval in shape and measuring 7 x 5 x 2 inches. The surface was raw and suppurating and the entire growth was completely detached from the subcutaneous tissues. The regional lymphatic glands appeared to be unaffected.

A melanotic sarcoma, weighing $7\frac{1}{2}$ ounces was removed under as stringent as possible aseptic conditions. The skin wound, 8 inches in length, was closed with interrupted mattress sutures, using a fine nylon thread.

Signs of regaining consciousness were noticed one and a half

hours after the intravenous injection was administered and an hour later she had fully recovered and was standing up.

She made an uneventful recovery, the wound healing per primam with the sutures left in situ. The operation was performed more than seven months ago and no signs of secondary lesions developing have so far been noticed. Apart from an occasional attack of rheumatism and an intense dislike for the writer, she is still in the best of health.

Discussions

The experience of the writer has been that chlorpromazine hydrochloride is of definite value as a premedicant to general barbiturate anaesthesia, especially when the subjects are wild and excitable, as encountered in Zoo practice. Not only is the handling made easier, but the amount of anaesthetic used less with subsequent quicker and easier recovery. The same has been experienced in two cases where monkeys were anaesthetised with "Sagatal" and "Largactil" for teeth operations. I also recall one case where an adult lion was anaesthetised with Sagatal without the premedicant. In spite of being given only half the computed dose intravenously it only regained full consciousness sixteen hours later and extensive abrasions to its head occurred whilst it knocked about in the final recovery stages. In another instance an aged and very toxic and fevered "Liger" never regained full consciousness for two days after pentobaritone sodium anaesthesia (i.v.) and died whilst in a coma.

Troughton and co-workers advocate the use of chloropramazine hydrochloride as a premedicant to patients in the following catagories:-

(1) Aged patients.

(2) Painful conditions where surgery is to be practised.

(3) Fevered animals.

(4) The presence of thyroid disease.

(5) Toxaemic conditions.

(6) Excitable animals.

ACKNOWLEDGEMENT

The Town Clerk of Bloemfontein for permission to publish this report.

The Director of Veterinary Services for doing the histopathological examination of the tumour.

Messrs. May and Baker Ltd., for making available to the author their products "Sagatal" and "Largactil".

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PRESS NOTICE.

THE WELLCOME TRUST

FOOT-AND-MOUTH DISEASE IN KENYA: GRANT FOR RESEARCH.

The Wellcome Trustees have made a grant of up to £80,000 to the Government of Kenya towards the cost of building and equipping a special establishment for research on foot-and-mouth disease in that country. The institute which will be sited in the Nairobi area will be under the control of the Kenya Veterinary Department and will be in charge of Mr. J. W. Macaulay, F.R.C.V.S., B.Sc., D.V.S.M. It will be known as 'The Wellcome Institute for Research on Foot and Mouth Disease.'

PERMANENT COMMITTEE FOR THE INTERNATIONAL VETERINARY CONGRESS

Utrecht, January 28th, 1957.
Biltstraat 168.

To the Members of the
Permanent Committee for the International Veterinary Congresses.
Dear Colleague,

From the Secretary of the International Association of Veterinary Food-Hygiene (I.A.V.F.H.) I received the following report on the First Symposium of the I.A.V.F.H., which was held in Utrecht from 27th of August to 1st of September, 1956, for your information. This report gives in brief outline a clear view of this Symposium.

Yours sincerely,
Dr. JAC. JANSEN,
General Secretary.

In accordance with the XIth. Resolution of the XVth International Veterinary Congress, Stockholm, 1953, passed on the proposal of section VIII (Food Hygiene and Public Health), the International Association of Veterinary Food Hygienists (I.A.V.F.H.) was founded in the autumn of 1955. At this moment 29 countries joined this Association.

The first Symposium organized by the I.A.V.F.H. was held in Utrecht (the Netherlands) from 27th August to 1st September, 1956. It was attended by 172 veterinarians working in the field of food hygiene, from 24 countries. The Symposium was opened by Prof. Dr. P. Muntendam, Director General of Public Health in the Netherlands. Prof. Dr. C. Sanz Egaña addressed the opening session on behalf of the Permanent Committee for the International Veterinary Congresses.

After a general lecture about the task of the veterinarian in the field of food hygiene by Prof. Dr. M. Lerche, Germany, the following subjects were dealt with:-

1. Principles of meat hygiene;
2. Principles of bacteriological meat inspection;
3. Principles of meat preservation;
4. Principles of fish hygiene;
5. Principles of egg hygiene;
6. Principles of milk hygiene;
7. Principles of poultry and rabbit inspection.

For every subject a general lecturer had been invited by the Board of the I.A.V.F.H. Moreover 30 communications were read by verterinary specialists. After each subject there was an opportunity for discussion.

The proceedings of the Symposium are available at the secretariat of the I.A.V.F.H., Sterrenbos 1, Utrecht, the Netherlands. Price Df.1. 12.—

The lecturers and the subsequent discussions led to the adoption of the following resolutions:-

1. For the protection of public health and to prevent misrepresentation or adulteration it is necessary that all food of animal origin be controlled by Veterinarians. These are: meat, poultry, game, seafood, milk, eggs and their food derivatives.
2. To encourage international trade it is important that inspection methods and standards for food, particularly meat-control, by both importing and exporting countries be uniform.
3. It is necessary to establish a board to attempt to determine whether a standard can be established on which food-hygiene laws could be based.
4. Countries exporting eggs and egg-products should take action to ensure that these products are free of vital Salmonella organisms.
5. It is desirable that independent chairs for Veterinary Food-Hygiene and Technology should be established at all veterinary faculties or colleges, as some countries have.
6. All intimations concerning the calling of a symposium of the I.A.V.F.H. shouldbe sent to Government and Veterinary Authorities and not only published in professional periodicals.
7. Papers for future symposiums of the I.A.V.F.H. should take into consideration preferably new and unpublished works, critical reviews of present knowledge and topics relating to problems concerned with education, organization and administration of Meat and Food-Hygiene papers.
8. Bearing in mind the importance from the point of view of Hygiene and Public Health, of certain recent trends in the use of insecticides and fungicides, pharmaco-dynamic agents and antibiotics, new approaches to sterilization, etc., considered in the light of their inherent danger for milk production, the Symposium recommends that these various problems be studied by all interested countries and the results submitted to the next Symposium.

The Committee of the I.A.V.F.H., the members of which were nominated for 4 years, consists of:

Dr. A. Clarenburg	President
Prof. Dr. M. Lerche	Vice-President
Mr. C. H. Pals	Vice-President
Dr. G. Brévot	Vice-President
Dr. E. H. Kampelmacher	Secretary-Treasurer

LETTER TO THE EDITOR

TARR, PAINE & PARTNERS,
340 Longmarket Street,
PIETERMARITZBURG,
Natal.

18th July,1957.

The Editor.
Journal of S.A.V.M.A.,
ONDERSTEPOORT.

Dear Sir,

I would like to give you details of an outbreak of mass bloat in a herd of cows in order that other practitioners may benefit from my observations.

The farm is in the Merrivale district, and there were approximately 70 Friesland cows in milk. The cows were receiving a liberal ration of maize ensilage at milking time, twice a day, and were allowed to graze on clover and ryegrass pastures for two hours in the morning, after which they were put into paddocks containing Kikuyu grass.

On 14.11.56 the supply of ensilage was exhausted and the cows had nothing to eat at the morning milking. As usual, they were then put into a pasture containing pure clover for two hours. At the end of this period they were removed and put into the Kikuyu paddock. It was then noticed by the farm manager that several animals had become bloated. Turpentine was dosed with no effect. The manager went to fetch a trocar and in the short interval he was away six cows died. The trocar was inserted, but the froth continually blocked the canula. In order to save the remaining cows, the manager decided that drastic action was necessary, and proceeded to cut the cows open with a penknife. Incisions about 3 inches long were made through the flank and rumen. Natives were instructed to pull out the rumen contents with their fingers. Thirty-two cows which had hoven were cut. The manager then phoned for veterinary assistance. When I arrived at 12 o'clock, I realised that this task was too much for one person and asked that every veterinarian in the district be contacted. Eventually another four came namely Drs. Wacher, Canham, du Casse and Jonker.

The cows were now over the bloat. One was down but the remainder had been driven to the cowshed and tied up. The flank wounds were badly contaminated by rumen contents and in several cases the peritoneal cavity contained ruminal contents. In one cow it was pulled out in handfuls.

Suturing was done mainly without any anaesthetic. Some cows were given planocaine locally and a few were dosed with chloral hydrate. Most of the rumen wounds did not coincide with the flank ones due to emptying of the rumen which caused the wound to be pulled medially and backwards. This meant that the flank wound had to be enlarged in order to pull the rumen wound to the exterior.

Suturing was done as for a rumenotomy using chromic gut but it was impossible to clean the cut surfaces and remove all the ruminal contents. Antiseptic powder was used liberally and each cow was given an injection of Benapen.

The last cow was stitched at 7 p.m., approximately eight hours after being cut. It was noticed that reddening of the peritoneum was already evident.

Between six and ten days later, the wounds of 16 cows broke open and had to be resutured. Two cows developed generalised peritonitis and had to be destroyed. All the remaining 30 cows recovered and are apparently in good health.

Yours faithfully,

R. A. SOLOMON.

LETTER TO THE EDITOR

RUTGERS UNIVERSITY,
Nichol Avenue,
New Brunswick, New Jersey.
June 24, 1957.

The Editor,
Journal of the South African Veterinary Medical Association,
P.O. Onderstepoort, Transvaal,
Union of South Africa.
Dear Sir,

LENGTHENING OF THE VETERINARY COURSE

I gather that important discussions are going on in South Africa among the "powers-that-be" concerning certain aspects which affect the future of our profession. I understand that one of the things that is being considered is the lengthening of the veterinary course given at the University of Pretoria (Onderstepoort).

I am satisfied that the course being given at Onderstepoort now (and in the past) is (and has been) an excellent one. I think that on the whole the graduates from there have measured up well to graduates from the best schools in the world. Their special training in local conditions is especially valuable anywhere on the African continent.

Although the standard of the graduate has been high we should not be complacent about it. Let it not be forgotten that the veterinary profession in other parts of the world has made tremendous strides, particularly in the past ten years. Our colleagues in South Africa will have to plan wisely and with foresight to keep pace with this competition and progress. I wish to make the plea that the standard of the training at Onderstepoort be never allowed to become inferior in any way and that improvement be continuously sought. I think that the time has come to lengthen the course, which I am sure is already under consideration.

Assuming that the course will be lengthened there will no doubt be much discussion as how to utilize the extra time. I would like to offer some thoughts which I hope may be helpful in this matter, but I also feel that graduates receiving some of the extra courses I mention would be better equipped to play a more effective role in this modern world. I may say that these opinions are entirely my own and are based on personal observations.

(1) **Nutrition.**

This subject is of enormous importance in both health and disease, and warrants more attention. When one considers that a high percentage of animals, especially cattle that die a natural death in South Africa, starve to death, this fact is of major importance from this aspect alone, but it is also realised that nutrition plays a very important role in the development and has a profound influence on the course of many disease states including the infectious diseases. I feel that the course in nutrition should be extended and should be one of the major basic subjects of a veterinary course.

(2) **Animal Husbandry.**

This should be stressed more than it is and the students should be taught to think more in terms of health rather than with an over-emphasis on disease. After all, the maintenance of animal health is our objective.

(3) **Business Methods, Book-keeping, Journalism and Article-writing.**

Instruction in these methods should be included in the course for the following reasons. No matter in what field a veterinarian works, whether it be as a research worker, an educator, a private practitioner or as a field officer in the government service, he has to write up his results, keep records, publish his findings in the scientific journals or even to write books. Many a fine concept or idea never becomes printed because the author simply does not know how to write it up. Furthermore, a well-written article certainly gives a good impression of the quality of a graduate.

(4) **Public Speaking.**

This is another very important subject that should be added to the course.

(5) **Specialization.**

This should not be undertaken until the student has had a thorough and complete training in the basic subjects because we should not have anyone becoming a specialist before he is experienced. When the general training has been received there is a definite need for specialization to bring the benefits of the enormous development of veterinary science in a practical manner to the animal world. This is in the field of the specialist.

I hope that you will be able to find space in your journal for the above suggestions on a subject which I fancy should be of current interest in South Africa.

Best regards,
Sincerely,
VICTOR. R. KASCHULA,
Associate Research Specialist
in Animal Pathology.

BOOK REVIEW

ANIMAL DISEASES IN SOUTH AFRICA, by M. W. Henning, 3rd Edition, 1956. Price £5.10.0. Central News Agency Ltd., South Africa.

The third edition of this work gives an excellent and up-to-date account of the infectious diseases of animals in South Africa. Diseases not described in the previous edition such as Rift Valley fever, Nairobi Sheep Disease, Porcine Babesiosis, Eperythrozoonosis, Corridor Disease, Ovine Abortion, Virus Pneumonia of Pigs, Swine Influenza, Rubarth's Disease and Q Fever have been included.

The book is large, consisting of 1,239 pages, and has useful illustrations, diagrams and comprehensive lists of references. The author has given an excellent account of calf diseases. If future editions are not to become too large and unwieldly, much at present included will have to be left out, for instance methods of vaccine preparation and much of the bacteriology, protozoology and virology. The book is probably beyond the scope of most stock owners but an extremely useful reference book and guide to veterinary surgeons. It is very useful for teaching purposes and is likely to become a standard text book for reference purposes.

G. D. SUTTON.

COUNCIL MATTERS

THE SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

THE MINUTES OF THE MEETING OF COUNCIL HELD ON THURSDAY, 6th JUNE, 1957, IN THE MEAT BOARD BUILDING, PRETORIA.

Present: Dr. P. S. Snyman (President), Drs. R. Clark, A. C. Kirkpatrick, E. M. Robinson, S. W. J. van Rensburg, R. du Toit, M. C. Lambrechts, R. A. Alexander, H. H. Curson, L. van den Heever and S. van Heerden (Hon. Secretary).

Present by Invitation: Drs. A. M. Diesel and H. Graf.

Apologies: Drs. P. J. du Toit and G. D. Sutton.

Dr. Alexander informed Council that Professor van den Ende, who was an honorary associate members, had recently died. The President asked those

present to rise for a few moments as a token of respect. Council agreed that a letter of condolence be sent to Mrs. van den Ende and that donation be made to the Virus Research Institute of the University of Cape Town, in lieu of a wreath.

MATTERS ARISING FROM THESE MINUTES.

Discussions took place on the Faculty Posts, the Cooperative Employment of Veterinarians and the Employment of Veterinarians by S.P.C.A.'s and like organisations. In view of the fact that they were fully discussed at the Annual General Meeting in August and that a full report of these discussions will be published in the minutes, no details will be given in this issue of the journal.

Animal Protection Bill.

Dr. Clark reported that a new bill had been drafted for consideration by Parliament, after the Select Committee had considered evidence on the "Prevention of Cruelty to Animals Amendment Bill."

Dr. Clark reported on a few further amendments, and that copies of this communication had been sent to the Ministers for Health and Agriculture.

Redrafting of Constitution.

The Hon. Secretary reported that he had obtained an intimation from Mr. Wegger that the redrafting had not been completed. Agreed to ask Mr. Wegger to have the amended and redrafted constitution available for consideration at the next meeting of Council.

Annual General Meeting and Congress, 1957.

It was agreed that the Broadcasting Corporation be approached with a request for five minutes broadcasting time on the three days of the Congress, to give a résumé of the subjects discussed.

Agreed that Dr. Hofmeyr be asked to compile and deliver these résumés, and to make further arrangements with the S.A.B.C. in this connection.

Vacation Practice for Students of Faculty of Veterinary Science.

After discussion it was agreed that the Association invite applications on behalf of Faculty for honorary clinicians with whom students would do vacation practice. The names of the honorary clinicians would appear in the calendar of the University of Pretoria.

Details of the advertisement to be obtained from the Secretary of the Veterinary Faculty.

Fintance — Maud Bales Scholarship.

Messrs. A. M. Day and L. Naude to be the recipients in equal shares for the current year.

General (1).

It was proposed and unanimously agreed that Drs. J. B. Quinlan, S. W. van Rensburg and A. M. Diesel be elected Hon. Life Vice-Presidents of the Association.

THE SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

REPORT OF THE AUDITORS FOR THE FINANCIAL YEAR ENDED 31st MARCH, 1957

We have examined the books, accounts and vouchers of your Association for the year ended 31st March, 1957, and have to make the following report thereon:-

INCOME AND EXPENDITURE ACCOUNT.

The excess of Income over Expenditure for the year amounts to £297-6-4 as compared with £338-6-0 for last year. The cost of enquiries and giving evidence, accounts for the decrease in the surplus for the year.

The excess of Expenditure over Income of printing and distributing the Veterinary Journal amounted to £152-1-9 as compared with £214-0-2 for the previous year, arrived at as follows:-

COST OF PRINTING AND DISTRIBUTING THE VETERINARY JOURNAL

		1956
Printing	£822 4 10	£754
Secretarial Fees	60 0 0	60
Postages and Stationery	12 15 6	21
Audit fee	14 0 0	11
	£909 0 4	£846

DEDUCT INCOME FROM VETERINARY JOURNAL

Advertisement charges	£578 4 10			431
Less amount written off	5 19 0			—
		572 5 10		431
Subscriptions	165 13 9			191
Less amount written off	1 13 6			—
		164 0 3		191
Sale of reprints		20 12 6		10
			756 18 7	632
Excess of Expenditure over Income for the year			£152 1 9	£214

BALANCE SHEET

Investments:

Investments have increased by £224-7-6, being the reinvestment of £800 Union Loan Certificates with interest received thereon, amounting to £1,010-0-0. This amount with additional funds, was invested in United Building Society as £1,200-0-0 fully paid up shares.

BOOK FUND

A net profit of £168-18-5 was made as compared with £89-15-11 the previous year. The percentage of Gross Profit on sale of Books and Instruments increased from 8.2% to 9.15%. The Book Fund is shown as follows:-

BOOK FUND

MEDICAL BOOKS AND SURGICAL INSTRUMENTS TRADING AND PROFIT AND LOSS ACCOUNT FOR THE YEAR ENDED 31st MARCH, 1957.

1956			1957
	Stocks on hand, 31st March, 1956:		
£283	Books	£189 8 0	
2	Instruments	4 17 7	
			£194 5 7
904	Purchases		1,845 19 3
98	Gross Profit for year carried down		180 12 1
£1,287			£2,220 16 11
£11	Audit Fees		£14 0 0
90	Net Profit for the year transferred to Prize Fund Reserve Account		168 18 5
£101			£182 18 5

1956			1957
£1,093	Sale of Books and Instruments		£1,963 11 0
	Sale of Veterinary Certificates		16 7 6
			1,979 18 6
	Stock on hand on 31st March, 1957:		
—	Certificates	£92 6 4	
189	Books	147 2 10	
5	Instruments	1 9 3	
			240 18 5
£1,287			£2,220 16 11
£98	Gross Profit brought down		£180 12 1
2	Postages recovered		1 18 10
1	Bank Charges recovered		7 6
£101			£182 18 5

Loans to Members and Students.

The following statement shows the amounts outstanding at 31st March, 1957.

H. J. W. Botes	£29 6 4
A. J. Snyders	60 16 7
A. J. Snyders	60 16 7
A. J. Snyders	40 8 10
J. T. R. Robinson	7 11 6
A. M. Lubbe — Student	50 0 0
	£248 19 10

Office Equipment.

A filing cabinet costing £36-5-0 was purchased during the year. The Office Equipment at depreciated value at 31st March 1957, is as follows:-

Desk and Chair	£13 14 6
New Typewriter	18 14 0
Old Typewriter	13 6 8
Cupboard	9 9 10
Filing Cabinet	33 11 0
	£88 16 0

General Fund.

The balance of this Fund increased from £4,167-6-3 to £4,455-3-7. Subscriptions amounting to £9-9-0 have been written off in accordance with a Resolution dated 7th September, 1956.

Benevolent Fund.

The balance of this Fund increased from £2,783-17-2 to £3,033-10-9 Assistance payments amounting to £110-0-0 were made during the year, made up as follows:-

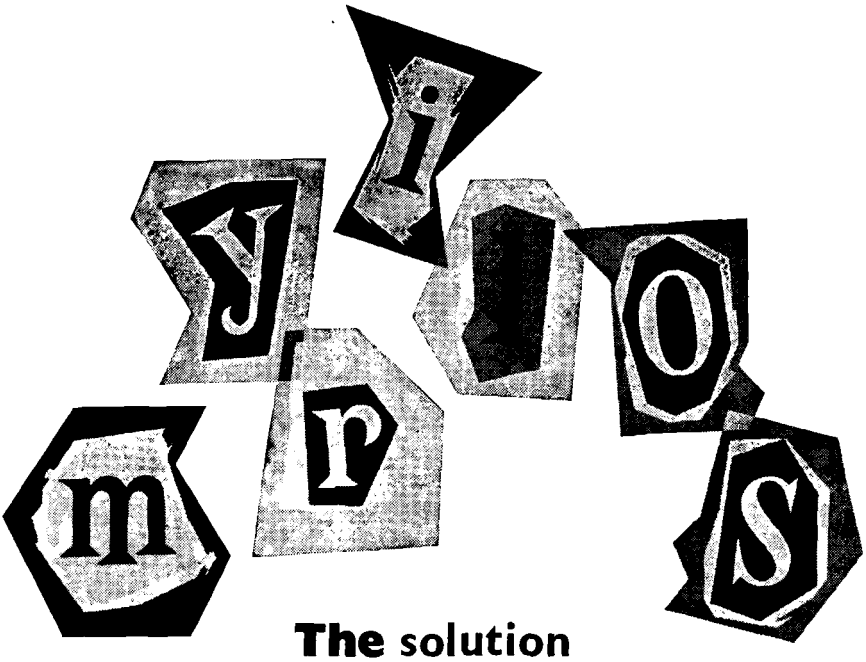
Mrs. Ziehn (Resolution dated 21st November, 1956)	£90 0 0
S.A. Red Cross re Dr. Valkayrie	20 0 0
	£110 0 0

Prize Fund Reserve.

The Fund increased by £209-1-0 to £1212-6-8 due to a profit of £168-18-5 on the sale of Books and interest accrued, amounting to £40-2-7, on the balance of the Fund.

PRETORIA: 16th JULY, 1957.

BODEL & POLLOCK
CHARTERED ACCOUNTANTS (S.A.)
AUDITORS



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THE PROFESSIONAL PROVIDENT SOCIETY OF SOUTH AFRICA

P.O. Box 6268 104, Cavendish Chambers,
Telephone 23-1560 Jeppe Street, Johannesburg.

The Society was established by members of the Dental profession some fifteen years ago and is an entirely *mutual* organisation devoted solely to the interests of its members.

MEMBERSHIP:

Membership has now been extended to include members of the Dental and Medical Associations of Southh Africa, the Pharmaceutical Society of South Africa and the Societies of Advocates of South Africa. An applicant for membership must be under 51 years of age and must submit satisfactory evidence of good health in the form of a medical certificate, the cost of which must be borne by the applicant. Applicants of 45 years of age and over are required to submit in addition, recent X-Rays of the chest and a recent E.C.G. tracing.

Although the Society has the right to refuse to admit applicants because of an unsatisfactory medical report, it will in certain instances grant membership subject to a restriction precluding benefits payable in respect of a sickness directly attributable to a specific complaint from which the applicant is suffering or has suffered at the time of admission to membership. Such restriction may be imposed for a limited period, or permamently.

SUBSCRIPTION RATES:

An entrance fee of one guinea (£1.1.0d.) is payable on acceptance as a member.

Subscriptions are assessed on an age basis according to the number of shares for which a member subscribes, the maximum being fifty (50) shares. The following are the subscription rates:

AGE NEXT BIRTHDAY AT ENTRY	MONTHLY SUBSCRIPTION RATE PER SHARE	PER 50 SHARES
30 years or under	2/9d.	£6 17 6
31 to 35 years	2/10d.	7 1 8
36 to 40 years	3/0d.	7 10 0
41 to 45 years	3/3d.	8 2 6
46 to 50 years	3/6d.	8 15 0
51 years	3/9d.	9 7 6

AGE NEXT BIRTHDAY AT ENTRY	ADDITIONAL SUBSCRIPTIONS PER SHARE FOR VETERINARY SURGEONS	
	Yearly	Monthly
Under 31	2/–	2d.
31 to 35	2/–	2d.
36 to 40	2/–	2d.
41 to 45	2/–	2d.
46 to 50	3/–	3d.
51 to 55	3/–	3d.

The monthly subscription rate at entry remains unchanged throughout membership. Increases in shareholdings are assessed at the rate applicable to the age at the date of purchase of the additional shares.

Members over the age of 51, but under 55, may increase their shareholding at the rate of 3/9d. per share per month.

Subscriptions may be paid monthly in cash or by bank stop order. A discount of 5% or 2½% respectively, is allowed on subscriptions pain annually or half-yearly in advance.

SICK PAY AND INCAPACITY BENEFITS:

After six months of membership, a member is entitled to claim for sick pay on the production of a medical certificate, provided the period for which

such claim is lodged exceeds six days. The rate of sick pay is 12/6d. per share per week. In any cycle of two years, a member may claim sick pay at this rate for six months and for a further period of six months at half this rate. The present maximum holding of 50 shares will allow sick pay at the rate of £31.5.0d. per week or approximately £135 per month. Provision is made in the Rules of the Society for an incapacity benefit in the event of permanent incapacity.

"ONCE A MEMBER, ALWAYS A MEMBER". Illnesses do not affect membership, as once accepted, a member, regardless of whether he may have become a bad sickness risk, is covered for any number of illnesses up to retirement age.

APPORTIONMENT ACCOUNTS:

Only a comparatively small proportion of the subscriptions paid is utilised for providing sick pay benefits, the balance being used to secure the provident aspect of membership. The total subscription income of the Society, after deduction of administration and sick pay costs, at the end of each financial year, is apportioned to members on a pro rate basis and placed to the credit of their respective Apportionment Accounts. *The amount credited to a member's Apportionment Account is in no way affected by any sick pay benefits received by him during the year.*

The average rate of allocation to members' Apportionment Accounts for the past five years has exceeded 2/6d. per share per month, and in 1955 was 2/8.85d. per share per month. For example, on the basis of a 2/6d. dividend rate, a member with the maximum shareholding at 2/9d. per share, pays £82.10.0d. per annum in subscriptions and receives a dividend of £75.0.0d. which means that the sick pay cover has cost him only £7.10.0d. for the year. In the case of a member with the maximum shares at 3/6d. per share, this cover is £30.0.0d. per annum (i.e. £105.0.0d. subscriptions less £75.0.0d. dividend).

Apportionment Accounts earn interest at the average rate of interest earned by the Society's investments which, for a number of years now, has been approximately 5% per annum.

PROVIDEND FUND BENEFITS:

The balances standing to the credit of members' Apportionment Accounts are payable on retirement at 60 or 63 years, or in the event of prior death or of retirement from practice. This also applies in the case of permanent departure from the Union. In the event of resignation for reasons other than given above, the balance in the Apportionment Account is paid out but subject to a small penalty.

Although it would be difficult to give any accurate figures of the ultimate value of an Apportionment Account, a rough calculation on the basis of the present figures would suggest that a member holding the full 50 shares would, after 20 years of membership, be entitled to over £2,200, whilst the figure after 30 years of membership would probably exceed £4,400. These figures are, of course, subject to the present dividend and interest rates being maintained.

TAX PROVISIONS:

Subscriptions qualify for the rebate of 1/3d. in the £ allowed for insurance premiums while sick pay benefits and all other payments made to the member are free from taxation.

MANAGEMENT:

The Management of the Society is vested in the members through a Board of Management appointed at the Annual General Meeting. This Board has at its disposal, the services of a consultant actuary, and the Society also has three trustees who are required to approve all investments which do not fall under the categories of investments required by Government regulations for such societies.

FOR APPLICATION FORMS AND FURTHER INFORMATION, WRITE TO:

The Secretary,
The Professional Provident Society of South Africa,
P.O. Box 6268,
JOHANNESBURG.

THE PROFESSIONAL PROVIDENT SOCIETY OF SOUTH AFRICA

EXTRACT FROM THE BALANCE SHEET FOR THE YEAR ENDED THE 31ST DECEMBER, 1956, SHOWING THE ASSETS OF THE SOCIETY

Investments (At cost plus accrued interest):	
Esco Lomcal Registered Stock	£18,155
Union of S.A. Local Registered Stock	33,041
City of Cape Town Local Registered Stock	3,028
Board of Management Virginia Loan	10,045
Union Loan Certificates	4,191
Savings Accounts with Building Societies	406
Subscription Shares in Building Societies	22,174
Loans secured by first ortgage bonds	95,771
Loans to members	7,556
	£194,367
Cash at Bank and on Hand	613
Sundry Assets	400
Total Assets at 31st December, 1956	£195,380
Total Assets at 28th February, 1957	£204,700

COMPARATIVE FIGURES FOR LAST FIVE YEARS

	1952	1953	1954	1955	1956
Assets at 31st December	£51,791	£70,575	£100,267	£142,993	£195,380
Dividend declaration per share per month	2/5.2d.	2/6.1d.	2/5.2d.	2/8.88d.	2/6.6d.
Average rate of interest on investments	4.75	4.909	4.92	4.88	5.39
Sick pay claims as percentage of income	11.7	10.8	13.4	4.81	9.77
Administration expenses as percentage of income	7.2	6.1	5.85	5.08	5.11
Total membership at 31st December	314	388	548	670	824
Total shareholdings at 31st December	9482	13038	20309	26642	33985
Average shareholding per member	30.2	33.6	37.1	39.8	41.2

OUTBREAKS OF SCHEDULED DISEASES IN THE UNION OF SOUTH AFRICA DURING THE PERIOD 1/6/57 to 31/8/57.

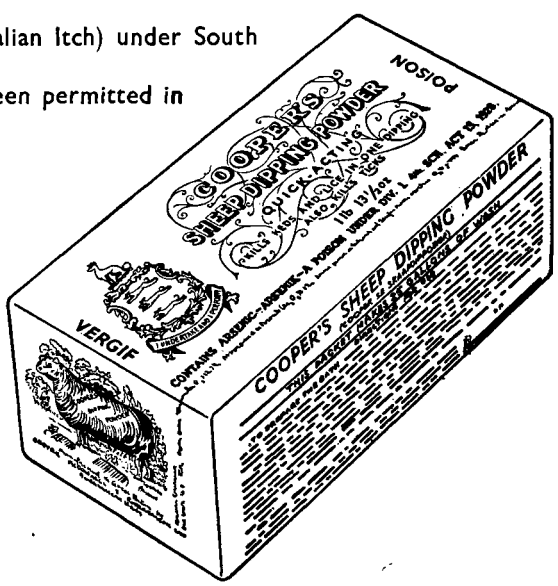
DISEASE	*CAPE*	*NATAL*	*O.F.S.*	*TRANSVAAL*
Anthrax	(4) East London (1) Worcester (1) Kingwilliams-town	(1) Nqutu	(1) Brandfort (1) Frankfort (1) Parys	—
Bacillary White Diarrhoea	(1) Graaff Reinet		(1) Bloemfontein (1) Fouriesburg	—
Foot and Mouth Disease	(31) Vryburg			(1) Heidelberg (2) Rustenburg (3) Springs
Fowl Cholera	(1) Bellville (1) Stellenbosch			
Fowl Typhoid	(1) Estcourt			
Lumpy Skin Disease	Fairly widespread over the Union. Incidence diminished during the past three months, probably seasonal.			
Mange (Equine)	(1) Kingwilliams-town (1) Kentani (1) Lusikisiki (1) Mt. Ayliff (6) Mqanduli (7) St. Marks (7) Tsolo (1) Tsomo (8) Umtata (1) Umzimkulu			
Mange (Goat)	(3) Butterworth (1) Tsomo (3) Glen Grey (6) Kingwilliams-town (4) Mqanduli (3) Peddie (3) Kentani (1) Umtata			
Mange (Porcine)		(1) Ixopo	(1) Boshof	(1) Lydenburg (1) Klerksdorp
Mange (Bovine)	(1) Warrenton			(3) Letaba
Rabies	(1) Postmasburg (3) Vryburg		(2) Bethlehem (1) Bultfontein (2) Boshof (1) Kroonstad (1) Odendaalsrus (1) Viljoenskroon (2) Bloemfontein	(1) Nelspruit (1) Pietersburg (1) Sibasa (5) Zoutpansberg
Scab (Sheep)	(4) Mafeking			
Trypanosomiasis (Dourine)	(2) Vryburg			
Tuberculosis (Bovine)	(1) Bathurst (1) Bedford (1) Fort Beaufort (1) Humansdorp (1) Jansenville (1) Port Elizabeth	(1) Durban (1) Hlabisa (1) Lower Tugela	(1) Bloemfontein (2) Bothaville (2) Fauresmith (1) Frankfort (1) Kroonstad (1) Thaba 'Nchu (1) Ventersburg (1) Wepener	(1) Belfast (1) Amersfoort (1) Potgietersrus (1) Pretoria (2) Rustenburg (1) Standerton (1) Warmbad

IMPORTANT STATEMENT

COOPER'S
SHEEP DIPPING POWDER
(QUICK-ACTING)

South African Veterinarians who are well acquainted with Cooper's Sheep Dipping Powder (Quick-Acting) will be interested to know the results of the Cooper Tests carried out at Ermelo, Transvaal. As a result of these experiments, registration of Cooper & Nephews' Sheep Dipping Powder (Quick-Acting) for the control of *Psorergates ovis* (Infectious or Australian Itch) under South African conditions has been permitted in terms of Act 36 of 1947.

Furthermore, the Authorities had previously accepted and registered C.D.P. (Q.A.) as a dip for the eradication of Keds and the control of Sheep Lice at single dippings.



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"TURNING SICKNESS"

Bovine Theileriosis in the Rustenburg District

K. C. A. SCHULZ and J. R. SCHUTTE

Onderstepoort — Rustenburg

SUMMARY

Bovine Cerebral Theileriosis (Turning sickness) as it occurs in the Rustenburg district has been described. It is not confined to that area and has probably a wider distribution than has hitherto been accepted. The condition is identical with that recorded by Flanagan and le Roux (1956) and very similar to "turning sickness" occurring in tropical East Africa. Contrary to the East African experience, the disease is confined to areas free from East Coast fever since the beginning of 1920. Its occurrence is usually sporadic, but sometimes epizootic, causing considerable economic losses. There is circumstantial evidence that it may become enzootic on certain farms. The disease is associated with the heavy tick infestations seen in those areas. Calves between the ages of two to six months are more prone to become affected than maturing and adult animals. The symptomatology and pathological anatomical changes have been dealt with in some detail. Its differentiation from East Coast Fever has been briefly considered. Cattle recovered from heartwater were not immune to this condition, (Turning Sickness). Treatment hitherto has been of no avail. The nature of this disease warrants further investigation.

Introduction and Historical Survey:- Neitz et al 1. (1955), in discussing "Corridor disease" of cattle pointed out that besides East Coast Fever and *Tzaneen disease*, a third form of Theileriosis has been encountered in South Africa. Subsequently, however, another type, namely *Bovine Cerebral Theileriosis* (*Turning Sickness"*) could be added to this list by Flanagan and le Roux. In a communication they briefly describe the symptomatology and pathology of two sporadic cases of this disease, one occurring in a three year old cow on *"Haakdoringboom,"* a farm in the vicinity of Onderstepoort (May 1955) and the other in a two year old bull on *"Nooitgedacht,"* a farm near Northam in the Rustenburg district (December 1955). It is of interest that the former animal had been purchased as a weaner on a property near Northam, the name of which could, however, not be ascertained.

The authors, on comparing their findings with those they observed in brain material of a typical case of "turning sickness" from Uganda, concluded that the condition in South Africa was very similar if not identical with that encountered in tropical East Africa.

It is rather astonishing that, in spite of careful investigations dating from the beginning of 1935 until the early part of May 1955, this disease apparently evaded diagnosis or remained unobserved. Nothing of this nature was observed locally, in the central nervous systems of a number of East Coast fever and *T. mutans* cases examined especially for this purpose. In fact the only abnormality recorded is the occurrence of *P. bigeminum* in a large number of erythrocytes in the small blood vessels of the hippocampus of one case (3).

Incidently "turning sickness," also known by its native names "Muthioko" and "Kazengerenga" in Kenya and Uganda respectively, attracted the attention of the field staff in the latter territory as far back as 1924. Its aetiology remained obscure however, until 1930, when Mettam succeeded in demonstrating structures similar to Koch's bodies in the brain of a typical case. His observations were first recorded in 1934 (4) and a more detailed report on this condition was published in 1936 (5). It is known to occur sporadically throughout Uganda, the greater part of Kenya and presumably in Tanganyika where East Coast fever is highly enzootic.[4,5,6,7,8]. Notworthy is that *"Turning Sickness"* has been seen in cattle which had recently recovered from or in those that had passed through an attack of East Coast fever many years before, i.e. in animals immune to this disease.

Contrary to the East African experience all our cases have occurred, hitherto, only in cattle confined to areas free from East Coast fever since the early part of 1920. The possibility of *turning sickness* being associated with *Theileria parva* (Theiler 1904) infection in this country is, therefore, very remote.

Whereas the workers in Uganda are still in doubt whether the parasite is identical with that of East Coast fever or is a closely related species, those in Kenya were at one time inclined to believe that the causal organisms of *turning sickness* and East Coast fever were identical. They had, presumably, transmitted the latter disease by ticks fed on a animal suffering from the former.[6] Subsequently, however, they did not succeed in confirming their previous experience.[7] All attempts by these workers to reproduce this condition experimentally have so far failed.

Sometimes however the brain lesions of cases with typical symptoms of *turning sickness* may show large numbers of *Trypanosoma theileri* in the blood but no Koch's bodies.[9]

Thus the circling movements in "turning sickness" are not pathognomonic of a Theileria infection. One cannot lose sight of the fact that these protozoa may merely be secondary invaders in a brain already infected by an agent such as a virus, the nature

of which is at present obscure, as previously suggested.[9] On the other hand it is well known that identical histological lesions may be produced by various aetiological factors. The similar syndrome observed in the theilerial and trypanosome infections may reasonably be explained thus without incriminating a virus.

The name "turning sickness" is rather unfortunately chosen, since there are a number of other nervous affections such as severe cases of internal hydrocephalus, cerebral tuberculosis, otitis media purulenta with involvement of the brain tissue, heartwater and others, which may be confused with that condition. It is suggested, therefore, to substitute for the term "turning sickness," *bovine cerebral theileriosis*," coined by Flanagan and le Roux,[2] in all cases harbouring the Theileria species in the brain. By adopting this procedure, much of the existing confusion could be eliminated.

Bovine Cerebral Theileriosis (Turning Sickness) may be defined as a fatal, peculiar nervous condition in cattle characterized clinically by circling or turning movements and an apparently afebrile course. It is associated with a protozoan encephalitis, the predominant features of which being haemorrhages of variable magnitude and the plugging or clogging of blood vessels of the central nervous system and its meninges by lymphocytes containing organisms indistinguishable from the schizonts of *T.parva* (Koch's bodies). In contradistiction to East Coast fever, it is most unusual to find these bodies in blood, lymph gland, bone marrow or spleen smears of such cases. Usually the condition occurs sporadically, but occasionally, however, as an epizootic. In the latter case the losses sustained may become of marked economic importance. Depending on the nature of the symptoms observed, the disease is locally known as "draaisiekte" or "turning sickness" or "dronksiekte."

Own Observation:

History:- As stated above, the first authentic case of *bovine cerebral theileriosis* in the Rustenburg district affected a two year old bull on the farm Nooitgedacht on the 9th December, 1955. A disease, locally known as *"draaisiekte"* or *"dronksiekte"* with symptoms suggestive of *"turning sickness"* was seen in a four months old calf on the farm Varkfontein about one month later. Blood, spleen and lymphgland smears of this animal, forwarded to Onderstepoort proved to be negative on microscopical examination. A tentative diagnosis of a *Coenurus cerebralis* infection was suggested. During February of this year, however, another outbreak was reported on the same farm, the disease appearing in an epizootic form and mainly calves being affected. Schutte, who investigated the disease, was inclined to associate it with *bovine cerebral theileriosis* or "turning sickness," his suspicion being subsequently confirmed by histological examination. Genuine lesions of this condition were observed in the brain material of several

calves, which had shown typical symptoms of "draaisiekte" prior to their death. The owner, being very much perturbed by its high mortality rate, brought two diseased calves to Onderstepoort for diagnostic purposes. "Draaisiekte" was investigated *in loco* during the first week of March last.

The farm Varkfontein is divided into two main portions, which may be called, for convenience, A and B respectively. It would appear that the disease is confined to the farm as a whole and presumably does not affect cattle on the incontact farms. On portion B on which the disease was investigated, the various types of cattle, i.e. cows not in calf and heifers, those pregnant, and tollies are kept in separate camps and drink at different watering places. The cows about to calve down and those in milk are kept in the homestead camp. Animals may become affected in all camps.

Apparently, the first case to contract this condition was a bull kept on portion A some years ago. The disease occurred next, during December 1954, in a four months old calf on portion B. Since then it has become of considerable economic importance to these farmers as is reflected by the losses incurred. On the latter portion from the beginning of November 1955, to the early part of January the following year, two oxen, four cows and one heifer had succumbed to a similar affection. The farmer suspecting heartwater inoculated all his cattle, including 77 calves, against this disease on the 14th September, 1956. (Incidentally, the whole calf crop for portion B had also been inoculated during 1956 for the same reason). Contrary to expectations the losses sustained during the period elapsing from the date of inoculation to the end of February 1957, instead of being reduced, actually increased very appreciably. The total mortality on portion B ascribed to this condition amounted to two cows, seven heifers, two to three years of age, a two to four year old ox, and 30 calves, their ages ranging from three to five months. From the 9th February until the end of June, 1957, there was an obvious reduction in the incidence of the disease, six deaths and four recoveries being recorded.

On the property A, the total losses during the book year 1955 to 1956 amounted to 30 head of cattle, the age of the majority of them ranging from four to five months and that of a few was two years. In the following year 42 animals were lost and in most the age varied from two to three months, *"Draaisiekte"* being mainly incriminated for this high mortality. It may be mentioned that towards the end of June a heifer contracted the disease on each property. These cases were severely affected about six days after the first symptoms appeared. Treatment proved of no avail. Calves recovered from heartwater are not immune to *draaisiekte*. The considerable concern *bovine cerebral theileriosis* caused the farmers and their reluctance in carrying out the heartwater inoculations during the following season, may readily be appreciated on taking the above data into account.

Subsequently, towards the end of March 1957, sporadic cases occurred on the farms *Nooitgedacht* and *Kromdraai*. These farms do not adjoin each other but are fairly widely separated. The condition is not confined to the Rustenburg district alone and may be much more widely distributed than one is inclined to believe at present. As corroborative evidence for this assumption, another sporadic case recorded during June of this year in a 15 month old heifer, bred and reared on the farm *Vlakplaats* in the Krugersdorp district may be mentioned.

Sex, age and breed are apparently not inhibitory factors although, since September 1956, calves between the ages of two to six months seem to be more prone to contract the disease than maturing and adult bovines.

Incidence:- A seasonal incidence may be assumed, since most cases are recorded from the beginning of November to the end of February of the following year. Thereafter, the number of affected animals is appreciably reduced until the end of June, when an occasional case may be encountered. *Draaisiekte* usually occurs sporadically but sometimes it may take the form of an epizootic causing losses of appreciable economic importance.

Mortality:- The mortality rate is very high; the disease being usually fatal.

Course:- The course may be acute, death occurring within a few days of the animal starting to show symptoms, subacute, the disease lasting for about two weeks or chronic, death occurring after several weeks or months. Sometimes however an animal appears to recover from the original nervous symptoms but gradually loses condition, being no asset to its owner. Chronic cases have been known to live for over a year. The majority of these are afflicted with impaired vision or total blindness.

Aetiology:- The nature of the disease is still obscure. Whether the parasite found in the brain is identical with that of East Coast fever, or is a closely related or an independent species or its presence merely accidental, we are not in a position to state and it does not fall within the scope of this paper. Smears of the blood and of various organs have been examined. A variable number of Koch's bodies were readily detectable in brain smears of all cases, in those of the anterior and posterior chambers of the eyes and those of the cerebral spinal fluid of some, and exceptionally and rarely in smears of lymph glands and of blood vessels entering a parotid lymph gland (one case); none, however, was observed in spleen and bone marrow smears. No parasites, except an occasional *Piroplasma bigeminum* or *Anaplasma marginale*, were detected in the erythrocytes of blood and organ smears. It is rather astonishing to find such grave lesions and large numbers of schizonts in the brain with the erythrocytic parasites rarely present[2] or completely absent. In this respect this disease markedly

differs from East Coast fever and other theilerioses. The brain in the last mentioned diseases may be only slightly affected and schizonts are rarely found, if at all, in smears or sections of this organ. Morphologically these schizonts are, however, indistinguishable from those seen in spleen smears of East Coast fever.[10]

Symptoms:- The condition is characterised by symptoms indicating the involvement of the central nervous system. The variable symptoms seen in the affected animals are probably dependent on the site, the size and the number of the lesions.

Manifestations of the nervous symptoms are circling or turning movements, either to the left or to the right, a swaying, uncertain, unnatural gait, stumbling, giddiness, ataxia, paresis or paralysis; *strabismus sursum vergens* (vertical squint) the visual axis of one eye being deviated upwards, and sometimes loss of consciousness. The neck is held rigidly, the head being twisted slightly upwards towards the side the animal is circling. The mydriases and strabismus may be associated with paralysis of the oculomotor nerve resulting from the encephalo-meningitis. The twisting of the head and the circling movements are probably compensatory, the animal attempting to accommodate its vision by this means. Often it is unable to attain its object, missing the object it tries to reach, for instance a calf may continuously walk past its mother in an attempt to suckle. There are signs of progressive weakness, the affected animal being unable to keep on its feet, lying down more frequently than usual and eventually remaining in a recumbent position, struggling in attempting to rise. Opisthotonos and galloping movements are occasionally observed in such cases. Sometimes an animal develops rabid symptoms, wildly rushing into stationary objects such as trees, thereby damaging or even breaking its horns.

Slight exophthalmus, dilated pupils (mydriasis), impaired vision and apparent insensitivity of the cornea are indicative of other eye affections. The pupils still reacts to light, yet its response is retarded to some extent. The retinal blood vessels may be markedly engorged and signs of petechial haemorrhages in evidence. On standing some distance from the animal the bright retina, seen through the dilated pupil, is very striking, its appearance being reminiscent of eyes reflecting light in the dark. Some individuals with disturbed vision seem to be more alert to sounds as evidenced by turning of the head, flickering and pricking the ears and a strained expression appears in the eyes (fig. 1). Others are apathetic, being listless, dull and indifferent to or not aware of any noise, standing with partially or totally closed eyes and drooping ears, not moving off on being approached (fig. 2).

The attacks of unconsciousness are of a transient nature and may last a variable length of time. Shortly after recovering consciousness, the animal attempts to rise and with help is able to get on its feet, moving forward and remaining on its feet for an appreciable time before lying down again.

Fig. 1
Calf with extended neck, twisted head, pricked ears and mydriasis. Note reflection of light from both retinae. Animal circled towards right side.

Fig. 2
Calf apathetic, dull, standing with extended neck, drooping ears and closed eyes.

Noteworthy is the apparent afebrile course of the condition, temperatures up to 101°F have been recorded in uncomplicated cases. With concurrent diseases such as pneumonia, redwater or paratyphoid, there is a distinct rise of temperature and occasionally just prior to death it may be subnormal.

Salivation to a variable extent may occur. Frothing at the mouth or excessive dribbling, presumably four to five gallons of saliva being secreted during the course of the disease, have been observed. There may be an apparent interference with the apprehension of food and the drinking of water. Some animals although nibbling at hay, appear to be continuously hungry. Others having access to water do not drink, the water, however, being taken with apparent ease on dosing.

In addition anorexia, obstipation, signs of dehydration, sunken eyes and a variable degree of dyspnoea may be evident.

Autopsy:- The pathological anatomical changes in the different animals may vary quite considerably, according to their site, size, nature and the course of the disease. They are mainly confined to the brain, the spinal cord and the meninges, but may be observed to a lesser extent in some lymph glands (pharyngeal), the hypophysis, the retina, the iris, around the optic nerve, the thymus, the myocardium and the folds of the abomasum, the mucosa of which is ulcerated. The lesions are manifested, in the acute cases, by congestion, thrombosis and haemorrhages of variable magnitudes, some of the larger circumscribed ones protruding over the surface of the adjacent tissue. Extravasations differing in degree may involve the cerebral hemispheres, (fig. 3) hte cerebellum, the caudate nucleus, the midbrain, the pons, the medulla, the spinal cord and the meninges. These structures need not all be affected in one animal at the same time, for instance in one case the most pronounced haemorrhages may occur in the cerebrum without any appreciable change in the cerebellum, whereas the latter or the midbrain is chiefly involved in another. In the vicinity of the larger extravasations the surface of the brain is reddish to brownish red in colour. Blood clots, their sizes varying from 1 to 3 cm., may be formed either at the periphery or in the ventricles. On occasions clots are found in both localities.

In the subacute and chronic cases the meninges are obviously thickened, having a distinct opaque, greyish appearance. The colour of the cortical surface and spinal cord may be a dirty yellow, especially in circumscribed areas of collequative necrosis. The most striking change in the subacute cases is the pronounced thrombosis of the smaller and larger blood vessels of the central nervous system and its meninges. The superficial blood vessels stand out clearly as rigid, tortuous, opaque cords in the sulci of the cerebral hemispheres (fig. 4) or cerebellum. They have a mottled appearance, the colour varying from dark red to grey. The difference in the tint depends probably on whether the erythrocytic or leucocytic cell content predominates. Blood vessels of

the grey and white substance may also be affected and haemorrhages differing in degree are present. The brain tissue is oedematously infiltrated and its consistence is considerably reduced. It can easily be crushed like soft butter by squeezing with the fingers.

There is a distinct increase in the cerebro-spinal fluid and the intracranial pressure may be considerably raised thereby. The amount, however, varies to some extent in the different animals. In some cases the intenser yellowish, tinted fluid is only slightly turbid, owing to an increased cell content. The fingers become very sticky shortly after handling the fluid. This phenomenon may, possibly, be ascribed to a higher globulin and total protein content as demonstrated for *turning sickness* on a previous occasion.[11]

In the chronic cases, however, the cerebral convolutions are less distinct and the brain substance markedly firmer than usual, appreciable difficulty being experienced in obtaining a brain smear.[2] The lesions are also not so noticeable.

Amongst the less significant morbid changes, loss of condition, retraction of the eyes into their sockets, signs of anaemia (in one case, an erythrocyte count of 3,940,000 per c.m.m. and that of leucocytes 8,700 per c.m.m., Haemoglobin 8.55 and red volume 24% being recorded) pale skeletal musculature, slight lymphadenitis (considerably less pronounced than in East Coast fever cases), several infarcts in kidneys (one case), slight tumor splenis and hepatis, signs of icterus occasionally (complication), focal necropurulent pneumonia (few cases, complication), slight hydrothorax, extensive subendocardial and myocardial haemorrhages (few cases), extravasations in the base of the pulmonary artery (one case), erosions in abomasal mucosa and hyperaemia of intestinal mucosa (some cases) may be mentioned. Although the calves seen during the investigation were only slightly infested with ticks, Schutte was inclined to associate the encephalitis with the severe "eartick" infestation previously observed in diseased cattle. This "eartick," according to information gained, is becoming a severe menace in that area and is actually the larval form of *R. appendiculatus*.

Histopathology:- The histopathology is characterised by a lymphocytic leptomeningitis, involving the membranes of the central nervous system, with extensive perivascular haemorrhages and thrombosis of blood vessels, a non-purulent perivascular encephalitis with focal areas of demyelination and glial proliferation. The majority of lymphocytes contain schizonts similar to those of *T. parva*. (East Coast fever).

Diagnosis:- In order to establish a diagnosis of *bovine cerebral theileriosis*, smears should be made from the opaque plugged or clogged blood vessels with a greyish tinge, since on histological examination the lumens of these vessels contain the largest number of affected lymphocytic cells. Smears taken from the haemorrhagic

areas are considerably less satisfactory. This may be expected since very few lymphocytes are found in these areas. Such smears should show the presence of schizonts closely resembling or identical with those of *Theileria parva*. In this condition it is most unusual to find these bodies in lymph gland, bone marrow or spleen smears. The blood may show the presence of rare small

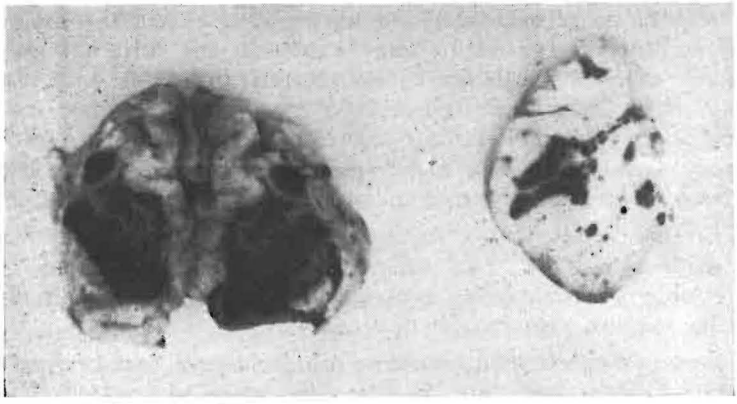

Fig. 3.
Extensive haemorrhages of variable degree involving different portions of cerebral hemispheres.

Fig. 4.
The cordlike appearance of the superficial blood vessels crossing the convolutions and passing along the sulci of the cerebrum is clearly seen.

piroplasms[2] the significance of which cannot be gauged at present. The possibility that the erythrocytic parasite may be associated with a *T. mutans* infection must, however, be considered.

Treatment:- All attempts to treat this disease have failed so far. Thus, no method of treating *bovine cerebral theileriosis* can be advised. Prophylactic measures such as the proper cleansing of

stock from tick infestations are strongly recommended since this disease may possibly be transmitted by one or other type of tick found on the cattle.

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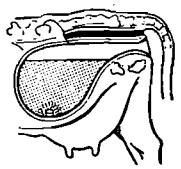

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HEMIPLEGIA LARYNGIS — RECURRENT NERVE PARALYSIS — COMPLICATED WITH PARALYSIS OF THE SOFT PALATE — PALATUM MOLLE — AS A CAUSE OF DYSPNOEA IN TWO RACE HORSES.

J. QUINLAN.

Durban.

Attention has been drawn previously to paresis or paralysis of the soft palate (palatum molle) as a cause of dyspnoea in race horses (Quinlan, van Rensburg and Starke 1949; Quinlan and Morton, 1957). Recently two further cases have been met with, which would appear to throw light on the suggestion put forward in the above-mentioned articles. The authors believe that the pharyngeal branch of the vagus (nervus pharyngeus) is occasionally involved in a similar degeneration to that of the recurrent branch (nervus recurrens). This results from the same aetiology, namely, auto-intoxication, usually following acute febrile conditions.

In those cases described previously, the involvement of the pharyngeal branch would appear to have developed independently, since the movement of the vocal cords was not impaired. In the two cases, now being described, however, both the pharyngeal branch and the recurrent branch would appear to be concurrently involved.

The horses which come under my care are of considerable value, consequently it is not possible to perform autopsies. My diagnosis is made from the nature of the noise produced by forced exercise, and local examination of the pharynx and larynx with the rhinolaryngoscope or through a laryngotomy incision. The results of the operations have also confirmed the diagnosis. It was only in the two cases, now being described, that the two operations of chordoventriculectomy and palatectomy were performed at the same time.

Since the symptoms, possibly aetiology and the operation for shortening the soft palate have already been discussed by the above-mentioned writers it is unnecessary to repeat them. It is my intention only to describe the variation in the two most recent cases.

Case 1: Bay thoroughbred colt, 3 years old. He had run three times as a two-year-old and had been placed twice. As a three-year-old he won twice and was placed twice in eight starts. He was rested on a farm during January, February and March 1957. While resting he had an acute attack of babesiosis, which was undoubtedly the cause of the nervous lesions that resulted

in degeneration of the recurrent and pharyngeal branches of the vagus nerve.

When the horse was put back to slow work in April 1957, his trainer noticed that he was making a "noise." He stated that his distress was rapidly progressive. This was confirmed by his veterinarian.

I examined the horse on 17.5.57 at the request of my colleague, Dr. K. Dalzell. I use an oval paddock, 100 yards by 50 yards, with deep sea-sand, to exercise horses for their wind. I trotted the horse once around the paddock, and then let him canter slowly with a loose rein. He had gone about 150 yards when I had to stop him as he was showing signs of suffocation. His head was held low; there was distressing coughing and marked inspiratory and expiratory dyspnoea. The distressing symptoms disappeared soon after the horse rested.

No obstruction was found in the pharynx or nasal passages when he was examined later in his stall. The distress was so acute that I suspected a polypus.

I repeated the examination on 30.5.57 with the same result.

I operated on the horse on 24.6.57 with the assistance of Dr. K. Dalzell. On opening the larynx I found the left vocal cord paralysed. Examination of the pharynx was negative.

However, the soft palate was much thinner than normal. It was pale yellowish-white in colour. Its surface was somewhat corrugated. It appeared as if the musculature had atrophied. Consequently it was decided to shorten it. The atrophied appearance was more marked on the left side. Chordo-ventriculectomy was performed at the same time.

Unfortunately I have never had a cytological examination made of the tissue removed from any of these cases on which I have operated. Such an examination would show muscular atrophy and definitely prove my contention. I trust I will be able to fill in this omission in the future.

I allow horses complete rest for two months after operations on the larynx and pharynx, before submitting them to forced exercise. During this period they are ridden at a walk only.

During his convalescence the horse had a cough which gradually improved.

I examined the horse on the 10.9.57 and found him sound of his wind.

I am quite satisfied that the cause of this horse's condition was degeneration of the recurrent laryngeal and pharyngeal branches of the vagus nerve as a sequel to biliary fever — babesiosis.

Case 2: Bay thoroughbred gelding, four years old: He had run three times as a two-year-old and had been placed once; eleven times as a three-year-old for one win with two places, and nine times as a four-year-old for one win. His last eight runs were disappointing and the owner suspected that he had gone

wrong of his wind. The horse had always been under observation and was not known to have been sick.

I examined him on 22.5.57 and found he had inspiratory dyspnoea, not marked, and an expiratory dyspnoea, with a loud snoring noise.

I operated on the horse on 3.6.57, with the assistance of my colleague, Dr. A. R. Thiel. On opening the larynx both vocal cords were static. Before proceeding further, anaesthesia not being sufficiently deep, the horse struggled and the right vocal cord became tense occasionally, not with every inspiration. The left cord remained static. When under deep anaesthesia there was no movement in the right vocal cord.

From the paralysed condition of the left vocal cord one would have expected a more marked inspiratory dyspnoea.

The expiratory, snoring sound, which I have associated with paralysis of the soft palate decided me to shorten the palate. In this case again the palate appeared thin and flaccid.

I am convinced that this case was similar to case 1, just described, but not quite so marked. Unfortunately, again I have no cytological proof of muscular degeneration of the soft palate. After two months rest I examined the horse on 6.8.57, and found him sound.

In his first race following the operation on 7.9.57, the horse ran prominently although, obviously, he could not be quite himself, being only a month in training.

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REFRESHER COURSES IN PHYSIOLOGY. III. THE MICROBIOLOGY OF THE RUMEN

F. M. C. GILCHRIST and R. CLARK.

INTRODUCTION

In carnivores the products of digestion assimilated by the animal are almost entirely simple cleavage products derived from hydrolysis of the food constituents. Thus carbohydrates are absorbed as monosaccharides and proteins as the amino-acids which formed the dietary protein. In ruminants the position is entirely different due to the action of the ruminal flora. Carbohydrates are largely fermented to volatile fatty acids of which acetic, propionic and butyric form 85% of the total. Of the more soluble carbohydrates at least, very little escapes ruminal fermentation to be digested to monosaccharides by the host. Proteins are broken down to peptides, amino-acids and ammonia, from which new microbial protein is resynthesised and later becomes available to the host. The protein ultimately digested by the host may, therefore, differ considerably from that of the diet. In addition, large amounts of vitamins of the B complex are synthesised. The extent and nature of these changes brought about in the food depend on the activity and composition of the ruminal flora. A knowledge of the microbiology of the rumen is therefore essential before the peculiar nutritional requirements, digestion and metabolism of ruminant animals can be appreciated. The object of this paper is to give a general summary of our present knowledge in this field.

The microbial population of the rumen can be divided into:-

(a) Functional types living in complete symbiosis with the host.
(b) Potentially harmful types usually present in insignificant numbers but capable of multiplication under abnormal conditions.
(c) Accidently introduced bacteria which either cannot survive in the rumen or which play no functional role.

It is a matter of controversy whether the functional symbiotic micro-organisms are obligatory ruminal organisms and must be introduced from other ruminants by contact, or whether they can develop from random infection from food. In the case of the infusoria, there is little doubt that the former is the case but this group will not be dealt with in the present paper as their contribution to the general function of the fore-stomachs has not yet been assessed.

The composition of the ruminal flora at any given time represents a dynamic balance reached through fierce competition and is dependent on the conditions pertaining in the rumen. The main factors contributing to the effective intra-ruminal environment will therefore now be considered.

THE INTRA RUMINAL ENVIRONMENT

The intra-ruminal environment is kept remarkably constant under normal conditions, mainly through the physiological activities of the host animal. The following factors must be considered.

(a) *Water Content.* The ruminal ingesta must at all times be kept semi-fluid in consistency. This is not only essential for microbial activity but also for the mechanical functions of the fore-stomachs, including mixing of the ingesta, rumination, absorption through the ruminal wall, and the passage of ingesta down the tract. The ruminal fluid is derived from the drinking water and water present in the food and the saliva. The ruminant is peculiar in that there is a practically constant and very copious flow of saliva amounting to 50 to 60 litres per day in the ox and 3 to 4 litres in the sheep (Dukes) as compared to 0.5 to 1.5 litres in man (Houssey).

(b) *Reaction.* The pH of the rumen is maintained between 6.5 and 6.8 under normal conditions. The regulating factors are:-

i. The saliva of ruminants is alkaline (pH 8.3, Mac Dougall) due to the presence of large amounts of bi-carbonate. Acid and carbon-dioxide are produced by the ruminal flora. The salivary bicarbonate and the carbon dioxide form a buffer system as the tension of the latter is kept more or less constant by regurgitation and absorption into the blood stream.

ii. The buffering of the acid and basic soluble phosphates mainly derived from the saliva.

iii. The amphoteric action of the proteins present.

iv. Selective absorption of volatile fatty acids by the ruminal mucosa.

Variations in pH outside the normal range markedly affect both the activity and composition of the ruminal flora.

(c) *Redox Potential.* Although the animal takes in small amounts of air during feeding and rumination, the oxygen introduced is rapidly removed by the fermentation activity of the ruminal flora. For instance, the ruminal gasses contain 5 vols per cent oxygen immediately after feeding and none four hours later (Brody). The ingesta itself contains sufficient reducing sustances to maintain anaerobic conditions at all times. This is shown by the fact that methylene blue is reduced within a few seconds when added to freshly withdrawn ingesta. The functional bacterial processes taking place in the rumen are therefore all anaerobic.

(d) *Mineral Salts.* Ions of Na, K, Ca, Mg, SO_4, Cl and PO_4 are essential for all bacterial activity. In addition trace amounts of Co, Cu and Mn are required. These are mainly supplied in the food. Ruminant saliva contains a high concentration of soluble phosphates which may reach twenty times that of the inorganic phosphate of the blood. This active secretion supplies the ruminal bacteria with much of the available phosphate required for growth and activity.

(e) *Vitamins.* Many species of ruminal bacteria form vitamins, especially those of the B complex, and so supply not only their own requirements but also those of other bacteria and the host animal. Ruminants are therefore independent of these vitamins in the diet provided the ruminal flora is normal and active. Although vitamins A, D and E are essential to the animal, there is no evidence that they are required by the ruminal bacteria.

(f) *Carbohydrates and Proteins.* The absolute and relative amounts, as well as the nature of the carbohydrates and proteins in the diet affect the ruminal flora as they supply the main nutritional substrate.

METABOLIC GROUPS OF FUNCTIONAL BACTERIA

The functional bacteria can be divided into metabolic groups according to the substrate they attack. In many instances the products of initial attack by one group form the substrate for other groups. The final end-products are therefore the result of the combined action of several separate groups of bacteria.

Cellulose Digesters. There are several known species of cellulose digesters which have been shown to occur in the rumen in significant numbers. All these can be cultivated on cellobiose and it is therefore assumed that cellulose fermentation passes through cellobiose. From here different pathways have been demonstrated in varying degrees of detail. For instance, *Ruminococcus flavofaciens* and *Bacteroides succinogenes* both produce succinic acid. This in turn is rapidly converted to propionic acid and carbon dioxide by *Veillonella gazogenes.* Other species are known to produce acetic, propionic and butyric acids while some produce butyric acid only. The main end-products of cellulose fermentation are therefore acetic, propionic and butyric acids which are absorbed through the ruminal wall and enter the metabolic pool of the host animal. In hay fed animals, cellulose fermentation would appear to be the main source of propionic acid production which in turn is the chief precursor of the blood glucose in ruminants.

The fore-stomachs are so constructed as to allow of the passage of only finely divided particles down the tract. The rate of cellulose digestion is therefore a major factor in determining the period over which fibrous foodstuffs are retained in the rumen and hence the amount that can be ingested over a given time. The *speed* of cellulose digestion is frequently of more importance than the

percentage of cellulose digested as it governs the daily intake and hence the total amount of nutriment made available. Figure 1 clearly illustrates the close correlation between the rate of cellulose digestion and the intake of hay.

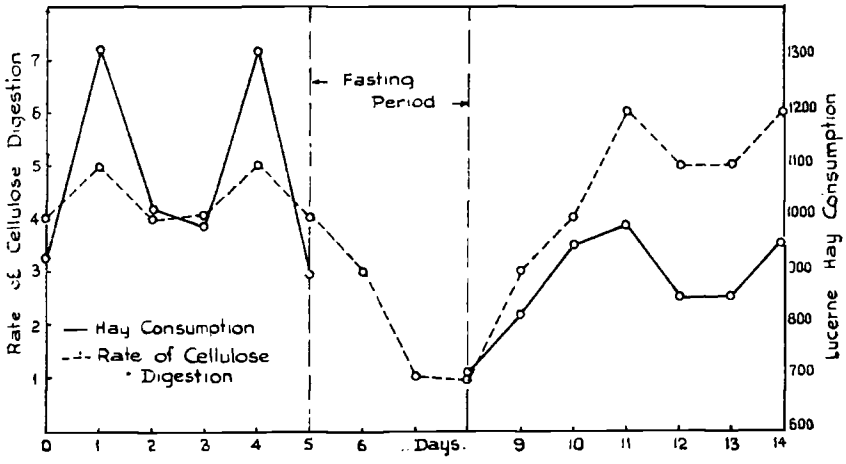

Fig. 1.

The effect of fasting on the rate of cellulose digestion and food consumption in sheep on a ration of lucerne hay.
The rate of cellulose digestion is plotted to an arbitrary scale based on the time taken for cotton threads to become dissolved. (e.g. O = no change in 120 hours, 7 dissolved in 54 hours and 14 dissolved in 24 hours). Note the gradual resumption of feeding after fasting and close correlation between intake and rate of cellulose digestion.
(Reproduced by courtesy of the D.V.S., Onderstepoort).

A further corollary to this fact is that fine milling of hay allows much of it to pass unaltered down the digestive tract and hence reduces its percentage utilization.

Excess protein, especially in the absence of adequate readily available carbohydrate, inhibits cellulose digestion. Figure 2 shows the effect of high protein on cellulose digestion and hay intake.

Gas, mainly carbon-dioxide and methane, is a by-product of all cellulose fermentation pathways and represents a loss of potential nutrients to the host. Experiments at Onderstepoort (unpublished) indicate that excess protein in the diet increases the amount of gas formed from cellulose at the expense of the volatile fatty acids. Such a gaseous and wasteful fermentation of cellulose on a high protein diet might be of importance, not only in regard to the nutritional value of the diet, but also possibly to the pathogenesis of bloat.

Differences in the end-products of cellulose fermentation under different conditions are probably due to the development of specific types of cellulose digesters. Recent work at Onderstepoort has shown that cellulose digesters isolated from sheep on a diet of teff hay are culturally different from those obtained from animals on succulent green feed and a high protein concentrate.

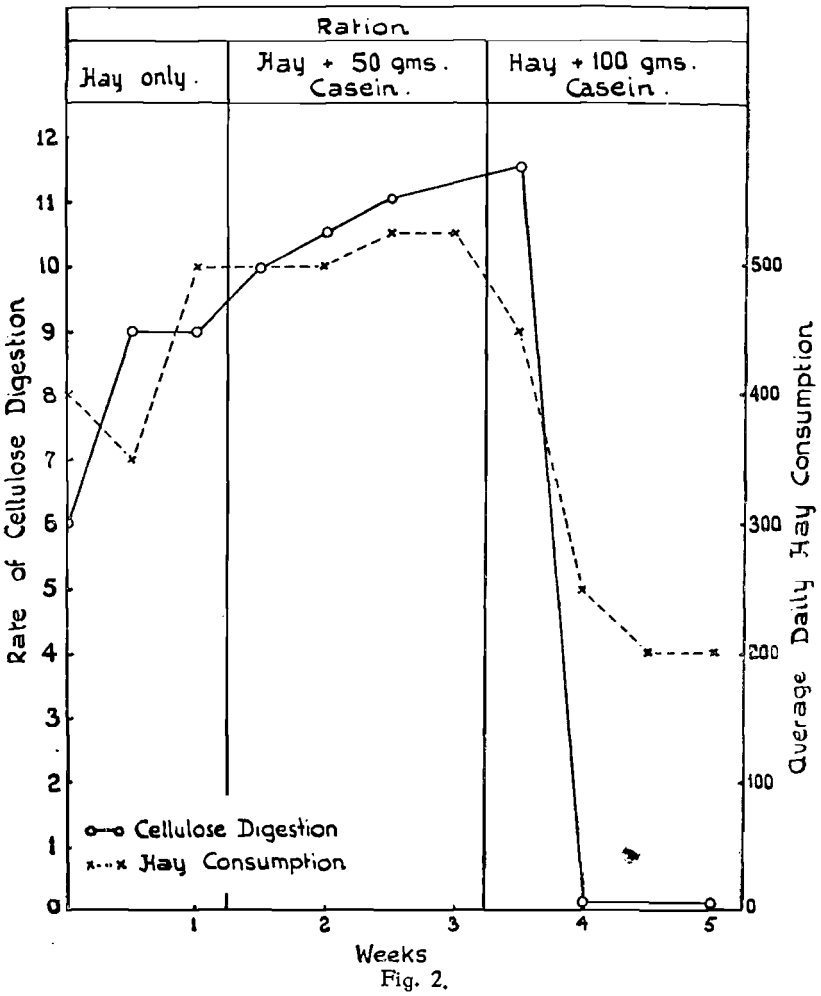

Fig. 2.

The effect of excess protein on cellulose digestion and hay consumption. Sheep fed grass hay (3% protein) *ad lib* and dosed casein daily. Note the maintenance of cellulose digestion and hay consumption with 50 gm. casein per day (total ration containing 9% protein) and the sudden drop in both factors when the casein was doubled (18% protein).

Another indication that these are two different species is that each grows well on a cellulose containing meduim enriched with ruminal fluid from the sheep from which they were isolated, but the organism from the sheep on a high protein diet will not grow on a medium containing ruminal fluid from the hay fed sheep. The 'rich diet' type is therefore more exacting in its nutritional requirements probably in regard to preformed amino-acids. Another point of difference is that the 'hay-fed' type occurred in very large numbers (approximately 10^7/ml) whereas the 'rich feed' type only numbered about 10^3/ml. This low count is offset by the facts that the cellulolytic activity of the 'rich-feed' type is greater than that of the

'hay-fed' type and the fibre in succulent feeds is easily digestible. As will be seen, propionic acid is also derived from starch and other readily available carbohydrates.

Excess protein is therefore detrimental to both the rate and efficiency of cellulose digestion. On the other hand, too low a protein ration cannot sustain the cellulytic organisms. On a diet of poor quality hay only, the consumption tends to decline after a few weeks due to a reduction in the rate of cellulose digestion. Roughly speaking, it would appear that cellulose digestion can be maintained on a 5% protein level and declines when this figure exceeds 10%. A ration of the latter composition is sufficient for maintenance but not for high production. Maximum fibre utilization and high production feeding would therefore appear to be incompatable. It must be remembered, however, that the nature of the fibre also largely determines its digestibility. Old, highly lignified material is very resistant. High producing animals should therefore receive only good quality roughage.

The amount of readily available carbohydrate (starch and sugars) also affects cellulose digestion. This was well illustrated in the case of an experimental sheep at Onderstepoort recently. The animal had been maintained for many weeks on a ration of teff hay *ad lib* while receiving 80gm. glucose and 10gm. urea daily through the fistula. Eighty grams of starch were then added to the above dose. Within six days the hay consumption fell to nothing and the cellulose digesting organisms virtually disappeared, while the pH of the rumen fell to below 4 due to the accumulation of lactic acid. Bacterial examination of the rumen also showed the presence of very large numbers of Gram positive cocci capable of fermenting starch and glucose, and of organisms capable of utilizing lactic acid. The animal was treated with calcium carbonate and ruminal fluid from a hay fed sheep, and given hay only. It recovered within a week. The probable explanation for the disapperance of the cellulose digesters is that they could not compete with the much more rapidly growing starch, sugar and lactic acid users for the limited supply of nitrogen. On a higher protein diet much greater amounts of readily available carbohydrate can be tolerated. In other words, a balanced diet must be fed, not only for the welfare of the animal, but also for that of the ruminal flora.

Starch, Sugar and Lactic Acid Fermenters. Starch is hydrolised through dextrins to soluble sugars which are then fermented to a variety of end-products including the volatile fatty acids and lactic acid. The latter is utilized by other organisms (e.g. *Veillonella gazogenes* and *Propionibacter*) with the production of propionic acid. Starch and sugar fermentation is accompanied by the very rapid formation of gas, mainly carbon dioxide.

NITROGEN METABOLISM

The Transformation of Protein.

Much of the dietary protein is hydrolysed by the proteolytic enzymes of the ruminal bacteria to peptides and amino-acids which may be deaminated with the release of ammonia. These end-products are then utilized for the synthesis of bacterial protein. Free ammonia only occurs in the rumen to any extent when excess protein is fed, (e.g. 3 mg/100 ml. on hay only and 30 mg/100 ml. when the hay was supplemented with 100 gm. fish meal). The bacterial protein is later digested by the host animal.

The proportion of the dietary protein so transformed will depend on:-

(a) *The total protein intake.* On a high protein intake the amount of protein will exceed the capacity of the proteolytic enzymes, and despite a marked increase in proteolytic types of bacteria, a greater proportion of the dietary protein will pass unchanged down the tract.

(b) *The solubility of the Protein.* Highly soluble protein is rapidly attacked. The rate of breakdown may exceed that of resynthesis so that, if given in excess, such protein may give rise to the accumulation of ammonia. Sparingly soluble protein, such as fish-meal, is only completely hydrolysed when given in small amounts while even less soluble types, e.g. zein, largely escape bacterial digestion.

Bacterial protein synthesis is also dependent on the presence of end-products of carbohydrate break-down, as a portion of the carbohydrate moiety of the deaminated amino-acids is attacked by other bacteria and converted to volatile fatty acids, and a portion of it dissipitated as gas. This must then be replaced from other sources.

It has been shown that all the 'essential' amino-acids can be synthesized by the ruminal flora. The ruminant is therefore not so exacting as other animals in regard to the quality of protein in the ration. Where the protein requirements are high, as in growing and producing animals, the supply of 'essential' amino-acids formed in the rumen may be inadequate and extra high quality protein in the diet becomes necessary. The high demand for sulphur bearing amino acids by wool-bearing sheep also cannot be supplied by the ruminal organisms alone.

The Synthesis of Protein. The fact that many of the ruminal organisms can synthesize protein from non-protein nitrogen (e.g. urea) enables the ruminant to utilize such nitrogenous compounds as a partial supplement to dietary protein. The urea is very rapidly broken down to ammonia by bacterial urease in the rumen. This ammonia can be utilized by certain species (e.g. *Streptococcus bovis*) for protein synthesis. It must be remembered that not all

bacteria can utilize ammonia, much of the formation of new amino-acids in the rumen is brought about by trans-amination of pre-existing amino acids rather than complete synthesis. Many species of bacteria also require certain, to them, 'essential amino-acids' for growth.

For amino-acid synthesis from ammonia, adequate carbohydrate is essential. The breakdown of cellulose is too slow to keep pace with the rapid release of ammonia from urea so that readily available carbohydrate such as starch or sugar must also be fed in adequate amounts. Commercial urea contains 42% N while protein contains 16% N. Theoretically therefore, one part of urea can give rise to 2.6 parts of protein. The same figure can be derived by saying that 100 gm. urea (42% N) will give rise to 42 x 6.25 = 262 gm. protein. Of this 220 gm. (84%) will consist of carbohydrate moiety. This would indicate that each part of urea must be accompanied by 2.2 parts of carbohydrate but it must be remembered that not all the carbohydrate is used for protein synthesis. In order to prevent the possibility of a dangerous accumulation of free ammonia, at least four parts of readily available carbohydrate should be fed to each part of urea and the intake of urea must be strictly limited.

If the total nitrogen intake were given in the form of urea, the growth of many of the functional ruminal bacteria would be limited to the protein synthesizing potentiality of the ammonia users and would be seriously curtailed. Furthermore, this potentiality is not great enough to supply the needs of the animal. For these reasons it is generally accepted that not more than one third of the total nitrogen should be given as urea. Assuming the daily requirement of protein for an ox to be 1 lb., this would allow the equivalent of some 5 oz. of protein being given as urea. The daily ration would then be 2 oz, urea.

Even those bacteria which can utilize ammonia as sole source of nitrogen, will use peptides and amino-acids in preference to ammonia. The amount of protein present in the diet therefore effects the utilization of urea. If the nitrogen needs of the potential ammonia users are supplied from other sources the ammonia will not be utilized and may accumulate to dangerous levels.

Normally, adequate sulphur is supplied in the protein of a natural ration but, where much of the nitrogen is given as urea, the synthesis of the sulphur-bearing amino-acids may become the limiting factor of ruminal bacterial activity. In America, the addition of sulphur to urea containing rations has given good results. This would not appear to apply to South Africa as the cane molasses used here contains considerable amounts of sulphur.

The limiting factors to the use of urea as a substitute for protein can be summarized as follows:-

(a) The number of bacteria capable of utilizing ammonia which are present. These must be built up by giving small initial doses which are gradually increased.

(b) The amount of readily available carbohydrate present in the diet.
(c) The amount and nature of the protein given.
(d) The fact that a large proportion of the functional ruminal organisms are dependent for their supply of certain amino-acids on the activity of the ammonia using bacteria and this supply must therefore be limited.
(e) The possibility of a shortage of soluble phosphate, sulphur, cobalt and other elements usually supplied in a natural mixed diet.

For these reasons it is contended in South Africa that urea should normally only be used to bring poor quality fibrous hay and winter grazing up to maintenance standards. Under these conditions the supplement not only supplies the required extra protein to the animal but also maintains fibre digestion at a high level.

RUMINAL GASSES

It has been shown that gas is a normal by-product of carbohydrate fermentation in the rumen. Readily available carbohydrates give rise to the rapid liberation of gas, mainly carbon dioxide, whereas the slower and more continuous fermentation of cellulose is accompanied by the steadier evolution of gas which is mainly methane. Figure 3 shows the total amount of gas produced from a single meal of lucerne.

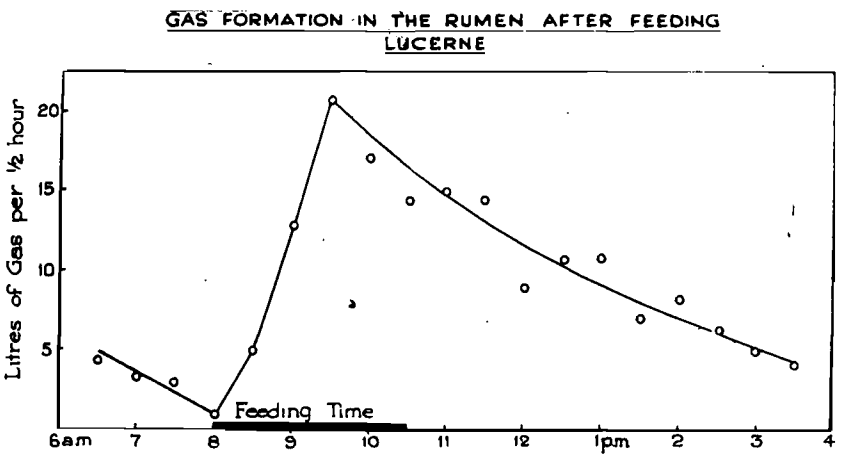

Fig. 3.

Gas formation in the Rumen after feeding lucerne.
(Adapted from Cole, Mead and Kleiber, Calif. Agric. Exp. Sta. Bull. 662, 1942).

According to Dukes, the percentage composition of these gasses, from lucerne and grain, at roughly corresponding times after feeding are as follows:

	Hours after Feeding.	
	2	24
Carbon dioxide	65	20
Methane	30	40
Hydrogen	5	0
Nitrogen	0	35
Oxygen	0	5

The oxygen and nitrogen present 24 hours after feeding are probably derived from air introduced during rumination. It will be noted that the oxygen disappears rapidly when fermentation is speeded up by feeding. The residual nitrogen would be washed out when the other gasses were being produced in large amounts.

THE RUMINAL TEMPERATURE

Considerable amounts of heat are formed during fermentation in the rumen, so that the intra-ruminal temperature is normally somewhat above that of the body. At the height of fermentation of lucerne, the intra-ruminal temperature may rise to 106°F. This extraneous source of heat in ruminants must result in considerable savings of body energy in cold weather and add to the difficulty of maintaining a normal body temperature in hot climates.

THE VOLATILE FATTY ACIDS

The volatile fatty acids, of which acetic, propionic and butyric form 85% of the total, constitute the chief end-products of normal carbohydrate fermention, and are absorbed through the walls of the rumen, reticulum and omasum. It is of interest to note that recent work in America has shown that the main stimulus to the growth of the papillae is the presence of these acids and the main function of the papillae would therefore appear to be the absorption of these substances. Acetic acid can be used directly for the production of energy and it is also utilized for the synthesis of fat. (The ruminant mammary gland, for instance is peculiarly adapted for the utilization of acetate for the production of milk fat). Propionic acid is converted to glucose by the liver and forms the main direct precursor of the blood sugar. It is also anti-ketogenic. Butyric acid is converted to ketone bodies by the epithelium of the forestomachs. Any rise in butyric acid in relation to propionic will therefore tend to produce ketosis.

The total amount of volatile fatty acid produced by an adapted flora depends on the nutritional value of the diet. Work at Onderstepoort has shown that the ruminal fluid of sheep on a generous mixed diet contains up to 180 milli-equivalents per litre of volatile fatty acids at the peak of production whereas the corresponding figure from sheep on poor hay is no more than 50.

The proportions of the different volatile fatty acids produced by an adapted flora remain remarkably constant on a wide variety of diets. Working in England, Elsden reports an average of

acetic 67%, propionic 19% and butyric 14% from sheep, cattle and deer. Table I gives the figures obtained at Onderstepoort from different species on a wide range of diets. The similarity of the proportions of the different acids and of the ratio between propionic and butyric acids should be noted.

TABLE I

Averages of Peak Volatile Fatty Acid Production in the Rumen of Sheep, Cattle and Wild Game on Different Diets.

Sheep

DIET GRAMS	Number of Animals	Adaption Time Weeks	Total Volatile Fatty acids m.eq./l.	Percentage Proportions			Propionic/Butyric
				Acetic	Propionic	Butyric	
Teff hay *ad lib*	7	3–4	59	73	18	9	2.0
Teff hay *ad lib* Crushed yellow mealies 200	3	4	70	73	23	10	2.3
Teff hay *ad lib* Crushed yellow mealies 400	3	3	74	63	25	12	2.0
Teff hay *ad lib* Crushed yellow mealies 200	6	14	46	70	19	11	1.7
White Fish meal 50							
White Fish meal increased 100	6	3	79	69	22	9	2.3
White Fish meal increased 150	6	3	73	70	20	10	2.0
Teff hay *ad lib* White Fish meal 400	2	3	57	69	21	10	2.1
Teff hay *ad lib* Lucerne hay *ad lib* Crushed yellow mealies 500 .... White Fish meal 125 Succulent green lucern 300	11	6	113	62	26	12	2.2

Cattle

Teff hay *ad lib*	3	4	70	75	16	9	1.9
Lucerne hay *ad lib* Cow meal 5000	3	4	89	70	18	12	1.5

Kalahari veld succulents *ad lib*	Springbok 1	adapted	73	76	16	8	20
	Gemsbok 1	adapted	72	73	17	10	1.7

THE ADAPTATION OF THE RUMINAL FLORA TO THE DIET

As already stated, the density and composition of the ruminal flora depends on the diet. A fully adapted ruminal flora can be defined as one that produces the maximum amount of normal assimilable products, in normal proportions, from a given diet. Should the diet be suddenly changed, the existing flora is no longer adapted and this is reflected either in the presence of abnormal products or in the production of abnormal proportions of normal products. Whether or not this in turn is reflected in the host animal depends on the extent of the change and on the conpensatory mechanisms within the animal itself. For instance, the high producing milk cow and the heavily pregnant ewe are already under stress as regards the maintenance of a normal metabolism especially in respect of blood sugar, ketone bodies, calcium, phosporus and magnesium. Even a relatively mild digestive upset may therefore produce metabolic disorder in one or more of the above factors. Dry cattle and sheep are much more resistant under such circumstances. The following examples of disturbances following sudden changes in diet may be cited, viz:-

(i) *Sudden Increase in Protein.* A sudden increase in protein may lead to ammonia absorption and alkalosis with a consequent disturbance in mineral balance in the blood especially as regards magnesium and ionized calcium. This in turn may give rise to tetany. For instance, a sheep adapted to a low protein intake may be poisoned within an hour by the administration of as little as 100 gm. of casein which would have no effect on a sheep on a normal protein intake.

(ii) *Sudden Reduction in Protein.* It has recently been shown at Onderstepoort that a sudden reduction of protein not only causes a sharp drop in the total volatile fatty acids, but also a decrease in the propionic/butyric acid ratio. Such a change might well lead to ketosis especially in an animal under stress.

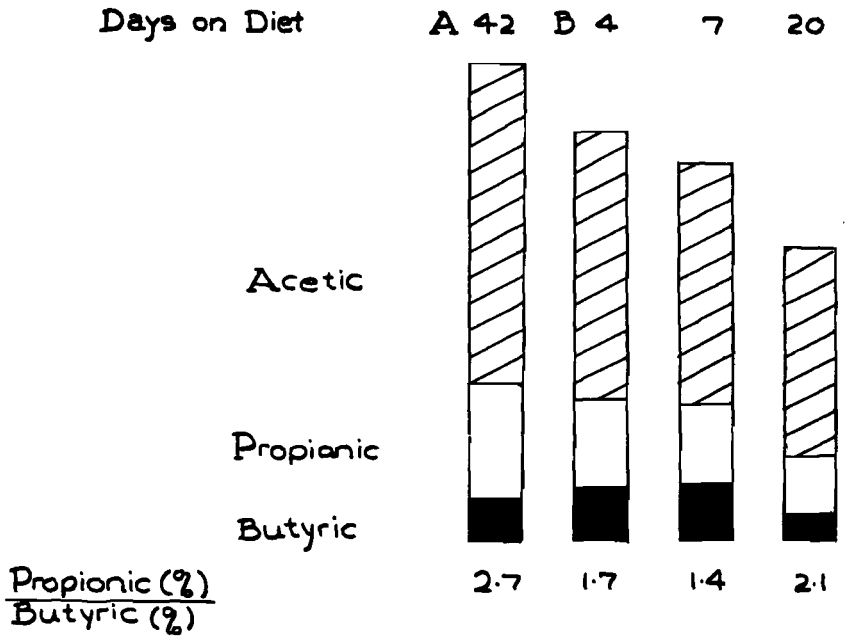

FIGURE 4

The effect of a sudden reduction of protein on volatile fatty acid production.

Diet A. Teff and lucerne hay *ad lib.*
White fish meal 125 gm.
Yellow crushed maize 375 gm.
Greenfeed 250 gm.

Diet B. Teff hay *ad lib.*

The sudden reduction of the ration was followed by a steady decline in total acids and a relative increase in butyric acid over the adaptation period. After three weeks the proportions of the acids were again almost normal, i.e. adaptation at a lower level had taken place.

The Effect of Dosing Sulphonamides into the Rumen.

In view of the importance of the ruminal flora, great care must be taken in dosing bacteriostatics *per os* to ruminants. Figure 5 shows the effect of dosing sulphadimidine (Initial dose 0.2 gm/kg. followed by 0.1 gm/kg. daily) to a sheep. The total volatile fatty acids fell from 40 m.eq./l prior to dosing to 27 on the fourth day. As will be seen from the graph, the propionic/butyric acid ratio fell sharply and the animal developed a considerable ketonaemia. It will also be noted that there was some sign of recovery even before dosing was stopped. This was probably due to adaptation of the flora.

It has been found that the recovery of the flora after dosing suphonamides is rapid, provided the animal has a normal flora initially. In sick animals, where the flora is already affected by a

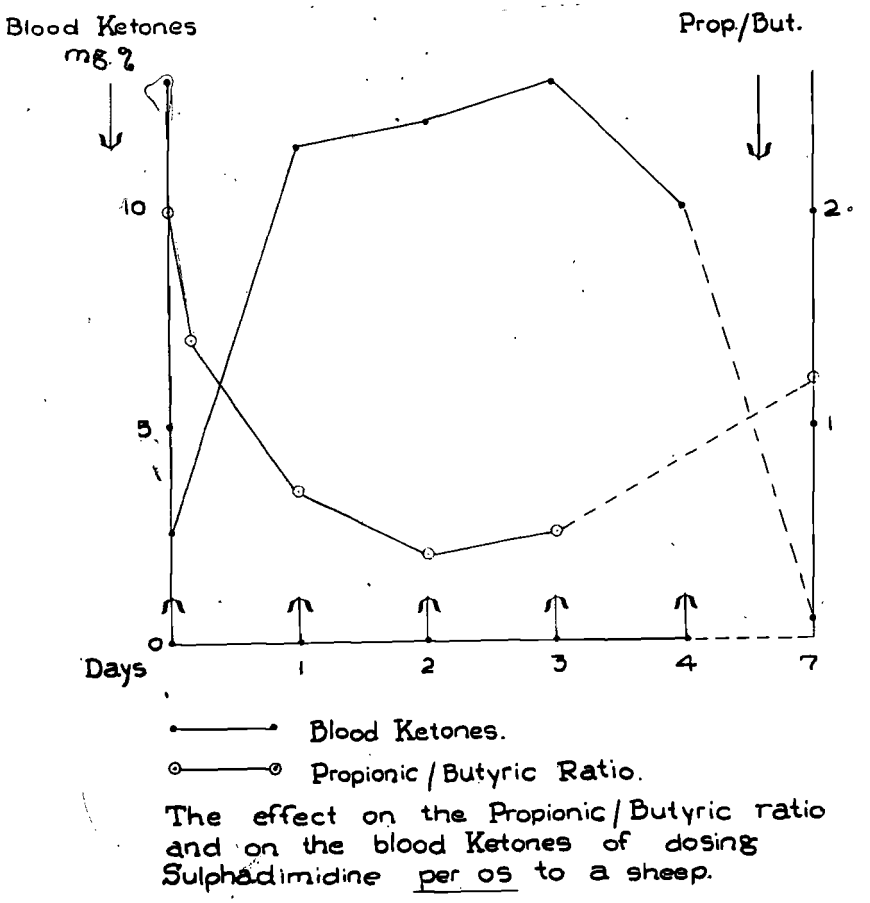

FIGURE 5

The Effect of Dosing Sulphadimidine to a Sheep.

diminished intake, serious disturbances may be precipitated. The general rule should therefore be not to dose such drugs to ruminants *per os*.

The Transfer of Ruminal Ingesta.

The experimental animals used in the Department of Physiology at Onderstepoort are subjected to violent changes in diet and other treatments calculated to produce upsets in the ruminal flora. At the conclusion of such experiments, the animals are rapidly re-restored to normal by dosing ruminal contents from a healthy animal on a diet similar to that on which the experimental animal is to be placed. Where necessary the pH of the rumen is also corrected by dosing acetic acid or calcium carbonate as required. The effect of such treatment is dramatic and it has become standard in our clinical work. It must be remembered that the ingesta must be fresh and from an animal on a suitable diet containing plenty

or roughage. The value of dried preparations of ingesta is open to considerable doubt.

CONCLUSION

In conclusion we may list the following golden rules in regard to the feeding of ruminants.

1. In order to make the greatest use of the ruminant's ability to utilize cellulose, always feed large amounts of good quality roughage. The whole tract is designed to handle bulk.
2. Concentrates must be balanced as regards protein and readily available carbohydrate.
3. Avoid all *sudden* changes in diet.
4. Where only poor quality roughage is available, this must be supplemented by protein (or urea and molasses) not only to maintain the animal's weight but also to allow of maximum fibre digestion.
5. Beware of dosing drugs which may effect the ruminal flora.

BOOK REVIEW

"HANDLEIDING OOR DIEREVOEDING"

By J. W. Groenewald

Die Drakensbergpers Bpk. — Durban — 1957.

The first edition of this popular and useful manual on the feeding of livestock appeared in 1950. The present new edition has been completely revised and brought up to date. The chapters have been reclassified, the headings have been increased in number and printed more prominently, many interesting illustrations and graphs have been added, and at the end of the book an appendix of more than a dozen most useful tables, assist the reader in the computation of balanced annual rations. An English translation is to appear shortly.

J.H.R.B.

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There is poor absorption of calcium after bowel infections such as enteritis. CALCI-OSTELIN, which provides easy calcium absorption, aided by Vitamin D, may come in useful in such cases. It has other uses, too. Sometimes of value in the treatment of delayed union of fractures, it may also exert a tonic action throughout the system and is frequently given for this alone.

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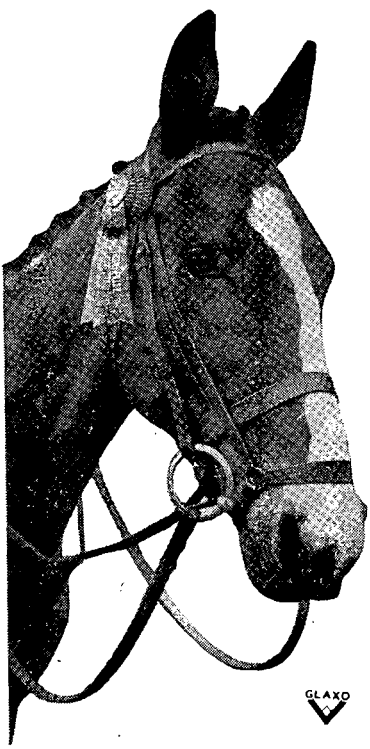

In the pink with B_{12}

Loss of condition in animals can be baffling. Even when the cause is known or suspected, and well tried measures have been taken, response falls short of the expected. Common among such cases are the debilitating digestive upsets caused by stress and strain, by dietary changes and by disturbances to routine such as the fuss of transportation. The animal loses appetite. The decreased food intake and the upset metabolism mean insufficient absorption of vitamin B_{12} without which even the little food ingested will not be utilised. In such cases, an injection of Cytamen (B_{12} Glaxo) quickly restores appetite, well-being, vigour, 'bloom' and performance.

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VISIT TO THE UNITED STATES, CANADA AND ENGLAND

5 OCTOBER TO 10 DECEMBER, 1955.

J. D. W. A. COLES

Onderstepoort

INTRODUCTION

This visit was the result of an invitation from the National Academy of Sciences in Washington to attend a conference on the use of antibiotics in agriculture. Both the Academy and the South African Egg Control Board afforded financial assistance, which was greatly appreciated.

It is manifestly impossible to cover all aspects of such a trip and it will perhaps be best to give some idea of the itinerary and then dwell on subjects of particular interest.

I travelled by way of Lisbon, where I spent a few most pleasant and instructive hours in the company of Dr. A. Taborda Duarte and Dr. F. Sousa Silveira. These two veterinarians very kindly showed me a good deal of the beautiful city and also the veterinary school and the poultry husbandry centre. I saw a case of Newcastle disease, which seemed to be prevalent in the Iberian peninsula, and also a typical outbreak of C.R.D.

In the United States and Canada I visited the Universities of Delaware, Iowa, Maryland and Massachusetts, Rutgers University, Cornell University, Purdue University, Michigan State University, the Army Medical School in Washington, the Beltsville Research Centre, the Connaught Medical Laboratories of the University of Toronto, the Regional Poultry Research Laboratory at East Lansing, the Northern Regional Research Laboratory in Peoria, Pfizer's laboratories in Terre Haute and Lederle's laboratories at Pearl River.

Naturally, I attended the conference in Washington on the value of antibiotics in farming and shortly afterwards was a guest at the annual October conference of poultry pathologists at Bear Mountain Inn, N.Y. This annual meeting, which is of inestimable value to the poultry industry, is sponsored by Lederles. At Cornell University I was invited to participate in a conference of the leading poultry breeders and hatcherymen of New York State.

In England I visited Cambridge University, Weybridge Laboratory and Houghton Grange Poultry Research Laboratory, and

then concluded the tour by attending the meetings of the British branch of the Worlds Poultry Science Association.

A singular pleasure awaited me in New York. I heard that Dr. K. F. Meyer had just arrived from San Francisco to attend a meeting. Besides imparting to me all the latest information on ornithosis in turkeys, which is now a minor public health problem in North America, he had much to say about his stay at Onderstepoort in the early days. Although over seventy, his zest for life, learning and enjoyment are unimpaired and he still treks from conference to conference in all parts of the world. To use his own words, "The Emeritus is still going strong."

International Conference on the Use of Antibiotics in Agriculture.

This conference was held in Washington under the auspices of the National Academy of Sciences from 19 to 21 October, 1955. Four hundred and thirty-one delegates were present and included several from foreign countries. The Soviet Union had three representatives. The Conference was divided into five sections, which met consecutively. They dealt with (1) Growth Response in Animals (2) Special Biological Problems (3) Mode of Action (4) Crop Usage and Food Preservation (5) Public Health Aspects. As was to be expected, there was a good deal of overlapping, but this was actually an advantage.

The Conference was well run and very successful. I was on the panel dealing with growth response in animals. Excellent reviews were read by Dr. McGinnis on poultry, Dr. Cunha on other animals and Dr. Clausen of Copenhagen on carcass quality in pigs.

I shall present those conclusions of most importance to veterinarians. All things considered, penicillin is the antibiotic of choice for fowls, turkeys and pheasants (ducks and geese virtually do not respond). For swine, most prefer chlortetracycline or oxytetracycline; for lambs, calves and colts it is chlortetracycline, in the absence of information on the value of any other; for dogs nothing exerts any benefit. For all practical purposes we can forget the other antibiotics, but streptomycin is the only effective one in controlling certain plant diseases.

It is a great pity that veterinary bacteriologists and virologists have been interested so little in the question of how the apparently sub-therapeutic doses of antibiotics function. Many nutritionists have endeavoured to show that antibiotics spare one or other vitamin or even protein, but the evidence on the whole is not convincing. As the result of work initiated by Coates in England, it is obvious that one and probably more "infections" exist that retard growth and may be subdued or even killed off by the administration of appropriate antibiotics. Animals raised in a germ-free environment, or even in new buildings with new equipment, show no response to the feeding of antibiotics. At some colleges the response has disappeared after a year or two, only to reappear

at a later date. Other experiments have shown that the "infection" is in the intestinal and respiratory tracts, and will pass through a filter with a pore size of 200 to 250. In the case of plants, streptomycin poured over the leaves and on the soil acts by destroying harmful bacteria and fungi.

It must be stressed that antibiotics cannot promote growth beyond the level attainable in a "clean" environment. Furthermore, they cannot make a good ration out of a bad one, but they do seem to help most when the diet is barely adequate.

It has become common usage to speak of nutritional levels when the amount of antibiotic does not exceed 50 p.p.m. Higher levels are known as therapeutic levels. Some investigators believe that chicks should be fed 200 p.p.m. during the first fortnight of life, when the value of penicillin is most pronounced, and then be switched to 5 to 10 p.p.m.

It is very doubtful whether the feeding of antibiotics can benefit adult animals or their progeny, in spite of claims that litters are bigger and piglets live better when the sows receive supplemented diets. Also there is good reason to believe that in the case of normal, healthy flocks egg production, fertility and hatchability are not improved.

Antibiotics are most effective for young animals, and the younger they are the more obvious is the response. Apparently the feeding of antibiotics to successive generations will not impair the fertility, growth and health of the following generations. There is no obvious merit in feeding combinations of antibiotics.

Chicks usually receive 5 to 10 p.p.m. of procaine or diamine penicillin until they are 8 weeks old, or until they are killed in the case of broilers. Growth is usually enhanced by 5 to 10%. Poults may show up to a 20% improvement and should be kept on the antibiotic until they are at least 14 weeks old.

Pigs receiving excellent rations and kept in proper hygienic surroundings show little response to antibiotic supplementation, but in the average poor environment they put on 10 to 20% more weight than controls, and there are fewer runts and less scouring. When the piglets are weaned at eight weeks, the weight improvement is often found to be five to ten pounds. It is desirable to provide the antibiotic up to the time of marketing. Pigs on good pasture profit less than do those in dry lot. Usually 10 grams of chlortetracycline or oxytetracycline are mixed in a ton of food. It is customary to creep-feed antibiotics during the suckling period. When pigs are given restricted diets in order to produce first class bacon, the feeding of antibiotics will not increase body length or the thickness of back fat or streak or change the percentage of lean meat or the chemical composition of the meat and fat. Generally speaking, antibiotics seem to give the most spectacular results when the pigs are for unknown reasons not completely healthy.

Calves should be fed chlortetracycline up to the age of 16 weeks. Not only is growth improved by 10 to 30%, but the incidence

and severity of scours are reduced. The daily dose for young calves is about 25 mgms. per 100 lbs. live weight. Milk replacement feeds should contain about 25 mgms. per pound of feed. It is probable that oxytetracycline will ultimately prove to be as valuable as chlortetracycline.

Further information on the value of antibiotic feeding is required in the case of lambs and foals, but it is known that they respond favourably and they should probably get the same amount as calves. Until we hear more, only chlortetracycline should be given to foals and lambs.

Personally I have no hesitation in recommending that all young chickens, poults, calves, lambs, piglets and foals should be fed antibiotics regularly, in accordance with the suggestions made above.

The Broiler Industry in the U.S.A.

According to the official figures that I obtained, about 34,000,000 broilers were slaughtered in 1934. Last year the number probably exceeded 1,100,000,000, and most were produced in Georgia. Even now the average American consumes only half an ounce of poultry a day, so there is still room for considerable expansion, particularly as the supply of beef and mutton cannot keep pace with the requirements of a rapidly growing population.

The salary of the average man in the U.S.A. is double that of his opposite number in the Union, but a broiler costs exactly the same there as it does here — about 3/3 to 3/6 per lb. retail. The cost of the broiler chick is also the same — 11½d. to 1/-. Mash costs about 50% more in the States than in the Union. The only reason why the American producer can show a profit at all is that one man raises about 10,000 at a time and the rations are somewhat superior to ours and their birds are bred to grow much faster. The two sexes will often average 3 lbs. liveweight at 9 weeks, with a feed conversion rate of not more than 2.8 to 1.

During the past three years the packers have demanded only birds with yellow skins and the plumage has to be white or nearly so. Naturally, only heavy breeds are crossed and the Leghorn seldom comes into the broiler picture. All this has meant the disappearance of the Barred Rock. While some N.H. strains are extremely good, packers refuse to pay normal prices because of their colour. However, N.H.'s are used on a very large scale for crossing, for instance, with Vantress males and Dominant White males. The only purebred raised on a large scale is the White Rock and it is amazing to see how many of these birds have crossed beaks, split wings and deformed feet. Everything has been sacrificed to rapid growth, just as some White Leghorn breeders have concentrated on raising the hen-housed average production to the exclusion of all else.

There is a negative correlation between rapid body growth and egg production, and so poor layers are usually the dams of the best broilers. However, the price of chicks cannot be kept

down if the dam lays badly, and so there is a sort of unwritten law to the effect that the dams should have a hen-housed average of about 150 eggs. The overall hatchability in the Delmarva Peninsula is 72% of all eggs set, which is good.

While most broilers are processed now in modern packing houses, the Americans have found it uneconomical to dress less than 10,000 daily in one of these centres. For this reason, we can hardly expect many packing houses to be built in the Union and dressing will have to be continued largely on the farms. My own view is that our health authorities should license these farm dressing plants, just as they do the dairies.

Many broilers are retailed in attractive plastic bags, but the majority are not specially packed. There is a strong tendency to dismember the carcass and sell only the breasts or legs or livers, for instance. These pieces are displayed in counter refrigerators in rectangular, strong cardboard punnets with cellophane lids.

There is a big trade in broiler — squabs (about 1 lb. dressed weight) besides broilers (2 to 2¼ lbs.). Roasters of about 3 lbs. are not so popular.

Chemical caponising with hormones has been largely forgotten, but some maintain it has a place in the production of roasters of fine appearance and texture.

Debeaking to prevent cannibalism is practised very widely. The outer third of the upper beak is removed, usually at the age of three weeks. The chicks are fed mash until two days after debeaking; then they are given pellets.

Towards the end of 1955 the Federal authorities approved the treatment of dressed broilers with chlortetracycline to prolong their "shelf-life." Most counter refrigerators run at 6 to 8°C and broilers kept in them begin to spoil after 36 hours. If the birds are dressed and drawn very cleanly and are then immersed for 45 to 60 minutes in ice water containing Acronize. P.D. (a preparation of aureomycin intended for this purpose) the "shelf-life" in the counter refrigerator may be extended to a week or more. The importance of this is very considerable, and no less to us than to the Americans. Cooking of the bird destroys any residual antibiotic.

There are at present seven random sample broiler growth tests in the United States and I visited the one in Amherst, Mass., where the best unsexed groups averaged just over 4 pounds at 10 weeks. These tests have contributed a very great deal to the development of the industry, because first class breeders have been given the publicity that was their due. No amount of slick advertising by poor breeders can compensate them for the loss of business due to the indifferent showing of their stock on these tests. Such tests should be established in the Union without delay, if we intend our broiler industry to flourish. The birds are usually held for ten weeks and then dressed.

The sprincipal broiler diseases are coccidiosis, synovitis infectious bronchitis, Newcastle disease and above all, C.R.D.

Egg Production in North America.

There were 489,000,000 layers in the United States in 1943 and only 400,000,000 in 1953. And this in spite of a human population increase of well over 20,000,000. Two facts are responsible for the drop in numbers; there are many more pullet flocks, and the hen-housed average has been raised considerably.

There has been a most pronouced swing towards the medium sized W.L., because this bird produces so many eggs so economically and can also be sold more readily, when discarded, on account of its white plumage. Many flocks of egg-type R.I.R.'s still exist, and also N.H.'s of the same small size. I am told that the Austra-white is in favour around Los Angeles, but the idea of a dual purpose bird has virtually been abandoned. You produce either eggs or broilers, and old hens are sold to the poorer people or converted into soup and canned chicken.

The general farmer in the Middle West is producing relatively fewer eggs and gets less for them, because shipping for long distances does not improve quality and the standard of hygiene on the general farm is lower than that on the highly organised egg farm near the big city.

Batteries are preferred by numerous egg producers in the Deep South and California, but elsewhere the pullets are kept in flock houses. As far as can be judged, neither system is really better than the other. I met the owner of an airconditioned plant in Iowa, where 4,000 birds were kept in batteries supervised by one man. It was the first of its kind in the state and was claimed to be functioning perfectly.

To cut labour costs, most farmers now feed only all-mash laying meal.

Generally speaking, egg packers prefer Keyes equipment.

I went into the question of oiling eggs at Beltsville, where a good deal of experimental work has been done. Only perfectly clean unwashed eggs should be oiled and they should be treated within a day of being laid, the earlier the better. The oil must be colourless and odourless and light enough to penetrate the shell. Although the oil must not dry out, the shell must not look oily. Nujol is very desirable for oiling eggs, which can then be stored, even at room temperature for two months if need be.

There is a marked tendency in the modern super-markets to sell eggs in attractive cardboard cartons, each of which holds a dozen.

Newcastle disease and infectious bronchitis play havoc with laying flocks and lead to the appearance of numerous worthless eggs of bad shape and colour. In areas where the danger is great, the producers have no option but to vaccinate the young pullets. Bluecomb, which is undoubtedly the same as our visceral gout, is prevalent all over North America and may lead to heavy losses in production. The cause of this condition is still unknown, and although potassium chloride, molasses, acetic acid and oxytetra-

cycline are all recommended cures, there is no definite proof of the efficacy of any one of them.

Breeding for Egg Production.

For sometime it has been apparent that the hen-housed average cannot be increased by present methods much above 200, even when only the members of the best families are incorporated in the breeding pens.

Some investigators have shown that the average fall may be even 30, within the space of four years, if the selection of breeding stock, based upon careful record keeping, is abandoned.

So on the one hand we are forced to go on struggling to maintain production, and on the other hand we are unable to make any further worthwhile advances.

Despite the doubts of geneticists, one organisation in particular decided to burst through the barrier to progress by crossing highly inbred lines, as was being done with maize. The trouble, of course, is that you need to make 17 or 18 brother-sister matings to give the same degree of homozygosity that may be expected from 4 or 5 generations of selfpollination in maize. And, unfortunately, fowls cannot stand more than 2 or 3 generations of brother-sister matings!

To cut a long story short, I need only mention that most if not all geneticists now admit that all the intensive inbreeding has not been worthwhile. Furthermore, most poultry pathologists feel that intensive inbreeding is very apt to increase the incidence of leucosis.

Reciprocal recurrent selection is now often thought of as a means to our end, but nobody has yet proved its value.

In the absence of anything better, the closed flock is favoured probably by everybody and, by definition, a closed flock is a pure strain. The coefficient of inbreeding has to be kept below about 15%, and the higher the percentage rises the more accurate must be the records, in order to offset the evils of even moderate inbreeding.

The search all over the poultry world today is for pure strains that will nick when crossed. Even though the hen-housed average of the hybrid may not be more than five or six above that of the better pure strain, the difference is most important economically. In the U.S.A. small breeders of pure strains are now cooperating with one another, so that they may join forces permanently if their strains nick.

In spite of the possibilities of heterosis, the enthusiastic small one-strain man is likely to survive, for very often the pure strain is better than a first class hybrid. The need to employ veterinarians and applied geneticists to improve breeding practices has led to a decrease in the number of breeders and an increase in the scope of their activities. The smaller breeders, who cannot afford such staffs, nearly always rely on lecturers in the various faculties of agriculture to solve their problems.

At Cornell, I went into the question of testing three sets of cockerels on one lot of pullets every year. It may pay them dividends, but I doubt if it would solve our problems at Onderstepoor. We have reduced malignancy from 50 to 4 or 5% and we need to get down to about 1.5%. Furthermore, the female is at least as important as the male in determining whether her progeny will be tainted. For us there seems to be more hope of success in testing one lot of cockerels on one lot of pullets, followed by another lot of cockerels on yet another lot of pullets. The best of the birds can then be reserved for future breeding purposes.

The more I see of the problems facing the breeders of well known college flocks, the more I realise that every flock is a study in itself and can be improved only by methods peculiarly suited to it. There is no golden rule, except that it seems to be necessary to retain a closed flock of some kind. Many breeders are probing in several directions to see how best to smash the barrier that is now represented by a hen-housed average of roughly 200 to 220, but so far the results have not been spectacular.

I must now allude to a very serious matter. On several occasions I found breeders spending much time and money on improving flocks still infected with epidemic tremor and C.R.D. They have put the cart before the horse and are now paying for their misfortunes. In the Union, we are only at the beginning of serious breeding work and we should be careful always to start with disease-free stock.

Our farmers are often told to raise and maintain the standard of their flocks by buying cocks or even day old cockerels from special breeders. Naturally, it is most unwise to bring anything over a day old on to a farm, because of the danger of introducing diseases, but we must also have grave doubts about the value of importing even the youngest males. With large animals it is customary to buy mainly males because for economic reasons we can hardly do otherwise; but with poultry which multiply so easily, the position is entirely different and, if a man is not completely satisfied with his hens, he should replace the whole of his stock with desirable birds of both sexes from a closed flock and not attempt to improve matters simply by buying males. This is extremely simple to do, as we know only too well. From the strictly scientific point of view, it is most doubtful whether the introduction of outside males often leads to significantly higher egg production.

While it is extremely difficult to increase the speed of a horse, or egg or milk production, it is relatively easy to breed better table birds, simply by selecting those that reach the desired weights most quickly and economically, and so I do not need to say more about the improvement of broilers and turkeys and ducks.

Egg Laying Tests.

I discussed these tests at great length with several authorities, including statisticians.

There used to be between 35 and 40 conventional laying tests in the U.S.A. and now there are only seven or eight and even these are expected to disappear in a year or two. I did not meet a single scientist who was willing to concede that ordinary tests could serve any further useful purpose. At the best of times, they had only slight educational value.

The advent of the random sample test has put an end to all thought of any other tests and made obsolete the R.O.P. and old poultry improvement schemes. Indeed, very few supporters of the R.O.P. scheme remain.

As was to be expected, the random sample tests did not get off to a good start. Nobody knew all the answers in advance, and there were disconcerting outbreaks of disease like C.R.D.

Random tests have been established in Utah, Wisconsin, Florida, Louisiana, New York, California and Missouri. More states are expected to join the scheme shortly. The supporters of the random sample tests are all progeny testers on a large scale. They may not enter unless they have at least 750 females representing any one strain or grade actually mated up. The eggs are collected during one day and then graded. Of those suitable for incubation, 150 to 180 are selected at random and sent to the test. All competitors' eggs are incubated together and the first 50 pullets discovered by the sexer usually constitute the sample. Records are kept until the birds are 500 days old and then they are sold, because from the point of view of disease control it would be most unwise to return any to the farms concerned. The published results reveal the mortality figures and causes of death, hen-housed average production, egg size, shell quality, prevalence of blood and meat spots, albumen quality, feed conversion rate, body size and profitability. When the profitability is determined, the catalogue price of the chick is taken into account. Information on freedom from show bench defects can also be supplied, if necessary. The competitor must state whether he is entering a pure strain, a strain cross, an incross, an incrossbred or a crossbred, so that potential buyers of his stock may know exactly what they are to get. The importance of this last point cannot be over-emphasized, for nobody wishing to go in for breeding himself would buy anything except a pure strain.

All experts on random testing are agreed that at least 50 pullets must be entered and that it is not wise to judge any breeder on the results of less than three tests.

Due to considerable trouble with respiratory diseases, particularly C.R.D., it is very likely that competitors in future will have to guarantee the freedom of their stock from diseases transmissible through the egg.

There is no denying the fact that many household names in the poultry world have become tarnished as a result of the tests revealing the weaknesses of the stock concerned. It is, however, equally true to say that the tests are doing much more than was

ever expected of them to raise the general level of the birds. For the first time in history, the breeders now know where they stand vis-a-vis one another and do not hesitate to concentrate on eliminating defects that are found. Some have saved time by scrapping their own inferior birds and replacing them with first class stock.

Random testing has many other advantages. There is no need to employ a small army of inspectors to go from farm to farm to see that records are being kept properly or that only decent birds are being put in the breeding pens, for the test itself will reveal whether the breeders are lax or dishonest. Provided a breeder has at least a pure strain, i.e. a closed flock, it does not matter much how he breeds. Indeed, the test has revealed the weakness of close inbreeding and shown that there is a good deal less in hybrid vigour than is generally supposed. It is remarkable how the best pure strains have more than held their own, even with the best strain crosses.

Before the advent of random testing, the state of affairs in the U.S.A. resembled the position prevailing in the Union at present. People often kept a breed for no reason other than that it was in fashion for the time being, generally due to slick advertising. Hatcheries could not predict with certainty what the next year's orders would stipulate and their egg suppliers were frequently in a quandary. Today all this is changed. The American egg producer now knows what strains lay best and most economically and where he can get the chicks. He no longer has to gamble as he does here. And, incidentally, the medium-sized White Leghorn has finally triumphed.

Random sampling has been the salvation of the careful American breeder and he now sells chicks to the multipliers at two to three times the price charged the farmers by the multipliers. This is as it should be.

It is common knowledge that most of our hatcheries buy eggs all over the place and do not worry much about the quality of the chicks they sell. This has led to a demand for the registration of hatcheries, but nobody has yet succeeded in defining precisely how a hatchery can qualify to be registered, particularly when no acceptable definition exists of a good chicken. It is no use making regulations that mean little if anything. The random test, as the Australians have realised, can solve our problem. We can register those hatcheries that obtain all their eggs only from (1) breeders who have proved their stock on the test or (2) multipliers who buy all their replacement stock every year from such breeders. Nothing can be simpler, and the general farmer purchasing his ordinary chicks will know for the first time in our history exactly where he stands.

It is interesting to observe that a dozen proven breeders would be able to meet the demands of the whole of our poultry industry.

The Turkey Industry.

During the past few years there has been a considerable change in the structure of the industry. Large flocks of 20 to 30,000 birds have almost disappeared and the tendency is to run flocks of only a few hundred. No one breed seems destined to oust the others, but the Empire White and Broad Breasted Bronze do not mate very successfully and artificial insemination is practised, not only in the U.S.A. but on a growing scale in Britain.

Because of our special interest in the Beltsville Small White, which seems to do so well in the Union, I visited Dr. Marsden. He assured me that we had nothing to worry about if our birds averaged only 80 eggs during the breeding period of $5\frac{1}{2}$ months. The Beltsville Small White does not convert feed quite as economically at the other breeds, but the quality and flavour of its flesh are unbeatable.

There are five random sample turkey growing tests and the birds are kept for 24 weeks. It is customary to market hens at 22 weeks and toms at 24 weeks. The flesh of the hen enjoys a premium of about 10 cents per pound.

Paratyphoid is still a serious disease and blood testing is regarded as a waste of time because far too many carriers, particularly those with pure intestinal infections, react negatively. Furazolidone seems to be of only limited value in controlling outbreaks.

Blackhead and hexamitiasis are troublesome except where the hygiene is specially designed to reduce their importance to a minimum. Synovitis is increasing slowly in significance, if rapidly in the broiler industry. Swine erysipelas often leads to much trouble even on land left idle for years; sick turkeys can be saved if the flock is examined three times daily and all birds off colour are treated immediately with penicillin. Many human cases are due to handling diseased turkeys. Spirochaetosis is apparently of consequence only in California and Leucocytozoon smithii infection occurs in the south-eastern states. Fowl typhoid is not very prevalent. Moniliasis, which affects the crop and proventriculus, is of little consequence, and so is staphylococcal arthritis. In states like Minnesota and Michigan, trematode infections of the oviduct can be very troublesome, not only in turkeys but in ducks and fowls. The birds become infected by eating dragon flies or their naiads.

A disease which has emerged as a minor public health problem is ornithosis, which has so far not been found in domestic poultry in the Union, although the infection exists here in pigeons and other birds. There have been a few deaths among packing house employees and five veterinarians and one assistant contracted the disease in a diagnostic laboratory. The incorporation of antibiotics in the feed of infected flocks has failed to control the disease.

An interesting condition, now being seen more and more in North America and Britain, is haemorrhage from the aorta. The

birds die suddenly. Broilers are also dying of the same complaint, the cause of which is unknown.

Turkey coccidiosis has increased in importance during the past few years and there are four species of Eimeria responsible for it. Nitrofurazone preparations are apparently useless for turkey coccidiosis and even sulphadimidine sodium has only limited value.

Sinusitis is a great problem and the causal pleuropneumonia-like organisms can pass through the egg, particularly when the disease is active in a flock. The condition may be cured by injecting terramycin or streptomycin into the swollen sinus, but recovered birds remain carriers of the bacteria and treatment is rather costly. Lesions may be seen in the lower respiratory tract and the resulting disease is then most severe. There is only one way to overcome infection due to pplos, and it is the same as that recommended in the case of C.R.D. in fowls. One strain of pplos, originally isolated in California, produces a fatal meningitis in turkeys and survivors develop the usual respiratory symptoms. Blood testing for pplos in turkeys has limitations, particularly in the case of the rapid plate test; too many known carriers react negatively and the tube test is preferred.

In conclusion, I should say that there is no possibility as yet of the turkey or the duck industry challenging the ordinary broiler industry. Table ducks and turkeys simply cannot be produced as economically as broilers.

Nutritional Problems.

Because of the rapid development of our broiler industry and the whole science of nutrition, it was only natural that I should devote particular attention to the feeding of poultry. It will perhaps be best to summarise briefly a few points of general interest.

(1) The balanced feed industry now ranks as the ninth or tenth industry in the U.S.A.
There are 6,000 brands of feedstuffs, sold by firms mixing 1,000 to 3,000,000 tons per year. In 1954 the industry handled 35,000,000 tons of mixed feedstuffs and 117,000,000 tons of all feedstuffs.
Poultry consumed 61% of balanced feedstuffs and pigs only 10%.

(2) In 1954 the balanced feed industry used 560,000 lbs. of antibiotics.

(3) While feed is sold in bulk on a large scale in the eastern states, in the Middle West it is nearly all bagged — 98%. Paper bags are not favoured. Bags of feed weigh 50 or 100 lbs., and not 150 lbs. as in South Africa. Every practical poultry farmer here knows how difficult it is to handle 150 lb. bags.

(4) Owing to labour shortages, very little grain is fed. All-mash rations are the rule and frequently they are pelleted.

Practically all broiler mashes are pelleted, if used after the age of three weeks. Virtually all duckling mashes are pelleted, because this reduces the risk of botulism. There is no decomposing wet mash on the floor to be eaten.

(5) The use of fresh greenfeed is declining owing to the high cost of growing it. Lucerne meal and synthetic vitamins can be added to the mashes to make good any deficiencies.

(6) Fermentation products, the one time sources of the "unknown factors," are not often used, because our knowledge of avian nutrition has increased so much and cheap synthetic vitamins are freely available.

(7) Milk products are hardly ever used in poultry mixtures, because they are inclined to be expensive, and again we have the vitamins to replace them. Furthermore, milk reducing sugars affect adversely arginine, lysine, tryptophane, histidine and other constituents.

(8) Wheaten bran has been shown to be of little value and is not often used now for chickens. About 10% may be incorporated in all-mash laying meal.

(9) Although the addition of methionine is thought by some to decrease cannibalism, the general opinion is that only poult mashes should be supplemented, and then only when there is a shortage in the diet.

(10) All chick, poult and duckling mashes are today fortified with synthetic D_3 at the levels recommended by the National Research Council.

(11) Synthetic vitamin A is used in all mashes so that the A. content is three times that recommended by the N.R.C. There is thus no risk of a shortage in spite of oxidation. So many of our lucerne meals contain only a fifth or even less of the A that can reasonably be expected in them, that we ourselves have no option but to rely more on the synthetic product. Some of our yellow maize contains none.

(12) It is probably best not to use over 12% fish meal in a chick feed.

(13) Instead of using peanut oil cake meal, it is better to use equal parts of it and a good sunflower oil cake meal. The proportion of sunflower meal to peanut meal may be even 2 to 1. This gives a better amino acid balance, for peanut meal is rich in arginine and low in methionine and lysine, whereas with sunflower meal it is the other way round. Unhappily most of our sunflower oil cake meal is not of high quality.

(14) Keep fibre in a broiler ration down to 4%.

(15) Total NaCl in a chick ration should be 0.5 to 0.7%.

(16) The choline content of a chick diet should not be less than

700 mgms. per pound. Most of our mashes contain only 450.

(17) The riboflavin content of a broiler mash should be at least 400 gamma per 100 grams, and not 300 as stipulated here.

(18) Lysine is perhaps the amino acid that is most easily destroyed by overheating of fish and other meals. Too little leads to decreased pigmentation of both chicken and turkey feathers. It seems to be imperative to define very clearly what quality fish meal may be sold in the Union. The expeller process leads to overheating of oil cake meals and destruction of lysine.

(19) Returned sacks in America are usually taken over by other companies, sterilised and then sent back to the feed manufacturers.

(20) The value of surfactants in poultry feed is extremely doubtful.

(21) The value of arsonic and arsanilic acids is disputed, but some of the best nutritionists feel it is worthwhile to incorporate them and consider that they improve feathering. The amount is usually 0.005%.

(22) High energy laying mashes are favoured. Birds on a 1000 Cals per pound diet consume about 2 lbs. less food per dozen eggs laid than birds on a 700 Cals diet. This is why our high maize diets are so much more economical to feed than European rations, and why our Leghorns eat $3\frac{3}{4}$ ozs. feed per day and not 5.

(23) Ducklings should receive about 90 to 100 p.p.m. niacin. Turkeys need virtually the same. We have seen the bowed legs in ducklings and distorted hocks in turkeys fed on our standard diets.

(24) Soaking and partially fermenting hard grains lead to a serious decline in vitamin E potency. Yet this is not an uncommon practice in the Union.

(25) Although very high energy rations containing animal fat give excellent results in broiler nutrition experiments, they are not yet being manufactured on a commercial scale, mainly because of mixing difficulties.

(26) Isoleucine deficiency definitely causes curled tongues in poults, a condition that we have diagnosed only once.

(27) No white maize is now grown in the U.S.A.

(28) Nutritionists are watching the folic acid content of chick diets more and more. The amount should be at least 100 gamma per 100 grams. Our rations are usually on the border line, if not a little below.

(29) Various authorities differ slightly when deciding how much of each amino acid should be in a 20% protein chicken mash. The following amounts are considered to be adequate

— lysine 1.1%, tryptophane 0.2%, arginine 1.4 to 1.5%, glycine 1.4 to 1.5%, methionine at least 0.45% and the sulphur group not less than 0.8%.

(30) Some firms advocate feeding ducklings for two weeks on a high energy 20% protein diet. Then the birds get an 18% protein mixture until eight weeks of age, when they are killed. It takes 19 lbs. feed to produce a duck of 6 to 6¼ lbs. a feed conversion rate of 3 to 1. Neither ducklings nor poults can convert feed as economically as broilers.

(31) Another recommendation is that ducklings should be fed on a 21% protein diet during the first three weeks and that not more than 7½% fish meal should be incorporated. The fibre should not exceed 7%. No choline is added. The amino acid requirements are still unknown. Allow ± 50 Cals for every 1% protein and add at least 30 grams niacin per ton. In all other respects the diet is virtually the same as a broiler ration. The only change made in the mixture for ducklings of three to eight weeks is a lowering of the protein to 17 or 18%. During the first three weeks feed ⅛" pellets and then 3/16" pellets.

(32) Some recommend a 30% protein diet for poults during the first four weeks.

(33) At Cornell I saw the experimental work on haemorrhagic diathesis of chicks, in which the subcutaneous tissues become full of blackish fluid. This peculiar condition is prevented by adding alphatocopherol acetate to the diet, but not by adding an antioxidant. Adding brewer's yeast helps but not torula yeast, and as brewer's yeast contains no E, it must have an E-like factor. It appears that when the diet contains relatively more unsaturated fat, there is a greater risk of nutritional encephalomalacia developing. In the presence of relatively more saturated fat, we are apt to see haemorrhagic diathesis. Both conditions must be considered to be vitamin E deficiencies.

The progress being made in avian nutrition in North America is remarkable and our feed manufacturers need to keep abreast of developments.

The Respiratory Diseases of Poultry.

Fashions change in both human and animal diseases, sometimes quickly and sometimes slowly. There are generally excellent reasons for these changes, notwithstanding the comments of the cynics. Changes in husbandry create new disease problems, changes in manufacturing processes confront the nutritionist with unforeseen difficulties, and the eradication of one major infection often leaves the field clear for the spread of another. And so in the poultry world we have surmounted, sometimes brilliantly and sometimes not so well, the obstacles labelled rickets, perosis, B.W.D., typhoid,

pox and coccidiosis, to end up with a real mess on our hands — the group of infectious respiratory diseases. These include Haemophilus gallinarum infection, fowl cholera, infectious bronchitis, infectious laryngotracheitis, Newcastle disease, aspergillosis, infection with the virus described by Fahey and Crawley and above all, chronic respiratory disease or C.R.D.

As C.R.D. is the only one of consequence to us, and is so widespread here and responsible for such financial losses, I spent much of my time studying the problem in various institutions. The disease in North America looks exactly the same as it does in the Union. It is a slowly spreading respiratory infection with an incubation period usually of two to three weeks. Symptoms are generally seen in chicks over a month old. Frequently only a few birds show head lesions. Poor weight gains are responsible for most of the monetary loss, as the mortality is only about 10 to 20%. The infection may pass through the egg, particularly when the disease is active in a flock, though on some breeding plants this appears to occur less frequently than formerly supposed. Nose and mouth secretions contaminate the food and water and so the disease spreads quite easily from one bird to another. The infection may be air-borne. As natural immunity is so poor, there is little likelihood of a vaccine being developed. It is extremely doubtful if any drug treatment is efficacious.

About the etiology, the last word has certainly not been said. While some believe that pleuropneumonia-like organisms (pplos or Mycoplasma gallinarum or Asterococcus avis) alone may be responsible, most are convinced that an additional factor is usually required to fan the pplo infection into flames. The virus of infectious bronchitis, for instance, may be the predisposing factor even though no symptoms of bronchitis are evident. Certain it is that a pplo infection can often exist on a farm for years without any visible outbreak of C.R.D. developing. From a practical point of view, however, we do not need to worry, for as far as we know there can be no C.R.D. without pplos and our aim must be to establish pplo-free breeding flocks.

Generally 50 to 95% of fowls in a pplo-infected flock react as carriers to the blood tests that have been evolved, particularly if the birds are over four months old. Thus it is seldom necessary to test more than twenty adult fowls selected at random in order to declare a farm infected. If 10 to 20% of adult birds are bled at random and are found to be negative, it is probably safe to consider the flock to be clean. There is no possibility of clearing up a plant by blood testing, because the pplos are spread easily through the food and water and some birds will always be in the incubation stage and so fail to react.

It cannot be over emphasized that the only way to rid a farm of pplos and hence of C.R.D. is to depopulate it for one month. All poultry of any age or species should go in order to play safe — certainly fowls, turkeys and ducks.

Depopulation may be carried out in easy stages, if no possibility exists of the new clean stock becoming infected from the old. The replacement stock should be obtained preferably from a known clean farm. Sometimes a breeder is fortunate enough to hatch only clean chicks from eggs from his own infected stock, but nobody can predict with certainty whether such a gamble will have a happy ending.

In the Union, we have very little cholera and no infectious bronchitis, infectious laryngotracheitis or Newcastle disease. Few poultrymen realise how fortunate we are that this is so and no pains should be spared to keep out these conditions.

Neoplastic Conditions.

No poultry pathologist can ever escape far from what is probably the greatest problem in medicine. For after we have eradicated or controlled all the infectious diseases and parasites and housed the hens properly and fed them well, we remain face to face with a mortality rate from all forms of cancer of up to 50%. The importance of heredity has been appreciated for many years by those who have studied closed flocks, and it is interesting to see how the random sample laying tests have confirmed their beliefs. The malignancy pattern is quite characteristic for any closed flock; year after year about the same percentage of birds will be lost and the types of cancer found will not vary much from one generation to another. For instance, the Onderstepoort White Leghorns are remarkable for the fact that roughly 1.5% develop gliomatosis and that only 4 to 5% succumb to all forms of malignancy put together. There is also a very strong familial predisposition to this gliomatosis. In apparently no other flock in North America or Britain is exactly the same state of affairs to be found; their problems are different, and usually worse.

Although it is virtually impossible to get accurate figures, a general belief exists and seems to be well founded that the crossing of inbred or even ordinary strains is very often apt to raise the cancer rate. It is incumbent on the vendors of day old chicks to make sure that the crosses they sell are not tainted in this manner.

It was interesting to find that experienced diagnosticians were all familiar with "outbreaks of lymphoid leucosis" such as are encountered in the Union from time to time. Not only does the aleucaemia appear as an epizootic, but the liver is nearly always markedly enlarged. If we believe that a virus is responsible, it is necessary to assume that the local strain is more than a match for the level of resistance inherited by the birds concerned. What has been said of this lymphatic aleucaemia is no less true of neurolymphomatosis.

I was most fortunate in being afforded every opportunity to see the extensive work on leucosis being conducted in the Regional Poultry Laboratory in East Lansing. However, the significance

of these investigations to us in South Africa is problematical, simply because the transmissible types of the disease with which they are experimenting are quite unlike the naturally-occurring and exceedingly common cases of lymphatic aleucaemia here, as well as our very rare cases of lymphatic leucaemia.

Because we hardly ever see sarcomata now in the Onderstepoort flock, or in birds sent by the farmers for examination, whereas they were not uncommon twenty years ago, when the same veterinarians did the diagnostic work, it has been suggested that all avian tumors found in selected laboratories should be examined microscopically for a period of thirty years or more. The picture in most laboratories has been obscured by the fact that staff changes have been relatively frequent and tumors have only occasionally been sectioned. In view of the dramatic increase in human pulmonary carcinosis, it is of the utmost importance to ascertain whether other types of cancer are on the increase or decrease, no matter what the outcome is of the controversy over smoking.

To those poultry breeders who hope for an easy solution to the cancer problem, I can offer no solace whatsoever. We must still keep accurate records of mortality, and exclude from the special breeding pens any hen whose sire, dam, brother, sister, son or daughter has developed any malignant condition. A cock may be used again if only a couple of his progeny have gone down, provided his father, mother, brothers and sisters have fulfilled expectations. It is not easy, but then nothing ever is easy in the breeding of first class animals.

The Connaught Medical Research Laboratories of the University of Toronto.

While virtually all manufactured pharmaceutical products are prepared by commercial concerns, there are generally three ways in which vaccines, sera, glandular extracts and so forth are made available to the public. Firstly, they may be produced by the State as at Onderstepoort, secondly by private enterprise as by Lederles & Mercks in the United States, and thirdly by non-profit-making organisations such as the S.A. Institute for Medical Research. The Connaught Medical Research Laboratories fall in the third category, and as they occupy such a unique position in the medical and veterinary worlds of North America, it may be worthwhile to say something about them. I got the opportunity to learn a good deal when I visited the Dufferin Division, where I went really to study C.R.D.

The Connaught Laboratories, which have been in operation for forty-two years, belong to the University of Toronto and are administered by the Connaught Committee of the Board of Governors of the University. The laboratories manage their own financial affairs and decide what staff to appoint and how to remunerate them. The whole organisation is strictly non-political and non-sectarian in nature.

There are four divisions of the laboratories:-

(1) The College Division on the university campus shares a magnificent building with the School of Hygiene. Liver extracts, A.C.T.H., insulin and other glandular products are prepared here, but much space is devoted to the excellent library maintained by the Connaught Committee, and there are class rooms in which doctors, dentists, nurses, veterinarians, engineers and others are instructed in public health.

(2) The Spadina Division is not far from the College Division and houses the administrative offices. Penicillin is prepared in this section and much research is conducted in the field of antibiotics.

(3) The Dufferin Division is a 145 acre farm with numerous laboratory buildings and quarters for animals, including 2,000 monkeys. It is twelve miles out of Toronto. I spent the greater part of three days there. Most of the work on human and animal viral diseases is concentrated in the Dufferin Division, and there are also first class facilities for the preparation of the various fractions of blood.

(4) The Western Division is accommodated in the Westbrook building of the University of British Columbia in Vancouver. The major problems investigated there are botulism and other types of food poisoning, and staphylococcal infections.

The total staff of all the sections is still increasing rapidly and now numbers over 600, of whom over 80 are university graduates.

The laboratories do not advertise their products or appoint sales agents, but they do issue catalogues and price lists to those who are interested. The federal and provincial governments and all the municipalities of Canada buy most of their requirements from them and so do the medical and veterinary practitioners. At the present moment they sell 61 medical and 40 veterinary preparations. They also prepare culture media for hospitals. Their well-known tissue culture medium No. 858 is exported on a large scale to the United States and elsewhere. Many of the preparations are sold in foreign countries.

About seventy research projects are in progress and many of the scientists work in collaboration with others. For instance, there is a fruitful relationship with the Ontario Veterinary College. Much of the research has no immediate practical application and no attempt, whatsoever, is made to concentrate only on projects which may bring in the dollars that are always needed.

To pay their way, the laboratories rely on what they get from the sale of their products, and on grants from the Federal Department of National Health and Welfare, the National Research Council, the National Defence Research Board, Provincial Health Departments, the National Cancer Institute of Canada, the Banting Foundation, the J. P. Bickell Foundation, the Canadian Red Cross and even outside bodies like the National Institute of Health,

Washington, and the National Foundation for Infantile Paralysis, New York. Private individuals have made big donations to facilitate expansion programmes, and so have the various Canadian governments and the Rockefeller Foundation. Whenever a real need arises, the money seems to be forthcoming.

Several staff members lecture in the School of Hygiene and this particular aspect of the activities of the laboratories seems to give all concerned much satisfaction. A fruitful and lasting relationship has been established between research, teaching and the preparation and distribution of high class biological products.

It may be worth mentioning that similar organisations have recently come into being in the University of Montreal and the University of Saskatchewan, and also in Chile. Unquestionably other countries will follow suit.

General Impressions.

As I had travelled extensively through North America and Britain early in 1952, I was most impressed by the changes that had taken place in so short a time.

Few if any will deny that the cold war is responsible directly or indirectly for the incredible speed at which things are happening. There is undoubtedly a feeling on both sides of the iron curtain that to a large extent, victory must ultimately go to those who can train to a high pitch of efficiency the greatest number of scientists. These men are needed not only to provide for us what we may not quite unequivocally call a "better way of living." They are needed for what all choose to call defence, and also for assisting undeveloped lands, where both sides are competing for the friendship and allegiance of somewhat bewildered, backward and poverty stricken peoples who, nevertheless, are quite often adept at playing off one would-be benefactor against another.

In spite of a scarcity of scientists, the veterinary schools of the English-speaking world are turning away many prospective students and not increasing their classes of about forty to fifty undergraduates, which are altogether big enough. The question often asked is whether the English-speaking world has sufficient veterinary colleges, considering the extent to which specialisation in agriculture is growing. In this section of the world are about 250,000,000 inhabitants and about thirty veterinary schools. In the U.S.S.R. we have good reason to believe there are forty to forty-five veterinary schools and between 50 and 55,000 veterinarians, not to mention about 30,000 veterinary technicians, who have been trained for three years in thirty special technical colleges. We have been told on the best authority that veterinary education in the U.S.S.R. is equal to that given in the West and that the state would not think of employing anyone but a veterinarian to manage a dairy herd of about 500 cows. But if the Russians are finding it profitable to employ veterinarians and veterinary technicians in more posts than we usually think of, we must not imagine that

the West is not moving in the same direction, if somewhat slowly, and with far fewer men. I have already stated how the large poultry breeding concerns in America employ permanently up to four or five veterinarians, and this is only one example.

In the United States only 12½% of the people are now on the land and there are surpluses of nearly all agricultural products. The tendency everywhere is for fewer and fewer people to work bigger and bigger farms, but the fewer people have to be infinitely better trained than hitherto. As South Africans, with some of the biggest agricultural problems in the world on our hands, we need to watch very carefully what is happening elsewhere.

Until recently the majority of veterinarians overseas were not very interested in animal husbandry, and any interest they did show was often resented by the nutritionists and breeders. Only now is agriculture beginning to learn what an exceedingly costly mistake was made when veterinary science and animal husbandry were developed as separate subjects. What is the use of a statistician or geneticist or nutritionist trying to draw conclusions from work on pigs infected with virus pneumonia — and over 50% of swine is diseased! — or chickens harbouring the causal organisms of C.R.D., infections bronchitis and even coccidiosis? What is the use of running public feed conversion trials with pigs, when entries are accepted from breeders with infected herds? Why should the owners of C.R.D. stock be allowed to jeopardise the chances of other entrants on national egg laying tests? How can milk yield be measured accurately when mastitis is in a dairy? It is refreshing to see that many of the younger scientists are alive to the necessity of experimenting with disease-free animals, when it comes to most work on genetics and nutrition, and the more progressive veterinarians realise that their functions extend far beyond diagnosing diseases, performing operations, and administering vaccines and medicines. Only the man who knows the healthy animal thoroughly, who has a perfect practical grasp of nutrition and breeding, can hope to understand the pathological states that confront him daily, and render worthwhile service to the farmers.

Another significant change is to be noticed. The sulpha drugs, the antibiotics and various other preparations and vaccines have been of inestimable value, but all attempts to control with them many of the most important diseases of livestock, like mastitis, virus pneumonia of pigs, ornithosis of turkeys and C.R.D. of chickens have failed. No solution to these formidable problems is in sight, other than by the establishment of socalled disease-free herds and flocks.

Veterinarians have already built up clean herds by raising piglets completely apart from their mothers. During the past twenty years poultry pathologists, of course, have been systematically establishing plants free of the diseases that concern them, such as fowl cholera, coryza, epidemic tremor and C.R.D. The Russians

have apparently gone far to solve the mastitis problem in this way, according to information received in Washington and shortly afterwards confirmed in London. It is essential to encourage in every possible manner the establishment of these disease-free farms, if any progress whatsoever is to be made in eradicating some of the major conditions still menacing the livestock industry.

As a South African poultry pathologist, I could not help being interested in synovitis, which is infectious and of growing importance to the broiler industry, and round heart disease, ruptured aortas and ruptured gastrocnemius tendons, which cannot be reproduced artificially. So far we have not encountered these last three conditions here and we do not know why.

Finally, it would be churlish not to say a word about the extreme kindness and helpfulness experienced wherever I went. Indeed, I always felt so completely at home that it was difficult to believe that I was frequently expecting strangers in busy laboratories, besides old friends and acquaintances, to contend with a barrage of questions and requests for favours that must often have tried their patience sorely. To all of them and to my colleagues who performed my duties in my absence, I am most grateful.

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HOW IMPORTANT IS "LIVER FLUKE DISEASE" IN SOUTH AFRICA

H. S. PURCHASE,

Johannesburg.

It is often prudent, salutary and of value to those interested, to assess the economic losses caused to an animal industry of a country by an "eroding" or more chronic but not always fatal disease. Distomiasis is such an infestation. It is an ever present menace where the intermediate snail hosts occur. "Liver Flukes" are known to attack many herds and flocks in South Africa.

Two approaches have been attempted in this article to gain some idea of the economic importance of this parasite.

(a) *By Questionnaires to State Veterinarians.*

With the co-operation of the Director of Veterinary Services a questionnaire was sent to all officers in charge of veterinary administrative regions in the Union, asking for estimates as to the number of sheep and cattle presently being dosed for flukes and also for estimates of animals which could with benefit be treated for this condition.

The answers have been condensed and are tabulated below:-

TABLE I

Area	NUMBER BEING TREATED FOR FLUKES		NUMBER WHICH WITH ADVANTAGE COULD BE TREATED FOR FLUKES.	
	Cattle	Sheep	Cattle	Sheep
E. Cape	900	35,000	154,400	147,600
Cape	300	50,000	5,000	250,000
Natal	—	—	94,700	64,000
O.F.S.	100,000	75,000	750,000	1,000,000
Transvaal	74,400	42,900	725,500	577.000
	175,600	202,900	1.729,600	2,038,600

From these figures it will be seen that only 1 in 10 of both cattle and sheep which are considered would with benefit be treated for Liver Flukes are in fact receiving treatment within the Union.

The latest available official stock census figures, those for 1954, show that there were 11,000,000 cattle and 33,000,000 sheep owned by Europeans, whilst Natives in that year possessed 5,000,000 cattle and the same number of sheep. From this information it appears, therefore, that not less than 10% of cattle

and 5% of sheep need treatment and it is probably justifiable to accept these figures as expressing the minimum percentage of stock exposed to fluke infestation. The true state of affairs is likely to be much worse.

To convert this information into annual monetary loss is complicated and not easy. Losses from Liver Fluke can be direct and obvious, such as deaths and abortions in females. The more insidious and less obvious damages, which are also almost always associated with other parasites, but none the less of economic importance, are loss of efficient production of meat, milk and wool. So also is reduced reproductivity due to bad condition and even severe cachexia.

Where animals are destined for meat, any interference with or persistent retardation of growth is in fact an additional financial burden for stock producers. Furthermore, many "fluky" animals also carry other worms and never achieve the finish and condition required for optimum sale value. Hence their carcasses are down graded and the producer receives less than if these animals had not suffered from helminths. The loss of livers to the consumer is dealt with later.

Taking all these detrimental factors into account, the average loss attributable to liver fluke is probably not less than ten shillings for each sheep and fifteen shillings for each bovine each year the infested animals are on the farm. This gives an annual cost to the sheep industry of over £1,000,000 and, similarly, the cattle raisers lose not less than £1,290,000. The total annual cost to this country attributable to fluke ravages is not less than £2,290,000. This figure, however unpleasantly large it may be, is in fact conservative and were a more detailed survey to be undertaken it would not be surprising if such investigation were to conclude that the losses were several times greater.

(b) *By Information from Abattoirs.*

A questionnaire was sent to the eleven largest abattoirs in the Union and of these seven replied, namely, Johannesburg, Germiston, Springs, Pretoria, Bloemfontein, Durban and Cape Town. The information received covered the period 1951 to some time in 1957.

TABLE II

Year	Cattle Killed	Livers Totally Condemned	%	Sheep Killed	Livers Totally Condemned	%
1951	507,646	28,775	5.67	654,286	12,175	1.86
1952	468,624	25,881	5.52	1,081,193	24,740	2.28
1953	512,262	23.550	4.60	1,604,313	24,393	1.52
1954	599,862	28,814	4.80	1,779,229	25,099	1.41
1955	533,582	32,420	6.10	1,920,753	36,752	1.92
1956	303,543	25,866	8.52	1,974,905	36,313	1.84
1957	279,468	19.288	6.90	1,102,586	39,921	3.62

During this period the seven abattoirs slaughtered 3,204,987 cattle and 10,117,265 sheep. Livers from 184,594 cattle and 199,393 sheep were condemned under the heading "Cirrhotic Livers." This gives a rate for total liver condemnation of 5.72% for cattle and 1.97% for sheep.

Table II clearly indicates that the average percentage of cattle livers totally condemned as "cirrhotic" is two and a half times that for sheep which is interesting when compared to the figures already given for the relative infestation rate of cattle and sheep, viz., 10% and 5%. It is also noteworthy that the annual fluctuation in the percentage figures for total liver condemnations is relatively small, except for the years 1956 (cattle) and 1957 (sheep).

Not all livers condemned as "Cirrhotic" are discarded on account of fluke infestation. The following are some of the other conditions which also come, in many abattoirs, under this generic name for statistical purposes:- *Stilesia hepatica* infestations, Abscesses, "Parasitic nodules," *Haemangiomata,* Perihepatitis with or without adhesions etc.

So far only total liver condemnations have been discussed. Meat inspectors often have to excise and condemn only part of a liver, passing the remainder for consumption. Such "partially condemned" livers do not appear under the heading "Cirrhotic."

Varying estimates can be garnered from these responsible for meat inspection services in South Africa which lead one to conclude that liver material "excised and condemned" probably equals the weights of livers which are condemned for reasons other than flukes but are recorded as "Cirrhotic." It may therefore be permissible, on a broad basis, to accept the figures for livers condemned as "Cirrhotic" as a fair approximation of total fluky liver material, both whole and parts of livers, found to be unfit for human consumption at the abattoirs.

TABLE III

Controlled Area	Average Liver Weights		Average Retail Price	
	Cattle	Sheep	Cattle	Sheep
	lbs.			
Witwatersrand	11.26	1.08	1/6 to 1/10	1/9 to 1/11
Cape Town	8.62	1.13	1/6 to 2/3	1/6 to 2/4
Durban	10.49	1.35	—	—
Port Elizabeth	10.20	1.40	1/6	1/9
Pretoria	11.12	1.12	1/6 to 2/-	1/8 to 2/-
Bloemfontein	10.75	1.01	1/3 to 1/6	1/6
Kimberley	10.41	1.04	1/3	1/6
East London	11.06	1.33	1/6	1/9
Pietermaritzburg	11.53	1.04	1/6	1/9
Average	10.60	1.17	1/6½	1/9

(Information obtained from the Meat Control Board).

The latest complete year for which records are available is 1956. Table II reveals that cattle slaughtered numbered 303,543, livers condemned 25,866 or 8.52%. The sheep slaughtered were 1,974,905, total livers condemned 36,313, giving 1.84%. Using this information together with that of Table III, then, in 1956, these seven abattoirs lost over 316,000 lbs. of livers, that is, not less than 150 tons. What the total wastage in South Africa of this valuable vitamin-rich high-grade protein food annually cannot be assessed. The yearly monetary loss to that portion of the country's meat industry served by only seven abattoirs is nearly £23,700. The total for the country must be many times more but no data are available for a firmer assessment.

In conclusion it can be said that liver fluke disease in South Africa is important on economic considerations. Monetary losses from this, admittedly only one of several parasitical problems, is not less probably than £2,200,000 a year.

With the present need for the utmost efficiency in farm animal production, the economic importance of liver flukes becomes yearly more pressing and calls for active research and enquiry as to how best to counter this parasite under local South African conditions.

Research into more modern and possibly better ways of controlling all stages of *Fasciola hepatica* and *F. gigantica* in both hosts, may take time to yield results. Hence, every effort is required to put into practice immediately the great volume of knowledge already available on this disease. This entails intelligently planned, persistent, propaganda and "extension work" by all those who are in the position to help alleviate the economic losses now eating away the stock farmer's income.

It should be emphasised, however, that "Liver Fluke Disease" is not the only major eroding disease caused by internal parasites affecting South African stock.

The author wishes to acknowledge the help received and thanks the several officers in the seven Municipalities mentioned also Dr. R. Alexander, Dr. R. M. du Toit and Dr. J. H. R. Bisschop, all of Onderstepoort. Thanks are also due to the State Veterinarians who willingly collected part of the data presented here.

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VETERINARY PRACTICE IN WESTERN GERMANY

H. KLEEBERG

Pretoria

The German Federal Republic is a little larger than Great Britain and the North of Ireland put together. It has a population of 51 million people of which 14% make a living from agriculture. Compared with farms in South Africa, German farms are extremely small. 60% have a surface-area of less than 5 hectares (about 12½ acres), 33% from 12½ to 50 acres and only 7% are over 50 acres in size. Total livestock numbers: (1956). There are about 11 million head of cattle of which 50% are dairy cows, 14½ million pigs, 1 million horses, 1,200,000 sheep and about 50 million poultry. About 19 million short tons of milk are produced yearly, which means an average of ca. 3,000 litres per cow with an average of 3.6% butterfat. Production of beef and pork amounts to about 1,100,000 short tons and 700,000 short tons respectively.

There are 7,590 registered veterinarians in the Federal Republic, 4,612 of whom are private practitioners, 136 professors and lecturers, 239 laboratory workers, 483 civil servants, 160 municipal veterinarians, 181 employees of the Farmers Coops. and 375 assistant veterinarians employed by private practitioners. A further 1,100 live in the Russian-occupied zone of Germany.

Faculties of veterinary science are found at the universities of Hanover, Giessen, München, West Berlin and Leipzig and East Berlin in the Russian zone. The Faculty buildings of five of the faculties were damaged by bombing, but subsequently have been rebuilt. At present 1,100 veterinary students are studying in Western Germany, a far greater number than is actually required.

There are 22 laboratories more or less equally distributed throughout the country. Each one is fully equipped to cope with specimens of any nature. Even the isolated practitioner is rarely more than 80 miles distant from such a laboratory. The field staff consists of 277 state veterinarians each of whom is in charge of a district. During outbreaks of infectious diseases and during special campaigns they are assisted by young assistant-veterinarians.

No vocation comparable to the local stock-inspector exists. Animal husbandry is in the hands of qualified husbandry men. Meat inspection in cities and meat factories is only conducted by veterinarians, in the country partly by veterinarians and partly by meat inspectors. 296 abattoirs have veterinarians as their directors.

In order to explain how it is possible for 4,612 private practitioners to make a living in a country as small as Western Germany, conditions in Western Germany will be compared with local conditions. Firstly, agriculture has been intensified to such a degree by modern farming methods that record crops are being reaped. That means that more animals are being kept on less ground, and all are fed very intensively. A large number remain permanently stabled. These are fed on concentrates and green food, cultivated and cut for feeding purposes only. As a result of the much increased prices of meat, eggs, milk and butter, the value of the individual animal is at present so high that the veterinarian is called in on the slightest pretext. Fees are on the whole below South African standards. The veterinarian's income in Germany is, therefore, very dependent on the number of calls he makes during the course of the day and night — Sundays and holidays included. The writer has occasionally had to visit from 20 to 30 farms a day of which about half were second or third calls, never doing more than 70 miles a day. A second important reason for the existence of so many private practitioners is the presence of a strictly enforced pharmacy act. This prevents non-veterinarians from obtaining drugs and vaccines and consequently treatment by a farmer is impossible. In the third instance farmers on the whole have a poorer knowledge of animal diseases than their South African counterparts, so that they are rarely in a position to make a diagnosis. Dogs are kept to such an extent in the cities these days, that many small animal specialists have been able to establish themselves. Furthermore, the German veterinarian is particularly concerned with the control of animal products for human consumption. Almost every practising veterinarian can rely on a definite, sometimes considerable, income from meat inspection. Within the past three to four years artificial insemination has established itself to such a degree that it constitutes an appreciable part of practice in some areas. About 20% of all cows are artificially inseminated. Until recently only veterinarians could perform artificial insemination of livestock. In recent years private practitioners have been used by the state for routine examination and treatment of large numbers of livestock e.g. the bi-annual tuberculin testing of all bovines and bleeding for brucellosis test.

The area in which a practitioner operates in has seldom more than a radius of 10 miles. Immediate visits, as well as night and Sunday visits are frequently requested and cannot be refused as in such a case another practitioner is immediately called in. There are some difficulties facing the private practitioner in Western Germany. The performance of an autopsy as a diagnostic aid is practically seldom possible. The owner will not usually permit destruction of even one animal for that purpose, nor will he call in the veterinarian to perform a post mortem on an animal already dead. By law the farmer is prevented from disposing of a carcass himself. This is done by special factories turning carcasses into

bone, meat meal and technical fats. The examination of blood-smears is of little or no value as no blood diseases occur. Often the work has to be done without any assistance at all.

Infectious diseases of importance to German livestock are tuberculosis in cattle and goats and to a lesser extent in pigs and poultry, brucellosis in cattle and sheep, foot and mouth disease, erysipelas and virus pneumonia of pigs, rabies and distemper in dogs, fowl plague, fowl pox, Newcastle disease, salmonellosis and coccidiosis in poultry. Infectious diseases occurring sporadically are equine strangles, equine encephalitis, equine infectious anaemia, leucosis in cattle and poultry, actinomycosis, swine fever, Teschen disease, vibriosis, pasteurellosis, salmonellosis in calves, leptospirosis and toxoplasmosis in dogs, tetanus and mange in cattle and sheep. Infection of cattle with Trichomonas foetus was very wide-spread for some time, but by means of slaughter of infected bulls, artificial insemination, treatment of cows, has been largely brought under control. Apart from trichomoniasis and coccidiosis no protozoan diseases of importance to the practitioner occur in Germany. The same applies to diseases caused by Clostridia — excluding tetanus. Many virus diseases occurring in South Africa are entirely unknown. Glanders, dourine, rinderpest, lungsickness and sheep pox have been eradicated completely. A number of diseases previously eradicated were reintroduced during the wars, and had to be combated anew. A large number of horses were made use of by Russian and German troops during the second World War. The German forces lost over two million horses during the war. The migration of over 10 million people, taking their belongings with them, which took place during and after the war, constituted an ideal method for dissemination of infectious diseases.

As far as tuberculosis is concerned, the farcical campaign based on slaughter of clinically diseased animals prevented any progress being made in the eradication of Tb before 1939. When the new campaign was launched in 1950, 40% to 70% of cattle were found to be reactors. This was only to be expected as spread is greatly facilitated by enforced stabling of animals for half or the whole of the year. By the close of 1956, 50% of cattle were accredited as Tb-free. The end of the campaign is already in sight. During 1953 for instance, total costs were about £20 million of which the government paid 15%. The bi-annual tuberculin testing of his cattle and goats costs the farmer nothing. Tuberculosis of pigs only plays a role in meat inspection. As about 70% of tuberculosis in pigs is due to the bovine type its decrease is understandable.

Of secondary importance only to tuberculosis is brucellosis. Unfortunately the spread of brucellosis was unwittingly facilitated during the Tb-eradication campaign, as animals sold as accredited Tb-free were not necessarily free from brucellosis. Inoculation with Strain 19 is not allowed in all provinces. The slaughter out policy is adopted in some. Stabling and close contact of animals out grazing also favours spread of brucellosis. For most shows

and auctions an official certificate stating that the animal concerned is free from C.A. and Tb. is required.

Panzootics of foot and mouth disease, starting in the west and spreading right through to the east, occurred in Europe during the years of 1911, 1920, 1926, 1938 and 1951. This rhythm is probably due to the fact that by that time a fully susceptible new generation of animals is encountered, giving the virus the opportunity of becoming very virulent. In 1951 in Western Germany, 1,300,000 cattle were affected in the first 9 months, of which 50,000 cases died. Type C was introduced from France during this panzootic and changed into Type A variation 5 in Western Germany. By the time a vaccine had been prepared the disease was already very wide-spread. Transmission was brought about by movement of stock and men, as well as by cold storage meat, feeds, straw, bags and vegetables. The total losses amounted to over £40 million. Countries having good natural barriers and making use of stamping out methods such as England, Ireland, Norway and Switzerland, were able to prevent the disease attaining epizootic proportions. In the Russian occupied part of Germany a wide barrier of immunised animals was created along the Western German border, by inoculation of a bivalent vaccine consisting of the formalin inactivated aluminium hydroxide absorbed F. and M. disease virus. When the epizootic reached this barrier the virulence of the virus decreased. The economic losses constituted only about 1/50 of that of Western Germany. Today there are again 550 farms infected in Western Germany, but 8 million cattle are immunised with the bivalent vaccine.

Annual immunisation of all pigs against erysipelas in the spring contributed handsomely to the private practitioner's income in the past. Since the advent of sulphonamides and antibiotics erysipelas is no longer a problem. A single injection of 300,000 units Penicillin is usually sufficient to cure a baconer in 12 to 24 hours. The incidence of virus pneumonia in piglets and young pigs, usually complicated by secondary infection, has increased appreciably with the modern method of housing 50 to 100 pigs in one sty. Naturally only the secondary invaders can be combated.

Rabies was always introduced sporadically from the East. However, during the severe winter of 1946/47 the Oder river froze and probably a number of rabid dogs and foxes entered the Russian zone of Germany. Rabies assumed almost epizootic proportions amongst the wild carnivores. The disease is now enzootic in Western Germany. Immunisation is prohibited.

A great part of the small animal patients seen by the private practitioner are dogs suffering from distemper. Mortality and the cases showing nervous symptoms have been decreased appreciably by immunisation and modern treatment.

Of great significance is the fact that whereas the control of infectious diseases was of primary importance during the first half of the century, sporadically occurring disease conditions such

as metabolic disturbances, deficiency diseases, intoxications, hereditary conditions and diseases resulting from poor constitutions, are now mainly encountered.

Cattle practice is concerned mainly with dairy cows and heifers in high production. They are expected to calve down yearly and a level of milk production is maintained which would have been thought to be impossible thirty years ago. This unnatural boosting of the production powers is an important factor in the aetiology of most disease conditions encountered today e.g. sterility caused either by disturbance of the oestrous cycle or specific or non-specific endometritis, mastitis, dystokia, prolapse of the uterus, retention of the placenta, different manifestations of milk fever, ketosis and paresis. The field of sterility and mastitis is too vast to elaborate on in this paper. A good experienced obstetrician is still the favourite practitioner with the farmer. The method of choice in delivery of relative or absolute oversize of the foetus is embryotomy. With a modern embryotome, a good wire saw, oblique sectioning and 3-4 assistants, it is possible to perform total embryotomy in 30 minutes. Manual removal of retained placenta is often necessary. For the treatment of frequently occurring hypocalcaemic conditions many practitioners still prefer pure calcium chlorate and magnesium chlorate as it is much more effective and works more quickly than borogluconate. As far as the digestive tract is concerned, oesophageal obstruction, rumen stasis, bloat and traumatic reticulitis frequently occur. Infection with Fasciola hepatica is very common in the North as result of frequent inundation of grazing by water.

Losses through calf diseases have notably decreased recently. These comprise navel-ill, white scours, calf diphtheria, pneumonia, salmonellosis and avitaminosis. Good polyvalent vaccines are available for the prevention of diseases of new-born calves. Calves are never allowed to suck, but receive measured quantities of milk and rations. Separated sour milk is often fed. For slaughter purposes, calves are kept till they obtain a weight of 200 to 400 lbs.

Twenty years ago little attention was paid to pigs apart from the control of infectious diseases. Recently, however, treatment of pigs has become an important source of income to the private practitioner. Pork and sausages are the most popular meat products. Pig farming can show handsome profits if approached in the right manner. 25% of the farmers income comes out of pig farming. Some of the feed is imported. All the diseases of bad management or feeding are encountered in the young pig e.g. deficiencies of vitamins A, B, D, E, mineral salts, trace elements and protein. Lack of fresh air, sunshine and green food and feeding protein in excess play a part. These also all reduce the resistance to lung and intestinal infections. Sudden heart failure in baconers causes severe losses in some areas. The cause has not yet been determined but it very probably stands in relation to deficiency of protein, excess of carbohydrates, unnatural housing and to the very marked stimulation of growth by modern feeding. Pigs are expected to

attain a weight of 240 lbs. by the time they are 6½ months old. In sows we find uterine inertia, dystokia, endometritis, agalactia and eclampsia. Breeders are aiming at 30 piglets per sow in 2 years. Many private practitioners are now doing the castration of both sexes of piglets.

It is interesting to note that veterinary schools in Germany owe their existence to a considerable extent to the army horse. Four of these schools are 160 years old, as it was then the war lords demanded military veterinarians. During the second World War equine surgery probably reached its highest peak. Now horse practice is dwindling continually. Farmers will not allow long drawn-out treatment, as horse meat butchers pay up to £80 each for heavy horses. Lamenesses, colic, phlegmosis, dermatitis verrucosa, azoturia, verminosis and broken wind are the most common ailments. Some practitioners prefer doing castrations in the standing position, as only a single assistant holding the horse is required. The operation is quick, fairly painless and makes a good impression on the owner.

Immediately after the war during the starvation period goats were kept extensively, but at the present moment they play virtually no part in private practice. The same can be said about sheep. Shepherds are rather distrustful of veterinarians, and prefer to treat cases themselves. State veterinarians combat infectious diseases. The breeding of sheep is on the decrease as a result of the ever increasing intensification of agriculture. Mutton is not very popular.

Toxicology is of little importance in large animal practice as poisonous plants are few and far between and dipping is not made use of at all.

Small animal practice has increased remarkably in post war years. In spite of licences being expensive, dogs are kept in their millions. Increased interest in the breeding of most breeds took place. 1956 saw a successful world congress of dog breeders held in Dortmund. All small animals including cats are so well cared for that most become patients sooner or later, even if they are only brought for euthanasia, something which is rather frequently requested. Private veterinary hospitals are rare, but equipment of consulting rooms is modern. More than half of the dog patients are suffering from otitis externa. Nephritis, eczema and injuries and fractures as a result of motor car accidents are very common.

A word about the drug question. All drugs are manufactured by private firms and close competition has resulted in these being good yet reasonably cheap. Leading the field is the former I. G. Farben group of firms, Bayer, Hoechst and Behringwerke. Some practitioners make a nice profit out of the sale of drugs to the farmers. As a result of the large number of proprietary preparations available the art of prescription writing is being lost. Prescriptions must be written in Latin.

The regulations concerning meat inspection are very exact and complicated. Every pig has to be examined microscopically for

trichinosis, resulting in the employment of up to 25 assistants at the bigger abattoirs just for this work on slaughter days. In cases of emergency slaughter and in certain pathological conditions a bacteriological examination must be conducted. The result is telephoned through by the institutes after 24 hours. On slaughter days the abattoir veterinarians are assisted by private practitioners. Often up to 10 veterinarians are busy working at one abattoir.

In the future, preventive veterinary medicine is a task awaiting the veterinarians once infectious diseases are eradicated. Great economic losses can be averted by intensive anti-helminth campaigns. From helminth conditions, mostly subclinical, losses are estimated to constitute a third of the total livestock losses in Western Germany. An increase in the productive life expectancy, in other words a stronger constitution would be of great economic value. At present the average life span of a cow is about $7\frac{1}{2}$ years. An increase of 2 to 3 years in life expectancy would make dairying much more profitable. With better education of the farmers and development of their farms, veterinary consultation will become more and more essential. In addition to a marked increase in work for the veterinarian an increase in schematic and bureaucratic activity is noticeable. South African and German veterinarians have this in common, that their remuneration is rather poor in proportion to their contribution to the national economy.

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A MOVABLE HAYRACK FOR HORSES

G. D. SUTTON
Onderstepoort

SUMMARY

A simple, movable hayrack which prevents waste of hay by horses is described.

It was found that when hay was fed to horses in paddocks or camps considerable waste took place because so much hay was trampled underfoot. A simple, movable hayrack that could be used in paddocks or large camps to prevent this waste was required.

After some preliminary investigation, a ground level hayrack made from 1 inch galvanized iron piping and closed on the sides by strong square mesh wire netting was found satisfactory. It was made rectangular in shape 6 feet long, 4 feet wide and 2 feet 6 inches high. An upper and lower framework of piping were joined together at the four corners and in the middle of the longer side by uprights of piping. The top and bottom were both left open. The sides were closed by strong square mesh wire netting with ¾ inch openings. The rack can be handled easily by two men and transported without difficulty on a suitable vehicle.

This hayrack can be used to feed hay or green food to horses, foals, mules and donkeys and almost completely prevents waste. So far not one of these animals has stepped into the rack. They are all content to stand outside the rack and feed quietly. The racks have been in use for a year and have been tested on some 300 equines. Cattle, on the contrary, almost immediately climb inside the rack and soon break it. This rack has thus not been successful for cattle.

TUBERCULOUS MASTITIS OF DAIRY CATTLE, WITH SPECIAL REFERENCE TO THE JOHANNESBURG MILK SUPPLY.

P. J. MEARA, M. M. GREATHEAD, and J. H. HUYSER
Johannesburg

INTRODUCTION

Veterinary control of milk supplies to the City essentially concerns those animal diseases which are transmissible to man through the agency of milk. Tuberculosis is probably the most important of these diseases, and in order to protect the milk consuming public against this infection milk samples from all herds supplying the City of Johannesburg are regularly tested. This paper describes the experience required, and also other difficulties encountered while performing this service.

No legislation exists whereby farmers can be forced to maintain tuberculosis-free herds, and no active national campaign directed at eradicating bovine tuberculosis is in progress. Thus in order to safeguard public health, the Municipal Veterinarians with the assistance of the dairy inspectors of the City Health Department aim at testing each supplier's milk quarterly, and eliminating immediately any tubercular contamination which may be discovered. Pasteurisation is regarded as a necessary additional safeguard, but primarily it is considered that milk should be derived from disease-free cows, and that the process of pasteurisation should not be used as a cloak for the production of dirty or diseased milk.

THE BIOLOGICAL TEST

The biological test is one of the main control measures adopted and in the test approximately 40 ccs of a herd milk sample are centrifugalized at 3,000 revolutions per minute for at least 30 minutes The supernatant fluid is then discarded and the centrifugalized sediment is mixed with 0.5 c.c. of gravity cream from the herd milk sample for intramuscular injection into the hind leg of a guinea-pig. Four to six weeks after inoculation the guinea-pig is killed, a post mortem examination performed and a search made for lesions of tuberculosis in the muscular and regional lymph nodes. A diagnosis of tuberculosis is established only when the presence of typical acid-fast bacilli in the lesions has been determined by examining smears stained by the Ziehl-Nielsen method.

Especially during summer, and in milk coming from a considerable distance, partial or total souring of the milk impedes the performance of the test. In extreme cases centrifugalization is not possible, but even when souring of the milk is slight the high bacterial content often causes the death of the guinea-pig in a short while and necessitates retesting another sample.

HERD INVESTIGATION

In the event of a positive test the state of the respective dairy herd is immediately investigated. All cows in milk at the time of sampling are stabled for examination and each animal is marked distinctively with chemical branding fluid for purposes of identification. The herd is numbered serially from one upwards, and depending on the size of the stable, total number of cows, and number of milkers, the animals are grouped in blocks of convenient size.

A sample of milk is taken from each cow, from her pailful of milk, for bulking with the other samples in that respective block. Each number is checked to ensure no cow is missed and the buckets are rinsed three times in between cows to prevent carrying over contaminated milk from one block to another. The milkers also wash their hands between cows as an additional precaution.

After the cows in the stable have been milked each cow's udder and the associated lymph nodes are carefully palpated for abnormality, and a "stripping" sample is taken from any suspected quarter.

Weir and Barbour (1950) describe a routine method of examination which basically we follow, although we find it easier to hobble the cow and examine the udder as recommended, in a squatting position. Each quarter is carefully palpated for induration and abnormally, and abnormal udders are recorded for future reference even although sampling may not be deemed necessary.

The investigation is continued in the laboratory by preparing block samples for biological testing as already described, except that duplicate guinea-pigs are employed for each sample, to overcome the difficulty of occasional guineapigs dying under test. Microscopic examination of quarter samples for acid-fast bacteria typical of M. tuberculosis is also carried out, by examining sediment smears derived by centrifugalization at 2,500 revolutions per minute for two minutes, and stained by the Ziehl-Nielsen method.

After identifying the affected cow(s) the farm is revisited and the owner's consent to destroy the animal obtained. The animal is slaughtered and the presence of tubercular mastitis established at a post-mortem examination and the block containing the affected cow is resampled in respect of the remaining members of this block.

Certain practical difficulties which complicate the task of investigation may be mentioned at this stage:-

(i) The antagonism of a farmer must be overcome and his co-operation obtained in the work to be done.

(ii) The investigation of an outlying herd is hampered by the great distance from the laboratory and the many trips required back and forth run up considerable mileage e.g. one affected herd in the Christiana District was 230 miles from Johannesburg.

(iii) The changing composition of a herd during the period of 4-6 weeks intervening between taking a herd milk sample and completing the biological test complicates investigation. Cows may have gone out of milk, or been sold or died.

(iv) The examination of a herd upsets normal milking operations. Milking is delayed, the cows give less milk and are less easy to manage.

(v) Water may not be plentiful. A deficiency of water in the stable complicates the rinsing of buckets and other hygienic precautions.

(vi) Mastitis may be prevalent in the herd. This obscures the examination of the udder and complicates the diagnosis.

(vii) A deficiency of milkers unduly prolongs investigation, whereas too many milkers in a large stable are difficult to control.

(viii) To cause as little disruption as possible to the farmer and to the milk depot he supplies, the investigation should be speedily performed. This applies not only to speedy milking so as to enable delivery of the milk at the usual hour, but also in respect of a speedy elimination from the herd of the source of tubercular contamination in the interests of public safety.

GENERAL PATHOLOGY

Tuberculosis of the mammary gland is usually of a chronic productive type (Gaiger and Davies, 1946). The lesion is a diffuse interstitial inflammation in which the outstanding change is an increase of cellular interacinous tissue consisting mostly of plasma and epithelioid cells. To the naked eye the tissue is a yellowish brown colour and is solid and firm — often easily differentiated from the normal. White or yellowish spots denote the onset of necrosis or caseation. In the healthy udder the tissue has a greyish white silky appearance macroscopically, with little or no discernible connective tissue between contiguous acini microscopically.

According to Gaiger and Davies usually only one quarter, or a portion of it, is affected, most often a posterior. The disease develops slowly, the gland becoming enlarged and indurated but remaining painless, the suprammary glands sometimes being affected. These findings have mostly been substantiated by our examinations but usually more than one quarter and often the forequarters are affected. Sometimes the disease develops with considerable rapidity. The supramammary glands are usually en-

larged and oedematous, though macroscopic caseous nodules are often absent. Weir and Barbour (1950) reported that tuberculosis is not uncommon in the forequarters.

DIAGNOSTIC PROCEDURE

1. *Clinical Investigation.*

(a) The general appearance of the cow is observed, — condition, respiration, cough and habitus. Superficial lymph nodes are palpated for enlargement and suppuration is noted. The udder is examined visually for symmetry, hypertrophy, and atrophy.

(b) The history of suspicious animals is investigated by questioning the farmer, the manager or foreman native. Special note is made of animals with enlarged udders which fail to respond to antibiotic treatment.

(c) After milking, the udder of each cow is palpated antero-posteriorly in systematic manner. Special attention is paid to the consistence of the tissue, the shape of each quarter, and the presence or absence of pain.

(d) A typical lesion is painless and the affected tissue has a hard stony consistence, irregular shape, discrete edges and may be only a few cms. in size or involving the entire quarter, possibly affecting also the remainder of the udder. More recent lesions are not so hard in consistence and less well defined. The lesions may be in any part of the quarter, usually in a posterior one but often in more than one quarter of the udder. In the advanced case the tissue surrounding the teat sinus is hard and the sinus feels like an inverted crater with sharp well-defined edges, and the quarter is considerably larger than normal.

(e) The supramammary lymph nodes are palpated and attention is directed to size, shape and consistence.

2. *Examination of Milk.*

It is best to examine *strippings* from suspicious quarters. Usually normal in colour, in advanced cases the secretion may be yellowish. It may be watery or contain flocculi, and on standing a deposit may settle.

Centrifugalizing the milk usually yields a creamy sediment, perhaps yellowish in colour, and microscopical investigation of this sediment often discloses cell groups which are closely associated with tubercle bacilli (Torrance, 1922). These cell groups consist of clusters of epithelioid cells scattered throughout the sediment. They should first be picked out under a low power and then examined for acid fast bacilli under oil immersion. Tubercular cell groups in a sediment smear offer a speedy means of detecting M. tuberculosis, but unfortunately they are absent in many instances and recourse must be made to searching laboriously the

entire smear under an oil immersion objective for acid-fast bacilli. Various types of mastitis cell groups may also be detected in the sediment derived from non-tubercular udders and these cell groups may mislead the inexperienced worker (Matthews, 1931). Tubercle bacilli alone are usually found but occasionally other types of bacteria are seen in association with them.

Differential Diagnosis.

1. *Physiological variations* of the udder tissue may simulate induration. A normal increase in connective tissue is usually symmetrical, affecting all quarters simultaneously, but sometimes one individual quarter may be more fleshy than the associated ones. A slight atrophy of one quarter may induce the misleading impression that the opposite quarter is enlarged.

2. *Post-parturient oedema* occurring in normal udders may be misleading, and also makes it difficult to palpate the udder tissues.

3. *Pendulous udders* are often fleshy, containing areas of fibrosis.

4. *Mastitis* due to other causes is prevalent in dairy herds. Pain is usually evident in acute cases. In chronic cases fibrosis is of a rubbery, more nodular type, the teat sinus is obliterated by tissue and atrophy of the quarters occurs. The milk usually undergoes typical changes, and a fairly large coagulum is precipitated by centrifugalisation. This sediment is sticky and tenacious, and difficult to spread evenly over a glass slide.

5. *Abscesses* occur especially in rudimentary quarters, or perhaps postero-dorsally to the udder itself.

REVIEW OF CASES

Since 1st May, 1953 we have found tubercular contamination of the bulk of milk of 31 herds. In another case (K.B.) the positive test was made by Germiston Health Department on a common supplier's bulk milk; the block tests were completed in Johannesburg. In five herds (L.A.N.; G.O.; I.S.; P.H.W.; and J.J.M.) infection was found more than once and three herds (J.E.N.; D.P.K.; R.C.T.) have supplied infected milk prior to the above date. Two herds (G.O. and J.J.M.) were found infected twice in the same year.

Discussion.

(1) In *five* herds (G.O.; P.H.W.; M.C.P.; K.B.; C.F.N.) more than one voider was destroyed at one investigation and in two cases (P.H.W.; and K.B.) one additional cow in each herd, not a voider but suffering from tubercular abscesses of the parotid and precrural lymph glands was also destroyed.

(2) In *four* of the voiders destroyed, tuberculosis lesions could only be found in the udder.

(3) In *one* case (J.Z.K.) the voiding cow had been sold before the herd examination was made. The block test was negative and a cow was traced to Rustenburg Abattoir where it had been condemned for generalised tuberculosis.

(4) In *one* case (G.J.Y.) the affected cow had died a week before the herd examination but the carcase could be exhumed to reveal a tubercular mastitis.

(5) In *eight* cases the bulk sample, from which M. tuberculosis was isolated, came from more than 100 gallons of milk; in *two* cases more than 200 gallons. This demonstrates the degree to which tuberculous milk may be diluted and lesions still develop in the guinea-pig. It also demonstrates the degree to which raw milk may be contaminated by one voider.

(6) In *one* herd (G.O.) investigations regarding a contaminated milk supply were in progress for 112 days of one year, and in another (M.C.P.) for 100 days. The milk supplies were restricted for these periods.

(7) *One* herd (P.H.W.) was first infected on 14th March, 1955, and three cows were destroyed. Subsequently on 5th April, 1957 the owner requested that two cows be examined for tubercular mastitis. Both were positive microscopically and were destroyed, revealing a very severe tubercular mastitis. A subsequent herd test showed two stables infected; on further examination two more voiders (No. 146 and No. 224) were isolated and destroyed. The block tests revealed two blocks contaminated. Further tests were then performed on the cows in the respective blocks and one more cow (No. 82) destroyed. Investigations are still in progress. In all 8 cows have been destroyed on this farm.

(8) *Eleven* of the infected herds have been dispersed after being found positive with no restriction on the sale of the animals whatsoever. The animals have gone into herds throughout the Transvaal.

(9) In all, 47 cows have been destroyed from 32 herds. In two other herds (not mentioned) two cows were found and slaughtered after individual tests had been performed on their milk, before contamination could be traced in the bulked herd milk. Both animals had recently been purchased on dispersal sales.

(10) *Fifteen* of the 32 herds were located in the Vereeniging district.

(11) In *one* case (De. L. Co.) a positive block test was obtained after a voider had been destroyed. Although subsequent individual biological cow tests proved negative, the respective cows were slaughtered, three being condemned for generalised tuberculosis.

(12) In *eight* positive tests the herd milk supply was also contaminated with mastitis and in *five* tests milk also contained viable Brucella organisms. Half of the herds had a positive mastitis record of more than 25 per cent. (See Table II). This seems to indicate that either the owners or managers of the respective herds are not particular in the care of their cows' udders or that tuberculosis of the udder develops more easily in animals from herds having a high incidence of mastitis.

Herds shown with a total of 12 mastitis tests or less had either been dispersed or had stopped supplying milk to the Johannesburg area. The records of P.L.B. and G. & C. are remarkable. In 36 tests the milk of the former showed no mastitis contamination; in the latter, though only 19 tests were performed, 500 cows being milked daily, clinical cases of mastitis occur frequently among them.

RECORDS OF COWS DESTROYED

In 14 herds detailed notes were made of 22 animals destroyed.

Discussion.

(1) *Breed.* 19 animals were of the Friesland Breed (the great majority of dairy cattle are Frieslands).

(2) *Origin of the Animals.*
Seven came from the Cape.
Four animals were owner bred.
Five animals were of unknown origin.
One animal was bought on Newtown Market.

It appears that a fairly high percentage of voiders have originated from the Cape.

(3) Nine cows were in good condition, ten in fair condition and only three in poor condition.

(4) One animal was a first calver (four years old) but most were considerably older.

(5) Udder lesions.

In 18 cows these were essentially in a hindquarter, but one of these animals also showed lesions in one or both fore quarters; three showed lesions only in the udder and not in the carcase. All lesions appeared as firm to hard, fibrous, irregular, painless masses varying from a few centimetres in diameter to more than a whole quarter of the udder, when palpated. They were often close to or surrounding the teat sinus. On section typical microscopic tubercles were found.

In two cows only was it difficult to demonstrate tuberculosis in the udder on post mortem examination due to the slight infection, e.g. one cow only showed several pinpoint lesions in the whole affected quarter.

(6) Other lesions.

Twelve coses showed marked peritonitis and pleuritis and involvement of the epicardium and myocardium.

In ten cases either mediastinal or bronchial lymph glands were infected. In two cases oedema of the skin of the udder was noticed but both animals had recently calved.

(7) The supramammary lymph glands were usually prominent, enlarged, oedematous or hyperaemic, though caseous lesions were not always present.

THE EFFECT OF SYSTEMATIC BIOLOGICAL TESTING OF THE CITY MILK SUPPLY

A gradual reduction in the amount of tubercle-contaminated milk is being effected by the policy of regular biological testing of samples from the producers who send milk into the City. Pullinger (1942) records that 2.5 per cent of samples were affected, and in 1950 Meara found only 1.0 per cent of tubercular herd milk supplies, whereas for the year ending June 1957 only 0.6 per cent of positive tests were observed (Abattoir & Livestock Market Department, Annual Report 1956-1957 (in the press).

This does not necessarily indicate a reduction in the incidence of bovine tuberculosis amongst dairy herds supplying the City with milk. The difficulties and hardships suffered by each dairyman found to be supplying tubercular milk are immediately publicised amongst the dairying community. This propaganda stimulates farmers to take precautions to avoid incurring a similar fate.

Futhermore the regular visits by the field veterinarians to dairy farms, and the ensuing discussion with the farmer concerning tuberculosis have the effect of making dairymen generally tuberculosis conscious. Of their own accord farmers tend to present animals with suspicious udders for inspection, and to submit milk samples to the laboratory for testing, so that animals voiding tubercle bacilli are removed from the herd before a herd milk test can react positively.

CONSIDERATIONS OF POLICY

(i) *Destruction of Voider.*

The City Health Department requires that a cow suffering from tubercular mastitis shall be removed forthwith from the herd and destroyed. Failure to do this entails the immediate cancellation of the farmer's permit to introduce milk into the City.

A stoppage of his milk supply causes a severe monetary loss to the farmer, more especially in the case of large herds producing many hundreds of gallons of milk daily.

It is understandable therefore that in most instances the farmer is glad to destroy the cow, even though he receives no monetary compensation whatsoever. In two cases animals were destroyed

which had just been purchased for £95 and £65 respectively, and in another instance an offer of £45 had been received only a few days prior to destruction.

(ii) *Compulsory Pasteurisation of Milk.*

After a voider has been destroyed the herd milk supply is allowed into the City, subject to compulsory pasteurisation as a safeguard in case other voiders are present which were not detected by herd investigation. The evidence reviewed above revealed one voider only in ±80 per cent of herds investigated, but it is nevertheless impossible for the veterinarian conducting the investigation to guarantee that he has removed the entire source of tubercular contamination and that the milk supply is completely safe for purposes of human consumption.

(iii) *Negative Herd Investigations.*

In isolated instances no voider can be demonstrated by clinical and microscopical means and various reasons can be advanced to explain this failure, e.g. an intermittent secretion of tubercle bacilli (Francis, 1947). Sometimes a farmer can provide reasonable evidence of the removal from his herd of a palpably infected cow. For instance, the carcase of a cow removed to a slaughter house subsequent to the taking of the original positive herd milk sample may have been condemned for generalised tuberculosis. In view of extenuating circumstances of this nature, considered too in conjunction with the fact that a careful investigation of each cow in the herd by an experienced veterinarian has failed to reveal evidence of tubercular mastitis, the Medical Officer of Health may permit the herd milk to enter the City provided it is pasteurised before consumption. As soon as biological block testing of the herd reveals that there is indeed no reason for alarm this restriction is waived, and a free herd milk supply is again permitted.

(iv) *Pasteurisation Expences.*

In the event of compulsory pasteurisation, the farmer who normally supplies a pasteurising depot is subjected to no additional expense, but the unfortunate farmer who supplies a raw milk depot will incur great inconvenience and expense. He may find it very difficult to find a pasteurising depot which will process his milk. This is very understandable because any information reaching the public that a plant is handling tubercular milk will certainly affect adversely its sale of milk, and also provide anti-pasteurisation cranks with propaganda useful for their purpose.

Should he find a plant which will accept his milk for pasteurisation a fee varying from 2d. to 4½d. per gallon may be charged,

and his charges over a period of six weeks are considerable. Largely on this account the Fresh Milk Producers Association has evolved an insurance scheme whereby members incurring expense on account of milk stoppage are compensated for as much as 75 per cent of their production for the period involved.

(v) *Tuberculin Testing.*

In the absence of a national programme sponsored by the state authorities to eradicate bovine tuberculosis the tuberculin test plays a Cinderella role in dairy practice. Indeed a dairy farme. not only receives no monetary benefit for attempting to eradicate tugerculosis in his herd by this method, but he also incurs many disadvantages. Initially his expenses are considerable in paying for a herd tuberculin test and mileage charges, and subsequent to testing he is faced with the problem of disposal of reactors, often at considerable economic sacrifice. He must also replace the reactors in order to maintain an adequate output of milk.

Furthermore, because a number of his cows have reacted positively to tuberculin, the Medical Officer of Health is positively assured that tuberculosis-affected cattle are present in the herd and is unwilling to permit the introduction of milk unless it is pasteurised. Providing the farmer can ensure that the reacting cows are kept separately from the main herd and their milk not included in the herd milk supply this restriction may be waived, but it is virtually impossible for the average dairy farmer to maintain two herds, with separate stabling, feeding and milking facilities. Certainly unless an adequate scheme of compensation and bonus payment to the dairy farmer can be provided, a general acceptance of tuberculin testing by milk producers is not likely.

The position of a dairyman supplying a tubercle-contaminated herd milk is even less to be envied. Subsequent to the veterinary investigation described above, and the elimination of the responsible cows, he is fearful of a recurring contamination of his herd milk supply. Because an abnormally high proportion of reactors is usual in a herd of this nature the penalty of tuberculin testing is likely in a herd of this nature the penalty of tuberculin testing is likely to be progressively more severe than in the average "clean" dairy herd, and no assistance is offered by municipal and state authorities to ease his burden. Consequently his usual procedure is to disperse the herd as soon as possible and start afresh, and his animals are distributed to dairymen throughout the area, spreading infection far and wide. Indeed it is common experience to hear from producers of contaminated milk, that the cow responsible for the contamination was originally purchased at a herd dispersal sale, under the circumstances described.

The expense of combating bovine tuberculosis is certainly needlessly increased as a result of this vicious cycle operating in dairy herds. From a national viewpoint too, the unlimited cost

of eradicating bovine tuberculosis must be consistently increasing, due to the dispersal of cattle from infected herds in alliance too with a policy of general procrastination.

STATE ASSISTANCE

The formidable problem of eradicating bovine tuberculosis has been ably discussed by Snyman (1955), and a fresh scheme for combating the disease is presently being evolved by the state authorities (Lambrechts, 1955).

To pretend this is an easy solution of this national problem would be foolish. *Surely however, the limited municipal problem presented by tuberculous dairy herds offers food for thought, and presents a field for experiment and experience.* Active tubercular foci are discovered from month to month, yet they occasion no interest and no effort is made to prevent a dissemination of disease.

Is it not possible that intervention along certain defined lines of approach be devised, with the ultimate object of at least reducing the spread of infection from these herds e.g.

(i) Quarantine measures to prevent the removal of cattle from herds supplying tubercular milk are urgently necessa.ry. At present infected herds may be dispersed without hindrance, even immediately after discovery of infection by municipal authorities.

(ii) Tuberculin testing of affected herds, and the branding of reactors, will afford an advance over the existing procedure. The degree of infection within the herd will be established, and tubercular animals positively identified to the benefit of dairymen generally.

(iii) A generous scale of compensation in respect of reacting animals may encourage the contaminated-milk supplier to slaughter reactors in his herd.

(iv) A scheme of bonus payment to a farmer achieving accredited status may induce the dairyman to adopt positive measures for controlling bovine tuberculosis. Certainly the cost of such a scheme will be large and will show a progressive increase, but is the ultimate cost of controlling bovine tuberculosis also not increasing apace, the while no active eradication procedure is being enforced?

At the moment milk is the focal point of investigation by the municipal veterinarian. As soon as the milk is adjudged free of tubercular contamination the herd of presumably rather badly infected cows is left to its own devices, and is probably dispersed without hindrance.

It is suggested instead that an infected milk supply should serve to direct attention to a tuberculous herd, and that a more radical procedure of herd investigation should be evolved to deal

with the disease while the herd is still a freshly discovered and undispersed unit.

As the municipal veterinarian is not endowed with legislative authority he cannot initiate proceedings. For instance, as he has no power of quarantine and cannot enforce destruction of infected animals he is unable to undertake more active measures than those being practised at present. It seems desirable therefore that the state veterinary organisation should interest itself in this matter. An appeal is made to state officials to give thought to the problem and to decide whether each contaminated herd milk supply cannot be handled with a view to a progressive animal husbandry policy, as well as the immediate requirements that the milk supply be freed from tubercle bacilli.

SUMMARY

1. Introduction.
2. The biological milk test technique.
3. The procedure followed in investigating a contaminated herd milk supply, and difficulties encountered.
4. General pathology of tubercular mastitis.
5. The diagnostic procedure for examining cows and milk, and the differential diagnosis of tubercular mastitis.
6. A review of cases encountered and points of interest.
7. Records of voider cows destroyed.
8. The effect of systematic biological testing of the City's milk supply.
9. Considerations of policy: (a) Destruction of the voider; (b) Compulsory pasteurisation; (c) Negative herd investigations; (d) Pasteurisation expenses; (e) Tuberculin testing.
10. A plea for State assistance in overcoming the problems presented to municipal authorities by tuberculous dairy herds.

ACKNOWLEDGEMENT

Thanks are due to the Director of the Municipal Abattoir and Livestock Market Department, Johannesburg, for permission to present this paper and to make use of data derived from official records.

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A CASE OF APPARENT SUPERFOETATION IN THE SOW

S. W. J. VAN RENSBURG

Onderstepoort

Superfoetation, or the fertilisation of one or more ova in an animal that is already pregnant, is a rare phenomenon, but has previously been reported in the cow, sow, ewe, mare, bitch and cat (Marx and Haase 1957).

The following case presents some interesting features, and is, therefore, considered worth recording.

On September 29, 1956, a Large White pedigree sow, Pelham Sheba, farrowed a litter of 13 in Port Elizabeth district. These piglets were not weaned as is customary at the age of 8 weeks, at the end of November, but only on January 2nd, 1957.

It is a known fact that, apart from an anovulatory and infertile oestrus within the first few days after farrowing, the sow normally does not come into heat while she is suckling piglets, oestrus usually reappearing about four days after weaning. Although, however, this sow was still in full lactation, she showed weak oestrus at the beginning of December, that is when she would normally have been on heat if the piglets has been weaned at the proper time. During this period she must have ovulated and released at least three ova. The owner reports that she escaped from her pen and stole a service.

Six days after weaning — on 8th January — she was in full oestrus and was mated again.

On March 28th, she gave birth to three normal and fully developed piglets, the result of the stolen service in December, and 33 days later, on April 30th, a second litter of 13 was farrowed. Five of these were small and did not live long, but the remaining 8 were normal and survived.

The probabilities are that maturation of follicles and ovulation during the weak oestrus in December, which resulted in the birth of only three piglets, were confined to one ovary, and that three foetuses all developed in the corresponding uterine horn. The thirteen constituting the second litter were probably all contained in the other horn. This is not impossible when it is considered that the record number of piglets produced by a sow in one generation is 24. Overcrowding in the one uterine horn was probably responsible for the five weaklings in the second litter.

Thanks are due to the owner, Commander R. W. Broadhead, for supplying the information and permitting its publication.

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United States Department of Agriculture,
Washington, August VT, VTEG.

For P.M. Release, Aug. 28.

Rats Can Carry "Crooked Snout" Swine Disease, Research Shows:

Rats have been shown to be carriers of atrophic rhinitis — a serious swine disease commonly called "crooked snout," the U.S. Department of Agriculture says:

Findings by scientists outside U.S.D.A. had previously indicated that cats and rabbits were carriers of atrophic rhinitis.

In the U.S.D.A. studies, pigs contracted the disease under laboratory conditions after receiving inoculations from rats that had previously been instilled with infective material from pigs suffering from atrophic rhinitis. The inoculated pigs not only developed the disease but in some instances developed it sooner than pigs that were inoculated directly with infective material from diseased pigs, scientists of U.S.D.A.'s Agricultural Research Service found.

Atrophic rhinitis was first reported in this country in 1944. It is non-fatal but retards the growth of young pigs. Neither cause nor cure is yet definitely known. A serious threat to the swine grower, the disease may be transferred to clean herds with swine purchased for breeding purposes.

Persistent sneezing is an early, often the only, symptom. The disease may also cause nose bleeding, nasal mucous discharge, and coughing or irritation of the snout, which causes the pig to rub its nose against some solid object to seek relief. Lesions often develop in the nasal passages, and in advanced cases the snout as well as surrounding facial features may be twisted and distorted. Pneumonia sometimes occurs as a secondary complication.

In the U.S.D.A. experiments, pigs developed atrophic rhinitis 15 to 45 days after being instilled with infective material from rats that had been given the disease. Similar pigs instilled with infective material taken directly from the noses of infected pigs took 24 to 51 days to develop the disease. Two-thirds of all pigs experimentally exposed to the infection, either directly from pigs or indirectly through the rat, developed atrophic rhinitis.

This research was conducted over a 3-year period at U.S.D.A.'s Agricultural Research Center, Beltsville, Md.

For P.M. Release, Aug. 28
U.S.A.D. 2551-57.

TRADE EXHIBIT AT THE 52nd ANNUAL CONGRESS

The Editor,
S.A.V.M.A. Journal,
ONDERSTEPOORT, Tvl.

11th September, 1957.

Dear Sir,

At Mr. Holmshaw's request, we are giving you the following brief notes on the products which were featured on our stand at the recent Veterinary Exhibition held at Onderstepoort, as we understand you would like to use them in a feature on the Exhibition to appear in a forthcoming issue of the S.A.V.M.A. Journal.

In particular our stand drew attention to the following products:-

(a) *'Protegan' with penicillin* — an association of 4% dibromopropamidine isethionate with 1% sulphathiazole and 50,000 i.u. potassium penicillin in 5 c.c. arachis oil/beeswax vehicle intended for the treatment of both penicillin-sensitive and resistant forms of mastitis.

(b) *'Lenticillin'* — procaine benzyl-penicillin suspension in rubber-capped vials of 10 c.c., each c.c. containing the equivalent of 300,000 i.u. benzylpenicillin.

(c) *'Strinacin'* — an association of triple sulphonamide and streptomycin granules indicated in the treatment of calf scours, piglets enteritis and general diarrhoea of all species.

(d) *'Strypen'* — an association of benzylpenicillin and dihydrostreptomycin in tubes of 5 c.c. indicated in the treatment of staphylococcal mastitis and other forms of bovine mastitis which are refractory to treatment with penicillin alone.

(e) *'Euthatal'* — a preparation of pentobarbitone sodium for use in voluntary euthanasia.

(f) *'Zoaquin'* — a brand of di-iodohydroxyquinoline which is in the treatment of bovine coccidiosis.

(g) *'Largactil'* — chlorpromazine hydrochloride indicated in sedation of small and large animals; premedication in anaesthesia, control of painful and pruritic states, enhancement of analgesia.

(h) *'Otoryl' ear drops* — an association of 0.15% dibromopropamidine isethionate, 0.1% benzene hexachloride and 2% promethazine hydrochloride, in propylene glycol indicated in the treatment of parasitic, bacterial and fungal infections of the external auditory meatus in the dog and the cat.

BOOK REVIEW

"BREEDING PROBLEMS AND ARTIFICIAL INSEMINATION"

by S. W. J. van Rensburg

249 pages, 72 illustrations. Published by Libagric, P.O. Box 15, Pretoria. Price: 27/6 in all countries within the African Postal Union. 28/6 elsewhere.

With almost a lifetime of experience in the practical and research aspects of the subjects concerned, the author has succeeded in writing a book which, though primarily meant for the stock breeder, student in agriculture and artificial inseminator, at the same time should form a valuable work of reference to the student in veterinary science and the practitioner.

Part 1, subdivided into nine chapters, deals with the anatomy and physiology, heredity and hereditary defects, breeding methods, infertility in cattle, horses and sheep and interruption of pregnancy.

Part II, in six chapters, deals with the advantages and dangers of artificial insemination, the instruments used, the selection and testing of bulls, the collection of semen and its evaluation, dilution, storage and transport, insemination technique in different species of domestic animals, and legislation in connection with A.I. in South Africa.

The latest information on the subjects concerned is given in a very clear and concise manner and this, together with the number of photographs and illustrations, renders the work of interest to both the laymen and the sterility worker.

The book was originally written in Afrikaans and the slightly shorter Afrikaans edition "Teelprobleme en Kunsmatige Inseminering," is available at 25/-.

M. d. L.

SUPPLEMENT TO A "WORLD DICTIONARY OF BREEDS, TYPES AND VARIETIES OF LIVESTOCKS."

by I. L. Mason.

For 1951, the Commonwealth Bureau of Animal Breeding and Genetics published a "World dictionary of breeds, types and varieties of Livestock," compiled primarily by I. L. Mason, the then Assistant Director of the Bureau.

The object of the dictionary was:-

(a) To list alphabetically the names of livestock which may be encountered in the literature, to indicate synonyms, recommend one form for English use, and to give for each breed, type or variety, briefly the origin of the name, the place of origin, present distribution, breed characteristics, economic use, and relationships to other breeds.

(b) To group the breeds under continents and countries and give them approximate numbers.

Since 1951, the dictionary has been checked by co-workers in many countries and the results, edited by Mason who is now on the Scientific Staff of the Agricultural Research Council, have now been published in the form of a supplement to the dictionary.

This supplement contains new breeds, additional synonyms to breeds previously noted, descriptions of breeds formally included but not described, corrections and deletions. These bring Part I of the dictionary up to date. Part II is brought up to date in the supplement by livestock statistics for 9 countries up to 1953.

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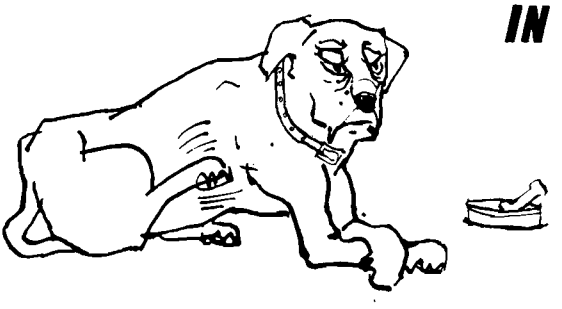

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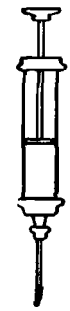

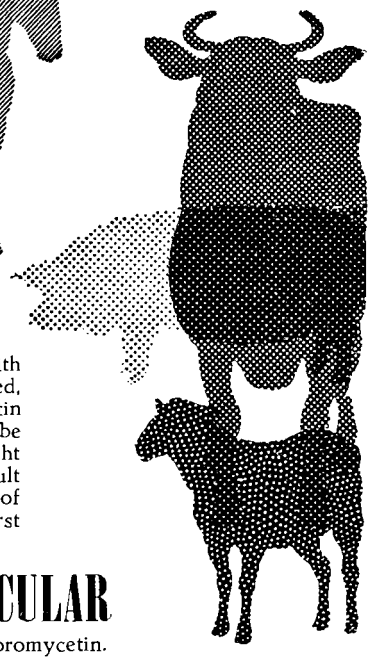

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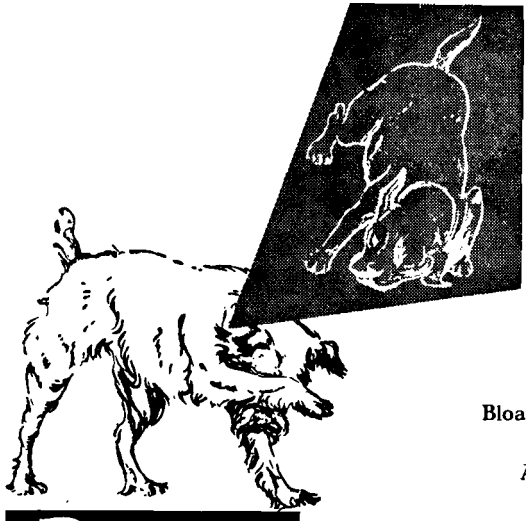

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DIE SUID-AFRIKAANSE VETERINÊR-MEDIESE VERENIGING

NOTULE VAN DIE SPESIALE VERGADERING VAN DIE RAAD, GEHOU IN DIE VLEISRAADGEBOU, OP VRYDAG DIE 6de SEPTEMBER, 1957

Teenwoordig: Dr. H. P. Steyn (Vise-president), Drs. G. D. Sutton, S. W. J. van Rensburg, E. M. Robinson, R. du Toit, J. W. Pols, M. W. Henning, R. Clark, C. F. B. Hofmeyr, L. v. d. Heever, S. van Heerden.
Apologie vir Afwesigheid: Drs. P. S. Snyman en R. Alexander.
Teenwoordig op Uitnodiging: Drs. H. Graf, W. J. Wheeler en M. C. Robinson.

Die Voorsitter het Drs. Pols en Henning welkom geheet en gelukgewens met hulle verkiesing as Raadslede.

1. INDIENSNEMING VAN VEEARTSE DEUR „S.P.C.A." KAAPSTAD.

Die Voorsitter het meegedeel dat dié saak na 'n onderkomitee verwys is, wat gedurende Kongres ontmoet en die hele aangeleentheid bespreek het.

Op voorstel van dr. R. du Toit, gesekondeer deur dr. van Rensburg, is dit besluit om die rapport, soos voorgelê te aanvaar. Dit is ook besluit dat die gefedereerde Raad van die S.P.C.A.'s, sowel as die Kaap Wes Tak, in kennis gestel word van hierdie besluit.

2. INDIENSNEMING VAN VEEARTSE DEUR KOOPERATIEWE VERENIGINGS.

Die brief van die S.A.L.U. gedateer 11.7.1957, aan die D.V.D. (veld) is voorgelees deur die Voorsitter. 'n Brief van dr. P. N. Collier, gedateer 21.8.1957, sowel as 'n konsep brief aan die Bestuurder van die Senekal Kooperatiewe Landbou Maatskappy Beperk, is voorgelees.

Na bespreking is besluit dat:-

(a) 'n skrywe gerig word aan die S.A.L.U. waarin gevra word dat 'n afvaardiging van die S.A.L.U. 'n afvaardiging van die S.A.V.M.V., bestaande uit drs. Diesel, Kirkpatrick, van Rensburg, Steyn en van Heerden ontmoet, sodat die beleid van die Vereniging duidelik gestel en bespreek kan word, om enige onduidelikheid uit die weg te ruim.

(b) die konsep brief afgesend kan word aan die Senekal Kooperatiewe Landbou Maatskappy Beperk.

(c) Dr. Collier se aandag gevestig word op die finale verslag van die Komitee van Ondersoek (Sameroeper dr. Diesel). 'n Afskrif van die verslag is aan alle lede gestuur voor die algemene vergadering in Augustus;

(d) 'n Uittreksel van die verslag van dr. Diesel se komitee aan alle persone wie getuienis, voor die komitee, afgelê het, gestuur word. Die persone sal dan ook gevra word, om, indien hulle sake voortspruitend uit die besluite van die Vereniging verder wil bespreek, die Ere-Sekretaris te nader, om 'n afspraak te maak om verteenwoordigers van die Raad te ontmoet.

3. AANSTELLING VAN STAANDE KOMITEES VAN DIE RAAD.

Die Komitees word as volg saamgestel:-

Redaksie: Dr. E. M. Robinson (Redakteur).
Drs. Clark, de Boom, Malherbe.

Finansies: Dr. van Rensburg (Sameroeper).
Drs. Alexander, Lambrecht.

Biblioteek: Dr. Clark (Sameroeper).
Drs. T. Adelaar, R. du Toit.

Algemene Sake: Dr. Steyn (Sameroeper).
Drs. Clark, Hofmeyr, R. du Toit en Wheeler.

Die Ere-sekretaris het daarop gewys dat die Vise-President die sameroeper van hierdie komitee, wat in praktyk die uitvoerende komitee is, behoort te wees. Die beginsel is deur die Raad bekragtig.

Boek fonds: Dr. G. D. Sutton (Sameroeper).
Drs. Malherbe en Pols.

Resolusies: Die Redaksiekomitee sal ook dié werksaamhede oorneem.

Op versoek van die Ere-sekretaris, dat 'n Kongreskomitee ook benoem word, is besluit dat die Algemenesake en Redaksie komitees, die Kongreskomitee sal wees. Hierdie komitee sal mag koopteer en dr. Steyn sal die sameroeper wees.

Fooie Beoordeling: Dit is ook besluit, om aan 'n suggestie wat gedurende die afgelope jaar in 'n Raadsvergadering bespreek is, uitvoering te gee, deur die aanstelling van so'n komitee.

Dr. Steyn (sameroeper).
Drs. Diesel, Hofmeyr en Pols.

3. KORRESPONDENSIE.

'n Brief van Gelukwensing met die 1957 Kongres, en goeie wense vanaf die Vereniging van Veeartse van Wes-Duitsland word gelees.

Besluit om ontvangs te erken en goeie wense vanaf S.A.V.M.V. oor te dra.

4. ALGEMEEN.

(i) Die Ere-sekretaris het die opinie van die Raad verlang of dit raadsaam en/of die gewens sou wees, aldan nie, om vasgestelde skale van besoldiging vir lokums en assistente neer te lê. Die Raad het egter gevoel dat dit nie in beginsel 'n skaal of skale kon goedkeur nie. Die Ere-sekretaris is verder versoek om nie advertensies van hierdie aard te sirkuleer onder lede van die Vereniging nie, tensy die besoldiging (en ander voordele) in die kennisgewing gemeld word nie.

(ii) Dr. van Rensburg het verlof gevra, om as gekose lid van die Raad te bedank, aangesien hy nou reeds sitting op die Raad sou hê as gevolg van sy verkiesing as Ere Lewens-Visepresident, sodoende sou hy plek op die Raad kan maak vir 'n ander gekose lid.

'n Mosie van dank aan dr. van Rensburg is aanvaar.

(iii) Dr. v. d. Heever oorhandig aan elke Raadslid 'n afskrif van 'n memorandum wat gaan oor die aanstelling van 'n direkteur van die streeksabattoir te Vereeniging.

Op versoek van die Voorsitter, lees dr. v. d. Heever die memorandum voor aan die vergadering.

Na bespreking, is besluit om 'n komitee bestaande uit drs. Steyn, v. d. Heever, M. C. Robinson te benoem, om die saak eers te gaan bespreek met dr. Bernstein (mediese gesondheidsbeampte van Vereeniging Stadsraad). Dié komitee mag lede koöpteer.

Dit is verder besluit dat die komitee ook die saak dan verder sal ondersoek, en dan aan die Raad rapporteer.

COUNCIL MATTERS

THE SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

Onderstepoort

THE MINUTES OF THE MEETING OF COUNCIL HELD ON MONDAY, THE 12th AUGUST, 1957, IN THE MEAT BOARD BUILDING, PRETORIA.

Present: Dr. H. P. Steyn (Vice-President), Drs. G. D. Sutton, E. M. Robinson, A. C. Kirkpatrick, R. du Toit, S. W. J. van Rensburg, H. H. Curson, R. A. Alexander, R. Clark, C. F. B. Hofmeyr, L. v. d. Heever and S. van Heerden (Hon. Secretary).
Present by Invitation: Drs. W. J. Wheeler, R. A. Solomon, R. B. Osrin, H. Graf, A. A. L. Albertyn, M. M. Greathead, D. J. Louw, A. J. Louw.
Apologies: Dr. P. S. Snyman (President), Drs. M. de Lange, M. C. Robinson, A. M. Diesel.

Dr. H. P. Steyn occupied the Chair and conveyed to the meeting the apologies of the President, Dr. P. S. Snyman, for being unable to attend the Congress.

I. MATTERS ARISING FROM THE MINUTES.

(a) *Cooperative Employment of Veterinarians*:

After some discussion Dr. van Rensburg drew attention to the report of the sub-committee dated 9th July, 1957. It was agreed that if the principle of "farming out" applied in such employement, the Association could not support it.

(b) *Employment of Veterinarians by S.P.C.A. and like organisations.*

Dr. Clark proposed that progress be noted and that the matter be referred to the Annual General Meeting.

Dr. Albertyn pointed out that he had been specially sent by the Cape Western Branch to participate in this discussion and that he had a document from the Cape Western Branch to lay before Council.

It was agreed to appoint a sub-committee of Drs. Steyn (Convenor), Hofmeyr, Pols, Albertyn and Flight to go into the Cape Town S.P.C.A.'s problem and to report to the Council again.

(c) *Schedule of Fees.*

Every member of Council present had been given a copy of the Schedule at the meeting. The preamble was read at the meeting.

Dr. D. J. Louw brought up the question of fees for treatment of animals of non-Europeans and natives. If fees below those stated in the schedule are charged to this group, it could not be interpreted as undercutting. This was generally conceded.

Progress reported — Agreed.

II. MEMBERSHIP.

New Members: Application for membership had been received from the following:-

M. Keep M.R.C.V.S., (Mrs.) P. J. Keep M.R.C.V.S., R. Sachs, J. D. Coetzee, P. S. K. Mason, G. M. H. Shires, P. V. A. Davies, I. Bacher, A. L. de Jager, G. E. Thompson, O. H. J. Mehnert, H. Kleeberg, E. MacNab, M.R.C.V.S., A. M. Lubbe, C. M. Breytenbach, W. Leibenguth, A. J. J. Maree, S. B. Friedman — Agreed.

III. RESOLUTIONS TAKEN AT ANNUAL GENERAL MEETINGS OF BRANCHES.

(i) *Orange Free State, Basutoland and Northern Cape Branch.*

(a) re: Supply of hormones to inseminators etc.
The Chairman stated that he had spoken to Dr. A. B. le Grange, as the latter was probably aware of the practice. Council agreed that a letter be addressed to Dr. la Grange asking him for his comments.

(b) re Policy of Council in regard to Faculty posts.
Agreed that a letter of appreciation be directed to the Hon. Secretary of this branch, for the support indicated in this resolution.

(ii) *Cape Western Branch.*

(a) re: Publication of Articles in departmental publications in which reference is made to Stock Diseases, by non-veterinarians.
Dr. Alexander reported that this matter had very recently been discussed by heads of Divisions, of the Dept. of Agriculture, and it had been decided that in future all such articles would be referred to the Director of Veterinary Services for perusal and comment before publication. It was agreed that the Hon. Secretary, Cape Western Branch be advised accordingly.

(b) re: Inadequacy of fees of State Veterinarians.
Dr. Alexander informed the meeting that it was his view that state veterinarians should do a little clinical work and that the charges which were lower than those raised by private practitioners, would ensure this. If however, state veterinarians indulged excessively in this type of work, he would instruct that steps be taken departmentally to obviate the practice. It was agreed that the resolution be forwarded to the Director of Veterinary Services for his formal reply.

IV. CORRESPONDENCE.

(i) Letter from Orange Free State, Basutoland and Northern Cape Branch, re Senekal Kooperasie.
Agreed that the Manager of Senekal Kooperasie be advised of the terms and conditions which are acceptable to the S.A. Veterinary Medical Association, on which a veterinarian could be appointed.

V. GENERAL.

(i) Letter dated 1.8.1957 from British Veterinary Association read. Noted and agreed that Dr. M. Sterne will represent the S.A.V.M.A. at this Congress.

(ii) Dr. van Rensburg proposed that the General Purposes Committee should draw up a suitable brochure setting out the privileges and responsibilities of members of the S.A.V.M.A., which should be handed or posted to new members of the Association.
Dr. Clark undertook to prepare a draft and arrange for enlightenment of senior students.

(iii) Dr. Hofmeyr expressed the opinion that Council had achieved much to effect an improvement of conditions of employment in the field of veterinary education. He wondered whether Council could not do something about promotion of improvements for, and increased use of veterinarians in the field of food technology.
After some discussion, Council agreed to a suggestion by Dr. Alexander, that he (Dr. Alexander) write a letter to the Secretary for Public Health in this regard.

(iv) Dr. Clark informed Council that Mr. Coert Steynberg had donated a bust of Sir. A. Theiler to Faculty and that Faculty was considering a scheme for incorporation of the bust into a suitable "foundation" at Faculty. As funds would be required to execute the project Faculty had in mind, Dr. Clark asked whether he could put the scheme to members at the Annual General Meeting. — Agreed.

(v) The Chairman asked whether Dr. Alexander had seen the Secretary for Health in regard to the Ante and Postmortem Inspection of meat. Dr. Alexander reported that he had done so, and that he would again raise the matter with the Secretary for Health.

(vi) Dr. H. P. Steyn enquired what had transpired in regard to the resolution taken at the last Annual General Meeting, in regard to the control of the sale of poisons such as strychnine. Dr. Clark reported on action taken, and suggested that we remind both the Secretary for Health and the Secretary for Agriculture to reply to the letters written to them in this regard.

SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

PROCEEDINGS OF THE 52nd ANNUAL CONGRESS, HELD AT ONDERSTEPOORT ON THE 13th, 14th and 15th AUGUST, 1957

The following members registered at the meeting:

Drs. A. N. S. Abrey, A. A Albertyn, M. Bachmann, C. W. A. Belonje, L. C. Blomefield, J. Bosch, H. J. W. Botes, P. B. Botha, W. H. B. Buhr, F. E. Cavanagh, C. L. Craig, A. J. du Plessis, J. L. du Plessis, E. de V. Erasmus, C. H. Flight, J. Grobler, F. J. D. Hempstead, C. F. B. Hofmeyr, A. C. Kirkpatrick, F. W. Langbridge, A. J. Louw, D. J. Louw, Jac. Louw, I. S. McFarlane, W. M. McHardy, J. H. Mason, M. J. N. Meeser, L. R. Morford, J. de Jager, C. J. Muller, J. J. Oosthuizen, R. B. Osrin, J. D. Poole, M. C. Robinson, T. Robson, R. A. Solomon, L. G. Steele, J. L. Stewart, D. G. Steyn, H. P. Steyn, A. F. Tarr, H. C. Theron, A. D. Thomas, J. A. Thorburn, D. J. Thornton, D. E. Truter, P. L. Uys, W. P. van Aardt, L. W. v.d. Heever, K. v.d. Walt, J. van Niekerk, I. van Schalkwyk, D. C. L. Wacher, T. C. W. Wessels, W. J. Wheeler, T. F. Adelaar, R. Alexander, J. H. R. Bisschop, R. Bigalke, J. M. M. Brown, R. Clark, J. D. Coles, R. B. Cumming, H. P. A. de Boom, R. du Toit, P. J. Fourie, H. Graf, D. A. Haig, M. W. Henning, P. G. Howell, B. C. Jansen, D. J. le Roux, J. M. W. le Roux, J. F. W. Grosskopf, W. O. Neitz, W. D. Malherbe, J. W. Pols, E. M. Robinson, K. Schulz, J. D. Smit, A. J. Snyders, G. D. Sutton, R. C. Tustin, G. C. van Drimmelen, J. S. van Heerden, S. W. J. van Rensburg, K. E. Weiss, J. G. Boswell, J. H. Huyser, D. G. Steyn, C. J. Howell, F. J. Veldman, M. M. Greathead, S. L. Snyders, P. J. Meara, A. B. la Grange, Z. Kempster, J. F. du Plooy, P. L. Louw, H. J. J. Terblanche, E. M. Hearn, L. L. Hansmeyer, L. Abrams, P. B. Winterbach, W. du Toit Malan, P. H. le Roux, F. W. B. du Casse, I. Mowat, W. E. J. Warnes, W. A. de Waal and W. J. Ryksen.

Apologies for absence were received from: Drs. W. C. Viljoen, T. A. T. Louw, L. Muller, Capt. Dickson, O. H. J.Mehnert, G. Martinaglia, W. H. Schatz (for members of the South West Africa Branch), J. R. Frean, Vera Morford, H. P. Thorogood, R. T. Bangay, J. G. Keppel, D. Lawrence, H. C. Watson, J. E. Dorrington, C. C. Wessels, M. de Lange and P. J. du Toit.

Messages of good wishes were received from: The British Veterinary Association, The South West African Branch Dr. E. F. de Abreu, The Director of Veterinary Services Southern Rhodesia, The Veterinary Association of the German Federal Republic.

TUESDAY — August, 13th:

At 9 a.m. the Vice-President, Dr. H. P. Steyn, called the gathered members, approximately 90, to order.

After welcoming the members, visitors and delegates Dr. Steyn welcomed the Minister of Economic Affairs, Dr. A. J. van Rhijn, and asked him to address the gathering.

In opening the Congress, Dr. A. J. van Rhijn said amongst other things: "I am very interested, as Minister of Economic Affairs, in what is being done here for the welfare of man and animal. It is of great economic value. We think of some of the diseases which have been conquered, e.g. Gallamsiekte, Rinderpest, Tsetse-fly, Anthrax, Rift Valley Fever. Science is not static. One does not

require large well equipped laboratories to make contributions to the scientific achievements of a country. Power of observation is the important factor.

The development of a country is dependant upon the scientific research which is done — it is the barometer of the development of the people of a country.

The Government is very sympathetic towards research in our country. A standing commission was recently appointed to advise the Government on scientific developments. More recently this Commission advised that there was a great shortage of scientists in this country. A few scientists were sent overseas to study nuclear fission, and then to draw up a programme of work.

In wishing the Congress every success, the Minister was aware of the fact that many veterinarians were not present, because they were at their posts fighting the scourge of foot-and-mouth disease. Dr. van Rhijn trusted however that those present would have fruitful discussions and deliberations, and that these would not only inspire those present, but that the achievements would be of great benefit to our country.

With the words: "May you return to your homes, after the congress, inspired and stimulated," Dr. van Rhijn declared the Congress open.

Dr. H. P. Steyn thanked the Minister for his opening address.

Dr. Steyn conveyed the apologies of the President, Dr. P. S. Snyman. to the meeting. Dr. Snyman had undergone a serious operation and had not sufficiently recovered to enable him to attend. Dr. Snyman wished the Association a successful and fruitful Congress.

The Chairman voiced the opinion that the Hon. Secretary address a letter to Dr. Snyman, wishing him a speedy recovery and return to duty. The meeting acclaimed this suggestion.

The Vice-President (Dr. H. P. Steyn) then welcomed the guests, amongst whom were representatives of the Department of Veterinary Services in Mocambique, the Department of Public Health. the Department of Agriculture and the C.S.I.R. Amongst the guests were also colleagues from England and Kenya. The Vice-President made special mention also of the non-veterinary professional staff of Ondestepoort, and the students of the Veterinary Faculty.

Obituary: Professor M. van den Ende, who was an honoray associate member of our Association. had passed on recently. The customary token of respect was offered.

Presidential Address: Approximately 101 people were in the hall when Dr. Steyn gave the Presidential address, which had been written by Dr. Snyman.

Opening of Trade Exhibition: The representatives of the firms exhibiting at the Congress then entered the auditorium. At the end of a short address, the Vice-President declared the trade exhibition officially open. In reply Mr. Stabler, President of the Medical Exhibitors Association, voiced the appreciation also of his fellow exhibitors. In appreciation of the opportunity for showing their wares, Mr. Stabler, on behalf of their Association, handed Dr. Steyn a cheque for £100, for the Benevolent Fund of the S.A.V.M.A.

After thanking Mr. Stabler and his collegues, for this genorous and spontaneous gift, Dr. Steyn accompanied by Mr. Stabler and Mr. Oversby (Hon. Sec. of the Medical Exhibitors Association) and Dr. R. A. Alexander, did a tour of inspection of the trade exhibits.

After tea had been taken, the Vice-President asked Dr. R. J. Ortlepp to address Congress on "The danger of worm infestation in artificial pastures."

Dr. Ortlepp pointed out that as the practice of running large numbers of sheep in small paddocks of artificial pasture was rapidly increasing in the Union, the concomitant danger of gross worm infestation had also increased appreciably. The use of Phenothiazine in licks for periods of a few weeks, repeated again after a break for a few weeks, gave very satisfactory results in killing off the developing larvae in droppings.

Drs. R. du Toit and McHardy questioned the speaker on the danger of soiling the fleeces with the phenothiazine, as this might do more damage than the worms. Dr. Ortlepp felt sure that unless the sheep were very crowded and "kraaled", the danger was small, but it did exist.

Approximately 150 people were in the hall when Dr. Belonje was called upon to address the meeting. A ciné film and colour slides were shown to depict clinical and pathological changes associated with Wesselsbron disease.

Dr. K. C. Smithburn gave a most absorbing address on Rift Valley Fever, Wesselsbron Disease and Middelburg Virus Disease.

Dr. Alexander elaborated upon the results obtained in immunizing sheep (in a field trial) with combined Wesselsbron and Rift Valley Fever vaccine, as Dr. Barrie was unable to be present to present his paper.

Drs. Smit, Adelaar, McHardy and others put several questions to the speakers.

At 2 p.m. Dr. Schulz presented his paper. Apologies were made for Dr. Schutte who was unable to be present because of the Foot and Mouth disease position. Dr. Schulz pointed out that the suggested name for the condition should be Turning Sickness and not Turning Disease.

Dr. Alexander, Director of Veterinary Services, then gave a most interesting and gripping account of the present Foot and Mouth Disease position in the Union, and the steps that had been taken by the Division of Veterinary Services to counter the outbreaks. It is interesting to note that in the case of the Springs outbreak, and also in the case of the Molopo outbreak(s) poisoning by caustic soda was suspected. The virus was subsequently typed by Dr. Galloway at Pirbright (U.K.) as S.A.T.I. After several questions had been put and replied to, the Chairman thanked Dr. Alexander for enlightening the large gathering of members of the profession.

After Dr Haig and Mr. Plowright in turn had elaborated on and outlined some of the procedures in tissue culture techniques, the meeting was adjourned to a laboratory in which several microscopes had been set up. The changes in the tissue culture cells caused by different factors were demonstrated — Drs. Haig, Weiss and Mr. Plowright stood by to answer the many questions and explain further details to the large number of interested members.

WEDNESDAY — August 14th.

When Dr. Kleeberg rose to deliver his address at 8.30 a.m., approximately 65 members and 21 students were in the hall. "A striking thing," said Dr. Kleeberg, "which South African and German veterinarians have in common, is that both groups are poorly remunerated in relation to their contribution to the economy of their respective countries." A comprehensive and detailed account of veterinary practice in Western Germany was followed by many questions, which Dr. Kleeberg replied to in German. Dr. van Drimmelen agreed to translate.

The Vice-President thanked Dr. Kleeberg for a stimulating paper which led to animated discussion.

Dr. Thomas and Dr. Neitz in turn addressed the Congress on "Toxicosis in Cattle, produced by Ticks". The discussion on these very interesting and topical addresses had to be curtailed by the Chairman because of pressure of time. The Vice-President undertook to grant an opportunity later for the discussion of the papers given by Drs. Thomas and Neitz.

Dr. J. M. Brown's paper on liver function was much appreciated by the large number in the auditorium.

Pressure of time however, forced the Chairman to curtail the discussion thereof.

Dr. Malherbe's paper on "The Problem of the Dog with Skin Irritation" was of great practical significance to the large number of private practitioners who frequently have to deal with this type of problem. The Chairman's gavel, however, brought the lively discussion to a close.

Dr. C. F. B. Hofmeyr addressed the meeting on "Thoracotomy in the Dog", after which a colour ciné film of the operation was shown to the audience

At 2 p.m. Dr. H. P. Steyn welcomed Drs. du Pre le Roux and Dormer, and asked Dr. Dormer to address the Congress on "Tuberculosis in South Africa". This most instructive and interesting paper introduced the symposium on Tuberculosis, and it was followed by a number of questions to Dr. Dormer from the floor. The Chairman thanked Dr. Dormer for his very appreciable contribution to our knowledge of T B. in South Africa.

The paper by Drs. Meara and Greathead, which was read by Dr. Greathead, followed.

At 3.45 p.m. Dr. D. Osterhoff read his paper on "Blood Groups in Domesticated Animals". Because of the lateness of the hour, the latter part of the paper was rather rushed through. The very intimate association between this work and that of veterinary serology and immunology was evident from the paper, as was the importance of this field of scientific endeavour to the application of artificial insemination of animals.

After thanking Dr. Osterhoff, the Chairman adjourned the meeting at 4.30 p.m.

At 8 p.m. guests and members and their wives or partners, foregathered at the Union Hotel in Pretoria for dinner. Approximately 84 people were present, including Dr. the Hon. and Mrs. A. J. van Rhijn, Dr. and Mrs. M. S. du Toit, Dr. Valadao and several other guests.

THURSDAY, 15th August.

Dr. H. P. Steyn, Vice-President took the Chair for the business meeting.

Minutes: The Minutes of the 51st Annual General Meeting having been published in the Journal of the Association, were taken as read and their adoption moved, seconded, and agreed to.

Faculty Posts: Dr. H. P. Steyn gave a resume of what had taken place during the past few months, following on the circular to all members dated 20th February, 1957.

Dr. Graf, Dean of the Faculty of Veterinary Science was asked to address the meeting, and announced the improved salary scales of the staff of the Veterinary Faculty, which had been communicated to him telephonically by the Department of Agriculture. (Dr. M. S. du Toit, had announced at the dinner, the previous evening, that considerable improvements in the emoluments of teaching staff had been agreed upon, by the Department of Agriculture and the University of Pretoria).

The salary scales for the full-time teaching staff as well as that of the part-time teaching staff, were put before the meeting (on a black board) and served as a basis for the discussions which followed.

Dr. Alexander stressed the fact that the profession — this meeting — should consider the matter rationally, clearly and sincerely. In his opinion the majority of faculty members should be full-time lecturers, but some part-time lecturers would also be necessary. The meeting should now consider whether the salary scales offered are commensurate with the social status of the profession and an adequate recognition of its contribution to the welfare of the country.

Council cannot and had not prohibited members from applying for the advertised posts, it had merely advised them, and it was gratifying to Council to note that members had wholeheartedly supported Council's action by not applying for the posts as advertised. Council had handled the matter in the very best tradition.

Dr. Steyn pointed out that the "clinical allowance" as offered was an arbitrary figure, arrived at by someone as an assessment of the value of the profession, without consultation with the profession. Council had asked for an interview with the Minister of Agriculture, and after it had been refused, for a round-table conference with representatives of the Department of Agriculture, the Faculty of Veterinary Science and the University of Pretoria. This request had been ignored, as had Council itself, during the whole of the negotiations.

Professor Henning thanked Council for what it had done in this matter. Had Council not acted, a professer, receiving less than a lecturer at the University, would have been appointed in the Faculty. He felt, however, that notwithstanding the improved scales, lecturing staff will have to become civil servants, and that this is most unsound.

The Chairman pointed out that the "clinical allowance" for professors and senior lecturers should be the same, because both incumbents are senior men in the profession and of equal proficiency.

Dr. Graf said he wished to move a motion that the Association accept the new conditions and leave it to individual members to decide whether they apply for the vacant post(s) or not. Drs. Adelaar and Clark seconded the motion.

In further discussions some members expressed the view that this matter should be referred back to Council. A motion to this effect by Dr. Morford, seconded by Dr. Meeser, was put and defeated.

Dr. Coles suggested that this meeting should decide on Dr. Graf's motion, but with the proviso that Council draw up a suitable circular to all members, anent the decision of the Association. This was necessary, as in a previous circular Council had advised members not to apply for posts until a further circular had been received from Council.

In reply to a question, the Chairman explained that we do not accept with finality the salary scales and other conditions, but that the decision to apply or not, be left to the judgment of individual members.

The motion was put to the meeting and was accepted unanimously.

After a break for tea, the meeting resumed, and Professor Henning moved:

"Hierdie vergadering wens, weereens, sy oortuiging uit te spreek dat dit nie in die belang van die publiek, sowel as van die professie is, dat die Veterinêre Fakulteit langer onder die Departement van Landbou resorteer nie.

"Dit wens die Raad van die Universiteit van Pretoria derhalwe vriendelik te vra om die nodige stappe te neem ten einde volle beheer van die Fakulteit deur die Universiteit te verkry."

Seconded Dr. Muller.

The motion was put and adopted unanimously.

(ii) Matters arising from the minutes of the Annual General Meeting held on the 13th September, 1956.

(a) *Co-operative Employment of Veterinarians.*

Dr. Steyn referred to the original report of the sub-committee which had been available to members at the last general meeting, and was subsequently circularised to members.

A further report had been sent to branches for the information of their members, and the additional report had recently been circularised to all members.

The sub-committee had done an enormous amount of work and sacrificed much time in drawing up these reports and taking evidence, and on behalf of the Association, he thanked them for their work.

He asked Dr. Diesel, Convenor of the sub-committee, to first address the meeting and then to answer members' questions.

Dr. Clark stated that the cardinal principle aimed at is that a veterinarian will not do clinical work for a non-veterinary organisation. If the veterinarian does clinical work, the fees must go to him and not to the organisation.

After further discussion Dr. Tarr moved that the principle underlying the recommendations of the sub-committee, as circulated to members in the supplementary report, viz

"The Association is not in favour of the acceptance of any type of employment, or remuneration by Veterinarians, from Cooperative Societies, Farmers' Veterinary Syndicates, Companies, Firms, Trading Concerns or Individuals, whose principals are not Veterinarians, if, in the opinion of Council, or of a committee appointed by Council, or by the Association, the veterinarian will:—

(i) Engage himself in clinical veterinary practice in a manner which gives him an unfair material and remunerative advantage over any veterinarian practising in the area;
(ii) be required to collect fees for services rendered, other than for his own personal use;
(iii) overload the area with veterinarians;
(iv) become embarrassed in his attempts to observe all aspects of the Guide to Professional Etiquette.

Provided however that this expression of principle shall not affect any veterinarians already employed or remunerated in a manner aforesaid.

The Association wishes it to be understood however, that it is generally in favour of:—

(a) The employment or remuneration of veterinarians by the organisations referred to above, if such employment or remuneration can be arranged in any of the following ways, within the principles previously stated:—

(i) On a guaranteed minimum income basis, the guarantors being liable for the difference between the annual amount earned and the annual amount guaranteed over a fixed period, or

(ii) on a retainer basis, or

(iii) on a subsidy basis, or

(iv) on a part-time salary basis

and provided further that none of the above forms of employment or remuneration will prevent the veterinarian from practising freely in the area, and that the services to be rendered by him in respect of any of them, will be conducted from his own established practice.

(b) The collection of fees on behalf of veterinarians in any manner which may assist him, and which allows him to receive the full fee less the usual commission imposed for its collection."

be accepted and implemented in course of time.
Dr. K. van der Walt seconded the motion.

In moving an amendment, Dr. Riiksen said that the report referred to did not contain any suggestions as to how the recommendations can be enforced. (Dr. Diesel pointed out that the supplementary report sent to all members, did so very clearly.) He felt that absolute control had to be incorporated into the "Code of Ethics". He could not accept the report as he felt that the dignity of the profession was at stake and that veterinarians, employed by Cooperative Societies as recommended in the report, were in unfair competition with private practitioners. He moved that the Association disapprove of any type of employment of veterinarians by Cooperative Societies and like organisations, and reaffirm the resolution taken two years ago.

Afther further discussion Dr. Tarr's motion was adopted unanimously.

(ii) (b) *Redrafting of Constitution.*

Dr. Diesel reported that the consultant, who was a specialist in this field, had not as yet completed his draft. It was agreed that the Hon. Secretary enquire into the matter again. 8he hope was expressed that the sub-committee of Council which was dealing with this matter, would report at the next Annual General Meeting.

(ii) (c) *Draft of Directive Fees.*

Dr. Steyn as Convenor of the sub-committee of Council, reported that a draft had been submitted to and passed by Council. It would be circularised to all members to serve as a workable draft, and be discussed at the next Annual General Meeting.

(ii) (d) *Resolution re Compulsory Postgraduate Training.*

The Chairman reported that Faculty had advised that the whole question of the veterinary course was being investigated.

Dr. Graf stated that a committee had been appointed to go into the matter, and until this committee's report and recommendations were received, this matter should stand over.

No other matters arising from the Minutes were raised.

(iii) *Election of New Members.*

The Secretary stated that applications for membership had been received from the following veterinarians. Their applications had been placed before Council, and Council now recommended them to the Meeting for approval:

M. Keep, P. J. Keep, R. Sachs, P. S. K. Mason, G. M. H. Shires, P. V. A. Davies, I. Bacher, G. E. Thompson, H. Kleeberg, E. MacNab, W. Leibenguth, J. D. Coetzee, C. M. Breytenbach, L. A. de Jager, O. H. J. Mehnert, A. M. Lubbe, A. J. J. Maree and S. B. Friedman.
Unanimously approved.

(iv) *Resignations.*

Dr. J. H. N. Hobday of Rhodesia, advised that as he was retiring he would reluctantly have to resign his membership of the Association.
His resignation was accepted with regret.

(v) *Balance Sheet and Auditors Report.*

The Balance Sheet had been circularised to all members, and the Auditors' report to Council Members. Adoption moved, seconded and accepted.

(vi) *Reports by Finance and other Standing Committees.*

These reports had been circularised to all members, and their adoption was moved, seconded and agreed to.

(vii) *Constitution of Council.*

The Chairman announced the constitution of Council.

Dr. P. S. Snyman — President.

Dr. H. P. Steyn — Vice-President.

Dr. G. D. Sutton — Hon. Treasurer.

Dr. S. van Heerden — Hon. Secretary.

Dr. E. M. Robinson — Editor, Journal of the S.A.V.M.A.

Members, 1956/58 — Drs. S. W. J. van Rensburg, L. W. van den Heever, M. C. Lambrechts, Professor R. Clark.

Members, 1957/59 — Drs. R. du Toit, C. F. B. Hofmeyr, M. W. Henning J. W. Pols.

The Chairman announced to the Meeting that Council recommended appointing Drs. A. M. Diesel, S. W. J. van Rensburg and J. B. Quinlan as Hon. Life Vice-Presidents. Drs. Diesel and Van Rensburg had been on Council for many years and rendered valuable service to both the Association and the profession, and he was pleased that their services and experience would not be lost to Council.

The recommendation was carried unanimously.

Dr. Diesel thanked the meeting for the honour, and was pleased to be able to help Council and the Association.

Dr. van Rensburg associated himself with Dr. Diesel's remarks and thanked the meeting for the honour conferred on him.

(viii) *Employment of Veterinarians by S.P.C.A. and similar Societies.*

After some discussion Dr. Boswell moved, seconded by Dr. Pols, that "the recommendations of the sub-committee, as set out in its latest report on Competitive Employment of Veterinarians, be adopted, as resolved in the discussion on Employment of Veterinarians by Cooperative Societies."

Dr. Kempster enquired what the position would be of veterinarians presently employed by the Johannesburg branch of the S.P.C.A. The latter society had received an intimation, a few years ago, from the Witwatersrand Branch of the S.A.V.M.A., that fees should be collected.

The Chairman pointed out that as the practice evolved and conditions changed, the Association was entitled to amend its directives. The purpose of the inquiry was to protect the interests of veterinarians and that his (Dr. Kempster's) interests and present position would not be overlooked or jeopardised.

On being put to the meeting, Dr. Boswell's motion was adopted with one dissentient.

Dr. Albertyn then asked whether he could, at this stage, present the report of the Cape Western branch on the employment of a veterinarian by the S.P.C.A. in Cape Town. The Chairman ruled that this matter had been dealt with by Council, who should further deal with the matter and not this meeting.

Dr. Diesel, as Convenor of the sub-committee enquiring into the Employment of Veterinarians by Cooperative Societies and like organisations, requested that the resolution of the meeting, be conveyed to all persons who had given evidence before the sub-committee. Agreed that Council deal with this.

(ix) *Standardisation of Procedures at Dog Shows.*

Dr. Hofmeyr said that South Africa had many big cities which regularly held dog shows. It was desirable to have some procedure laid down for these dog shows, and arrangements made with Kennel Clubs to have this adhered to and certain facilities provided.

Dr. Diesel proposed that a sub-committee consisting of Drs. C. F. B. Hofmeyr and S. V. O'Brien go into the matter and report to Council. Agreed.

(x) *General.*

Dr. Alexander then requested permission to address the meeting.

He thanked the members for their support and assistance to the Division of Veterinary Science in the present outbreak of Foot and Mouth Disease. Not

only the staff of the division, but private practitioners in the affected areas had worked tirelessly and ceaselessly, and he appreciated this opportunity of thanking them. He appealed to members to notify the authorities immediately they suspected an animal on any farm to be affected, as early diagnosis was a great factor in fighting the outbreak.

Dr. W. A. de Waal enquired about the appointment of a veterinarian to the Senekal Kooperasie. The Chairman reported what Council had done in this matter.

Several members raised remarks in connection with the running of the Congress.

The Chairman pointed out the difficulties which had arisen, and asked members to bear with the organising Committee. The mistakes and shortcomings had been noted and every effort will be made to avoid these at future congresses.

Dr. Osrin proposed a vote of thanks to the Chairman for the efficient manner in which he had conducted the meeting. Carried unanimously.

(xi) *Resolutions.*

(1) Moved by Dr. H. J. Terblanche: "Hierdie vergadering versoek die Raad om in te gaan op die moontlikheid om privaat veeartse deeltyds te gebruik en ooreenkomstig te besoldig vir Staatswerk."

(2) Dr. van Schalkwyk expressed his concern at the Increase of Chronic Respiratory Disease in Poultry, which was assuming a threat to the country. Resolution: "We record our concern over the apparent lack of diagnostic facilities and control measures pertaining to Chronic Respiratory Disease in our poultry industry, and urge that positive action be taken to combat this serious disease."

Seconded: Dr. R. B. Cumming. Agreed.

(3) Dr. Thornton expressed the opinion that the Association should endeavour to obtain an improvement in the remuneration of state veterinarians. Council's action had resulted in an improvement in the emoluments of members of Faculty, and action by Council would achieve the same on behalf of state veterinarians. Proposes: "That the S.A. Veterinary Medical Association appoints a committee to consider and report on ways and means to bring about an improvement in the remuneration of all veterinarians employed by the State."

(4) Dr. C. F. B. Hofmeyr proposed: "Die 52ste Jaarkongres van die Suid-Afrikaanse Veterinêr-Mediese Vereniging, verteenwoordigende alle Veeartse in die Unie en Suidwes-Afrika, spreek sy vertroue uit in die Afdeling Veeartsenydiens, om die toestand geskep deur mond en klou siekte die hoof te bied.

Gesekondeer: Dr. H. Graf. Aangeneem.

(xii) *Adjournment.*

Dr. Steyn expressed his thanks and appreciation to Dr. Alexander for placing the Faculty Buildings at the disposal of the Association for the Congress, also for the assistance he had given in other directions. He thanked the contributors for their various papers and expressed his regret that it had been necessary to cut short the discussion of some of the papers because of lack of time.

The Chairman thanked the Programme Committee for the work they had done, also the Hon. Secretary and Treasurer, who had done an immense amount of work in connection with the Congress.

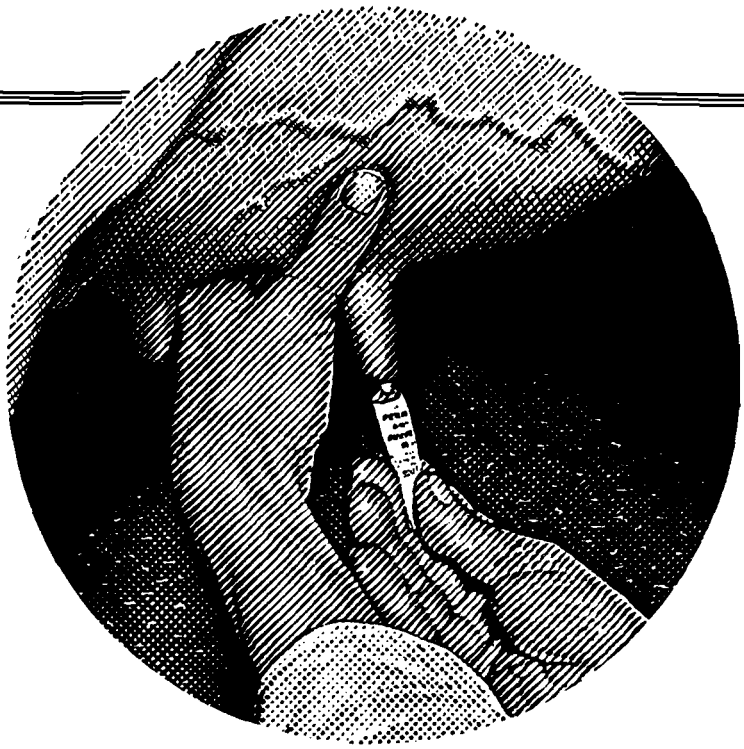
To the Ladies who had arranged the Social Functions he expressed the thanks of the meeting, which would later be "said with flowers".

The Meeting adjourned at 1.20 p m.

At 2 p.m. a number of members and guests visited the S.A. Bureau of Standards 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. A number of members and guests also gathered at the tennis courts for an afternoon of tennis.

At 6 p.m. members and guests gathered for drinks, snacks and dancing at the Pretoria Country Club. Approximately 100 people attended this function which ended at 11 p.m.



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The problem of mastitis has to be approached using drugs that give a wider coverage than does penicillin alone. The inclusion of unrelated anti-bacterials in various combinations with and without penicillin resulted in the production of the **M&B** range of Veterinary Products for the treatment of this condition. Together they cover the requirements of most cases of bovine mastitis.

Detailed information is available on request.

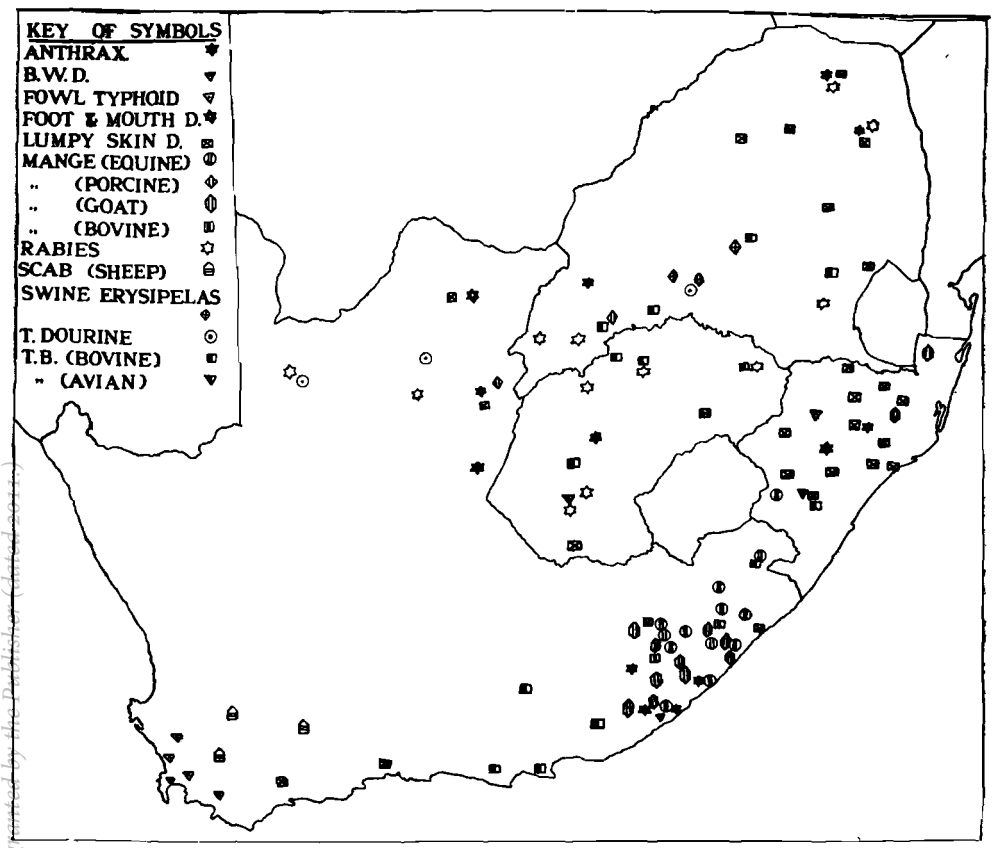
VETERINARY PRODUCTS M&B

M&B *brand Veterinary Products*

MAYBAKER (S.A.) (PTY) LTD · P.O. BOX 1130 · PORT ELIZABETH · TEL: 89011 (3 LINES)

OUTBREAKS OF SCHEDULED DISEASES IN THE UNION OF SOUTH AFRICA DURING THE PERIOD 1/9/57 to 30/11/57.

DISEASE	*CAPE*	*NATAL*	*O.F.S.*	*TRANSVAAL*
Anthrax	(1) Barkly West (1) Cathcart (3) East London (1) Herbert (1) Kentani (1) Kingwilliams-town	(1) Babanango (1) Msinga	(1) Brandfort	(1) Letaba (2) Lichtenburg (1) Zoutpansberg
Bacillary White Diarrhoea		(1) Lions River		
Foot and Mouth Disease	(32) Vryburg			
Fowl Typhoid	(1) Belville (1) East London (1) Malmesbury (1) Stellenbosch (1) Wynberg	(1) Dundee		
Lumpy Skin Disease	(1) Barkly West (1) George (1) Port St. Johns (16) Swellendam (1) Vryburg (1) Barkly West (1) Indowe	(1) Babanango (6) Estcourt (1) Entonjaneni (2) Eshowe (2) Klip River (5) Lions River (1) Mtunzini (3) Ngotshe (1) Nongoma (19) Paulpieters-burg (1) Umvoti (20) Vryheid	(1) Bethlehem (1) Bethulie	(11) Barberton (8) Letaba (1) Lydenburg (5) Pietersburg (1) Potgieters-rus (2) Zoutpansberg
Mange (Goat)	(1) Butterworth (1) Elliotdale (3) Glen Grey (1) Kingwilliams-town (10) Middeldrift (5) Mqanduli (1) Nqamkwe (1) St. Marks (1) Stutterheim (1) Umtata	(1) Nongoma (1) Ingwavuma		(1) Johannesburg
Mange (Equine)	(1) East London (1) Engcobo (1) Kentani (1) Libodi (1) Mqanduli (1) Mt. Currie (3) Qumbu (9) St. Marks (4) Tsomo (1) Tsolo (12) Umtata (1) Xalanga	(2) Impendhele		
Mange (Porcine)	(5) Barkly West			
Mange (Sarcoptic)	(1) St. Marks (1) Umtata			
Rabies	(1) Gordonia (1) Kuruman		(1) Hoopstad (1) Edenburg (1) Kroonstad (1) Reddersburg (1) Vrede	(2) Klerksdorp (2) Ermelo (4) Letaba (1) Schweizer Reneke (1) Sibasa (1) Wolmarans-stad (1) Zoutpansberg
Sheep Scab	(6) Ceres (1) Laingsburg (17) Worcester			
Swine Erysipelas				(1) Krugersdorp (1) Pretoria (1) Newtown (Johannes-burg)
Trypanosomiasis (dourine)	(1) Gordonia (2) Kuruman			
Tuberculosis (Avian) (Bovine)	(1) Caledon (1) Albany (1) Humansdorp (1) Mt. Currie (1) Pearston (1) Port Eliza-beth	(1) Pieter-maritzburg	(1) Edenburg (3) Bothaville (1) Bloem'ontein (1) Kroonstad (3) Vrede	(1) Carolina (1) Klerksdorp (2) Potchef-stroom (1) Pretoria



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