South African
Veterinary Medical Association



55th Annual Congress

ONDERSTEPOORT,

AUGUST 29th — SEPTEMBER 2nd, 1960.

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PROGRAMME

of the
55th Annual Congress
Faculty Buildings, Onderstepoort,
August 29th to September 2nd, 1960.

MONDAY — August 29th

8.00 a.m.	Registration.
9.00 a.m.	Convocation of meeting.
9.05 a.m.	Welcome to guests, delegates and members attending Congress: Dr. R. A. Alexander, Director of Veterinary Services.
9.10 a.m.	Official opening of Congress by Dr. S. M. Naudé, President of the Council for Scientific and Industrial Research. Reply on behalf of the Association: The Vice-President.
10.00 a.m.	The Presidential address: Dr. H. P. Steyn.
10.30 a.m.	Opening of Trade Exhibition by the Vice-President (Dr. R. du Toit).
10.45 a.m.	Tea.
11.15 a.m.	International Animal Disease Control: Dr. M. C. Lambrechts.
12 noon	Foot and Mouth Disease in Southern Africa: Dr. R. A. Alexander.
12.30 p.m.	Lunch
2.00 p.m.	The continuous flow culture of micro-organisms as a research tool in biology and biochemistry: Dr. H. Weyland.
2.30 p.m.	The diagnosis of pulpy kidney disease: Dr. B. C. Jansen.
3.00 p.m.	Meeting of the Public Health Group of the Association.
3 p.m.	A report on naturally-acquired besnoitiosis in bovines, with special reference to its pathology: Prof. K. C. A. Schulz.
3.30 p.m.	Preliminary observations on the mechanical transmission of cyst organisms of *Besnoitia besnoiti* (Marotel, 1912) from a chronically infected bull to rabbits by *Glossina brevipalpis*, Newstead, 1910: Dr. R. D. Bigalke.
4 p.m.	The role of infested dung in the transmission of nematode parasites of cattle: Dr. R. K. Reinecke.

TUESDAY — August 30th

8.30 a.m.	The study of foam formation in the rumen in relation to frothy bloat: Miss E. A. Rous.
9 a.m.	Icterus and photosensitivity: modern concepts regarding aetiology: Dr. J. M. M. Brown.
9.30 a.m.	Bacterial icterus in sheep: Drs. T. F. Adelaar and R. C. Tustin.
10 a.m.	Notes on trace-element requirements according to a rapid survey of the Union: Dr. J. W. Groenewald.
10.30 a.m.	Tea.
11 a.m.	Symposium on the rearing and care of newborn animals: (i) Some aspects of the nutrition of the calf: Dr. M. W. Henning.

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(ii) The care and management of thoroughbred foals: Dr. T. Toms.
(iii) Management of the newborn pig: Dr. R. K. Loveday.
(iv) The rearing of puppies: Dr. J. R. Frean.
Opener of discussion: Dr. M. W. Henning.

WEDNESDAY — August 31st

Symposium on animal reproduction:

8.30 a.m. General introduction to symposium: Dr. J. S. van Heerden.
9 a.m. Artificial insemination under ranching conditions: Dr. I. G. Horak.
9.30 a.m. Some remarks on the problem of poor fertility in dairy herds served by the Natal A.I. co-operative: Dr. D. J. le Roux.
10 a.m. The physiology of reproduction in the mare, with special reference to the thoroughbred: Dr. C. W. A. Belonje.
10.30 a.m. Tea.
11.15 a.m. The control of brucellosis in sheep and goats by means of vaccination: Prof. G. C. van Drimmelen.
11.45 a.m. Shy breeders in canines: Dr. J. L. Doré.
12.15 p.m. Lunch.
2 p.m. Discussion on some veterinary aspects of artificial insemination by S.A.V.M.A. members in committee:
(i) The training of inseminators.
(ii) The treatment of cows by inseminators.
(iii) The role of the veterinarian in A.I. organisation.
7.30 p.m. Formal dinner.

THURSDAY — September 1st

8.30 a.m. Lumpy skin disease: Dr. K. E. Weiss.
9 a.m. Newer developments in the field of anaesthesia of large animals: Prof. O. Dietz, Humboldt University, Berlin.
10.30 a.m. Tea.
11.15 a.m. A closed-circuit method of anaesthesia in small animal practice: Dr. P. H. le Roux.
11.30 a.m. Short film: Inhalation anaesthesia in small animals: Dr. J. L. Doré.
11.45 a.m. Considerations regarding shock and the poor-risk surgical patient: Prof. C. F. B. Hofmeyr.

FRIDAY — September 2nd.

8.30 a.m. Group meeting of agricultural faculty and agricultural college lecturers in veterinary science.
8.30 a.m. Film.
9 a.m. Medical clinical forum.

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10 a.m. The value of the determination of transaminase activity in plasma as a screening test for liver disease in animals: Dr. W. D. Malherbe.

10.30 a.m. Tea.

11.15 a.m. Annual General Meeting of the Association—Agenda:

(i) Confirmation of minutes of 54th Annual General Meeting, held at Onderstepoort on 27th August, 1959.
(ii) Matters arising from these minutes.
(iii) Annual report of Council—The President.
(iv) Membership:
  (*a*) Deaths
  (*b*) Resignations
  (*c*) Election of new members
(v) Consideration of reports:
  (*a*) Balance sheet and Auditor's Report.
  (*b*) Standing Committees.
(vi) Venue of Congress
(vii) Notification of election of Council.
(viii) Special resolution to change constitution (enclosed notice).
(ix) General
(x) Resolutions
(xi) Adjournment.

12.30 p.m. Lunch.

2 p.m. Continuation of Annual General Meeting.

IMPORTANT NOTICE — BELANGRIKE KENNISGEWING

Change of Address.

The attention of registered veterinarians is once again directed to Section 13 (a) of the Veterinary Act (Act No. 16 of 1933) from which the importance of keeping the Registrar of Veterinarians, P.O. Onderstepoort informed of any change of address is evident.

Adresverandering.

Die aandag van geregistreerde veeartse word andermaal gevestig op Seksie 13 (a) van die Veeartswet (Wet 16 van 1933) waarvolgens die belangrikheid om die Registrateur van Veeartse, Pk. Onderstepoort van enige adresverandering op hoogte te hou duidelik sal blyk.

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THE CONTINUOUS FLOW CULTURE OF MICRO-ORGANISMS AS A RESEARCH TOOL IN BIOLOGY AND BIOCHEMISTRY

H. Weyland*

When a small number of mono-cellular micro-organisms is added to a suitable liquid medium in a conventional culture flask, multiplication of the cells will follow a period of adaptation. In time, this multiplication will result in the attainment of a characteristic maximal population. This maximum can be maintained for some time, but usually the stage is soon reached where the cells start dying off. In order to maintain the process of growth, it is then necessary to repeat the procedure outlined above, that is, to make subcultures. This is the conventional way of propagating micro-organisms in batches.

During the process of growth of a batch culture, the population passes through a series of developmental phases, characterised by different growth rates. The temporary lag phase, when development is non-existent or very slow, is succeeded by a period of increasing growth rate, then by a phase of maximum and constant development (the log-phase or exponential phase), before finally phases of decline set in. These changes are accompanied by changes in the metabolism of the organisms from one phase to the other. At the same time the medium surrounding the organisms is constantly changing, because by increasing cell density the concentration of nutrients is decreased and the formation of catabolic products is increased.

For certain investigations both the constant changes of chemical and physical composition as well as the changing cell density and growth rate of the organisms are extremely undesirable. It is often necessary for the investigator to stabilize a specific stage of actively growing cells, and to keep the composition of the surrounding fluid constant. This is of importance to the industrial biochemist too, when a specific biosynthesis by a micro-organism occurs only at a certain point in the growth cycle of a batch culture. It might be limited to a very short period in relation to the complete cycle. This so-called "time factor" can be eliminated by the use of continuous culture methods.

A sound theoretical basis for continuous cultivation systems was advanced independently by the Frenchman Monod[5] and the Americans Novick and Szilard[6] in 1950. During the ten years that followed, this method has developed into an indispensable aid to both industry and research.

PRINCIPLES

A "continuous culture of micro-organisms" in the broadest sense of the word can be attained in different ways. The original method of keeping a population of micro-organisms growing indefinitely consisted,

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for example, of removing daily part of the culture-suspension and replacing it with fresh medium, thereby in effect eliminating the lag phase and the phase of decline by keeping the cells in the phase of active proliferation. These methods are really "continuous batch cultures" of the organisms, and present no solution for the discontinuity of the process. The cell density, the composition of the medium, and the physiological state of the cells are still subject to the same fluctuations. However, even in the latest literature this method is still called continuous culture, especially in the cultivation of animal cells, where continuous culture in the real sense of the word, namely "continuous flow culture", still presents great difficulties.

The technical difference between a culture with periodic renewal of nutrients (the terms "continuous batch", "semi-continuous" or "periodic continuous culture" are sometimes used for this type) and a "continuous flow culture", is to be found in the length of the interval between two additions of medium. Technically a continuous flow culture is only an extreme case of the semi-continuous culture because renewal of medium can still be intermittent, but with the smallest possible interval between additions.

The physiological difference however, is more marked, and manifests itself in the dynamic equilibrium, that is, the steady state, which characterises the continuous flow culture. In this steady state the concentration of growing organisms, the substrate and other variable factors remain constant indefinitely.

The conditions necessary for the attainment of a dynamic equilibrium, include a method of balancing the growth rate of the organisms, i.e. the increase of cell concentration per volume per unit time, by diluting the culture volume with fresh medium at the same rate, i.e. the dilution rate. With other words: when the cell concentration has been doubled, the culture volume, too, has to be doubled. Such a statement implies that the culture volume must increase. In practice, the volume of the culture may also be held constant by removing culture fluid from the culture vessel at a rate corresponding to the rate of addition of fresh medium to the culture. Thus the culture volume is held constant but the culture itself continually flows.

The necessary control of the flowing culture can be done in two different ways. In the first a batch culture is allowed to grow in an optimum medium until the exponential phase is reached, where the growth rate is maximal and constant for the specific organism and medium. At any convenient point in this phase of constant increase of cell concentration the addition of medium is started at the same rate as that at which the organisms grow. At the same time an equal volume of culture medium plus cells is allowed to escape from the flask through an overflow device. The dilution rate is thus brought into line with the growth rate. In this way it is possible to stabilize the cell density in an exponentially growing culture, and thereby to create a true dynamic equilibrium, which could, theoretically, be maintained as long as desired. This method of continous flow culture is characterised by the fact that the organisms grow at a maximal rate, dependent on the inherent possibilities of the nutrient medium and the specific

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organism, which cannot be changed. The flow rate must be adapted to the growth rate which acts in this case as an independent constant.

On the other hand it is possible to control the cell density, within the limits set by the exponential growth phase, by the choice of the starting point for the addition of medium at the onset of the experiment. If it is desired to increase the density during a run, it is only necessary to stop the flow, allow the cells to multiply until the desired density is reached, and then to continue the process. If a decrease in density is desired, the dilution rate can be increased to a point above the growth rate, whereby the cells are washed out of the system faster than they can multiply, and the density is lowered. At the desired density, the dilution rate is returned to the value compensating the growth rate. In both cases a state of dynamic equilibrium is again obtained.

An apparatus which makes this type of continuous flow culture possible, is called a "turbidostat" because the primary measurement of the microbial density or turbidity by a photo-electric system, is used secondarily for the adjustment of medium inflow by an automatic feeding device. In this way deviations of the density are corrected, and a steady state stabilized.

Another apparatus which meets the requirements for the attainment of an equilibrium between the growth rate and the dilution rate, is the so-called "chemostat". Here the culture volume is also kept constant by equal in- and out-flow of medium. But contrary to the "turbidostat", in which the concentration of micro-organisms is held at a fixed value and the dilution rate allowed to find its own level, in the "chemostat" the dilution rate is set at a predetermined value and the microbial concentration allowed to find its own level.

The key of the mode of action of the chemostat, however, lies in the way in which the exponential growth rate depends on the concentration of a "limiting (or controlling) growth substrate" in the culture medium. If the composition of the medium is selected in such a manner that all essential growth substrates are in excess except one—the limiting substrate—then the exponential growth rate is found to vary with the concentration of this limiting substrate. The range, within which the growth rate depends on concentration, has an upper as well as a lower limit. The upper limit is the concentration of the limiting factor which allows maximum growth of the organism. The lower limit is probably set by some mechanism which causes the organisms to go into lag phase; they may even cease to be viable when the concentration of the growth factor becomes too low.

In a batch culture, substrate is consumed as the organisms grow; the substrate continually decreases and consequently the growth rate decreases, both eventually falling to zero.

In the chemostat, however, the continued steady addition of fresh medium containing a limiting concentration of a controlling growth substrate fixes the substrate concentration, and hence the growth rate. The chemostat is therefore a device for controlling growth rate through control of substrate concentration and the substrate concentration of a given medium is, in turn, controlled through the dilution rate.

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This system then, in contrast to the previous one, has the very desirable property of being self-regulating. It offers the advantage of allowing experimentation at different values of the exponential growth rate. This may be achieved by simply adjusting the value of the dilution rate to the numerical value of the desired exponential growth rate which must be smaller than the maximum growth rate. This system will then automatically adjust itself to a steady state in which the concentration of the micro-organisms, their metabolism, the chemical composition of the environment, the pH-value and all other variables remain constant indefinitely, so long as the dilution rate remains unaltered. In practice, therefore, we can force the organisms to grow at any desired exponential growth rate by merely adjusting the dilution rate. In practice, a pump or another device is required for the exact fixing and controlling of the inflowing medium. Another requisite of the continuous flow system of that kind is that the composition of the culture fluid is the same at all points of the culture space. Therefore the culture has to be stirred very well. The culture leaving the culture vessel is collected in sampling tubes or a receiving vessel, which can be refrigerated to stabilize the product.

In order to be able to understand and to predict the experimental conditions that will lead to nearly any desired state in the culture, a rigorous theoretical treatment of the system is necessary; this is beyond the scope of the present discussion. Mathematical description of the steady state, equations from which the steady state concentrations of organisms and substrate in the culture vessel can be predicted for any value of the dilution rate and concentration of inflowing substrate were derived by a number of workers following the basic work of Monod[5]. To a certain extent it is also possible to predict the product formation and the output of cells for different continuous flow systems. Certain interesting deductions, concerning the chemostat system may be mentioned:

1. The steady state concentration of the limiting substrate in the culture vessel depends only upon the dilution rate.

2. The steady state concentration of the limiting growth substrate in the culture vessel is independent of the inflowing limiting substrate concentration.

3. By increasing the inflowing limiting substrate concentration only the cell concentration is increased without affecting the steady state substrate concentration in the culture vessel. The cell concentration may therefore be controlled by altering the concentration of the inflowing limiting substrate.

4. The steady state concentration of the limiting growth substrate in the culture vessel is extremely low over the greater part of the possible range of dilution rates.

It may be stated that, independent from stabilizing a phase of development of a population, certain variables, which are completely beyond control in a batch culture, become independent variables in a continuous flow culture of micro-organisms, thus enabling the experimenter to work with numerous different steady states.

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APPLICATIONS

Some advantages of the continuous flow culture method in relation to the conventional batch culture may be summarized here:

1. Micro-organisms may be propagated with the exponential growth rate over boundless periods.

2. The exponential growth rate may be controlled and regarded as an independent variable.

3. The concentration of the continually propagated organisms may be varied.

4. The chemical and physical environment as well as the physiological state of the organisms can be stabilized and controlled, thus the very important "time factor" in batch cultures is completely eliminated.

5. Any particular essential growth factor can be selected as limiting growth factor, and its concentration can be controlled and maintained constant at very low values.

6. A marked reduction in processing time is possible for the same holding capacity as compared to batch procedure.

Some examples may illustrate these advantages:

(*a*) In certain production processes the continuous flow system can be superior in comparison with batch techniques since in a piece of equipment of a specific size one can get more product in a given time if the process is continuous. If for example the cell mass, number or concentration of organisms is the desired end-product, an optimal cell concentration may be collected from a continuous system for 24 hours a day for long periods. Therefore, in certain branches of industry, continuous flow culture is in routine operation, for instance in the mass-production of yeast, yeast products and acetic acid. The method is used also in waste disposal processes. A number of commercial organizations have conducted continuous fermentations of antibiotics and biochemicals (Vitamin B_{12}) to various stages of development.

In research work, bulk growth of micro-organisms of defined metabolic activity is often necessary for enzyme studies. Here continuous flow culture is used with success by biochemists.

(*b*) That the continuous flow culture of micro-organisms may imitate conditions *in vivo* better than the batch type culture is exemplified by the work of Harris-Smith *et al.*[3]

It was found that a toxin is produced by *Bacillus anthracis in vivo*. Later on this lethal oedema-producing toxin could be detected in *in vitro* batch cultures of the virulent N.P. strain as well as in the attenuated immunogenic Sterne strain. But in these static cultures, traces of the toxin appeared only in very young cultures over a short period of growth. Attempts to maintain the toxin concentration or to increase it by addition of various nutrients to batch cultures failed. Only by continuous culture have the organisms been kept in their toxigenic growth phase.

Previous non-recognition of the toxin *in vitro* was due to its early appearance and rapid disappearance under ordinary growth conditions

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in batch culture. Later, a toxin-destroying mechanism was demonstrable in organisms from cultures grown for more than a few hours. Apart from the possibility that other toxins or other unknown metabolic products of micro-organisms may be detected only, or more easily, by the application of the continuous flow culture method, this experiment clearly shows that a biochemist has to apply the continuous culture method in order to isolate such a toxin in quantitative amounts for further investigation.

(c) Continuous culture methods have found widespread application for the study of mutation and mutation rates. Attention is directed to the work of Novick and Szilard[7], who have used the method to develop a new phenomenon. In the study of the mutagenic effects of certain purines in a bacterium-bacteriophage system, they have shown, by means of the continuous flow culture, that certain compounds possess anti-mutagenic properties. In the presence of the anti-mutagen, the appearance of chemically-induced as well as spontaneous mutations was reduced to a minimum.

(d) The method of continuous culture can be said to provide experimental conditions exceptionally favourable for revealing selective phenomena not observed by conventional methods.

By growing *Escherichia coli* in a Turbidostat under conditions suitable for revealing selective pressures in microbial populations Cocito and Bryson[2] could demonstrate that mutants of *E. coli* resistant to a certain type of a phage were able to outgrow their phage-sensitive parents at an exponential rate. From analysis of the selection curve it was concluded as one possibility that the predominant parental strain was liberating a substance inhibitory to itself, possibly a colicine. Biochemical work then resulted directly in the discovery of an antibacterial protein produced by *E. coli*, the existence of which had not been previously suspected.

(e) One of the advantages of the continuous culture method is that it may lead to previously unattained speed or precision on the accumulation of experimental data. Sensitive bacterial strains frequently produced resistant mutants, able to grow in the presence of toxic or selective agents. These mutants characteristically attain their resistant properties through a series of intermediate stages requiring successive experimental isolations at increasingly higher concentrations of the agent active against the parent strain. Bryson and Szybalski[1] developed a procedure of exposing cells to a continuous concentration gradient of selective agents in a continuous culture. In this manner, selection for neomycin-resistant forms has resulted in the attainment of subcultures able to grow at neomycin-concentrations of 16 times the original inhibiting concentration. Selection for resistance to a quaternary ammonium compound resulted directly in a 16 times increase in the amount of this substance required for sterilization.

(f) During a study of the metabolism of mycobacteria in a flow system, Kuska[4] made the following experiment: A strain of *Mycobacterium tuberculosis*, which was resistant to 100 micrograms iso-

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nicotinic acid hydrazide (INH)/ml. in a static culture, was cultivated simultaneously in batch and continuous flow culture. Both cultures contained 50 micrograms INH per ml. Two other cultures, one batch and one continuous culture, without INH served as controls. No significant difference in the growth was found between the two batch cultures. However, significant suppression of the growth occurred in the flow culture containing INH.

The organisms resistant to 100 micrograms INH/ml. in a batch culture were already affected by 50 micrograms INH/ml. in the flow culture.

This result explained to a certain extent the differences between the values of the resistance found *in vivo* and *in vitro*, where the degree of resistance of the strains *in vivo* is smaller as a rule. It may be assumed that in this flow culture the microbes were in a physiological state which bore a likeness to that *in vivo*.

The important point to be underlined is that the advantage of continuous culture lies not only in the continuity of the growth process as such, but in the possibility of adjusting different steady states.

In the quoted examples, which could not be dealt with in detail, it was important to select the most suitable steady state by variation of the growth rate, cell density, the type and concentration of the limiting substance and other factors. In this possibility lies the power of this method as a research tool: previously uncontrollable dependent variables become independent variables.

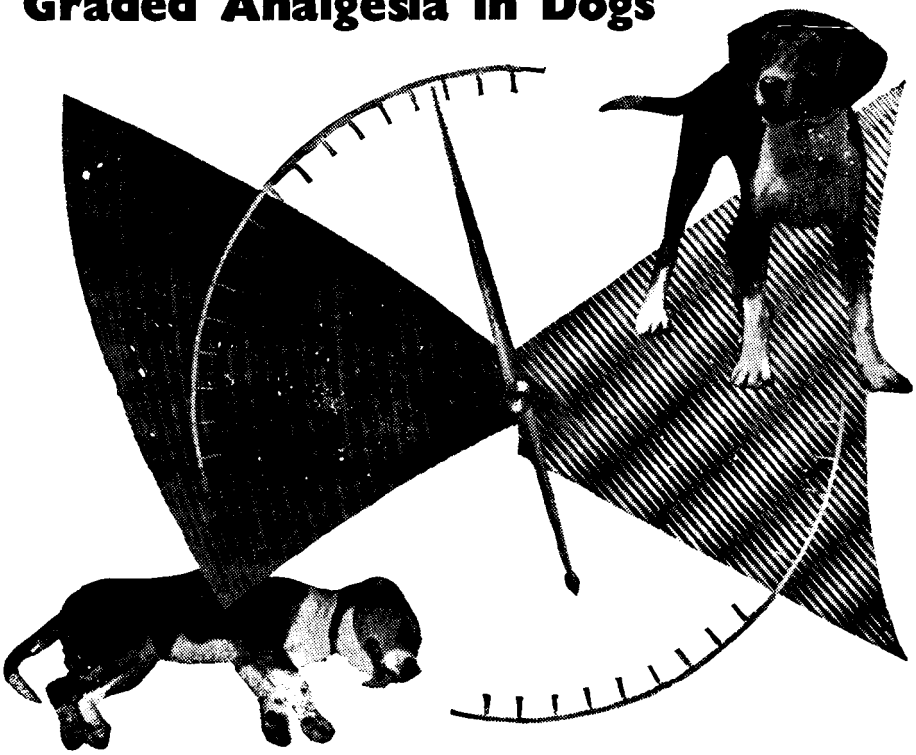
The few examples quoted may give an indication of the possibilities of this tool in industry, and in applied and basic research. Therefore it should be clear why, at this institute, we should take an active interest in this method.

REFERENCES

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THE DIAGNOSIS OF PULPY KIDNEY DISEASE

B. C. Jansen*

Pulpy kidney disease or enterotoxaemia is an acute disease of sheep which leads to sudden death. In practice clinical cases are seldom seen. The occasional cases that are seen display nervous symptoms and sometimes slight diarrhoea. At post-mortem no signs pathognomonic of this disease can be detected. Since these circumstances readily lead to confusion in diagnosis for the field worker it seems appropriate to consider the different aids that can be applied when in difficulty. Before, however, discussing the criteria for establishing a diagnosis individually, an account will be given of the pathogenesis of pulpy kidney disease as it is generally understood at present:

Bennetts[1] isolated *Cl. welchii* type D from the abomasum of two healthy sheep in an area where the disease had occurred. Bullen[2] detected the same organism in the alimentary contents of 46 of 100 normal sheep killed at the Cambridge abattoirs. It is, therfore, well established that the causal organism can be present in the intestinal contents of normal sheep and it seems likely that the disease develops when conditions in the intestines become suitable for its multiplication and toxin production.[3]

Bullen and Batty[3] give an account of the development of a method of experimental reproduction of enterotoxaemia and mention the following findings:

Continuous dripping of actively growing *Cl. welchii* type D culture into the duodenum of normal sheep for hours does not produce the disease as long as the sheep are fed on a normal diet. The disease can, however, be reproduced if the diet of the sheep is suddenly changed to rich food e.g. wheat and they are allowed to over-eat before the culture is dripped into the duodenum. When a digestive upset occurs under these circumstances undigested starch granules escape from the rumen and enter the small intestine. Growth and toxin production of *Cl. welchii* type D is stimulated by the starch granules leading to an accumulation of bacteria and toxin in the lumen of the intestines. Whereas normally a high concentration of toxin and bacteria is rapidly cleared from the gut by the onset of a diarrhoea, under these circumstances the clearing mechanism cannot cope with the accumulation of toxin.

A low concentration of epsilon toxin in the gut is harmless and remains virtually unabsorbed, but lethal amounts of toxin are rapidly absorbed if high concentrations are maintained in the gut for some time. High concentrations of toxin in the lumen increase the permeability of the gut wall. The absorbed toxin is directly responsible for the death of the sheep.

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The above observations explain the rationale of some of the criteria applied for arriving at a diagnosis. An evaluation of the diagnostic aids follows:

(1) Post mortem appearance:

As a result of the development of a relatively simple method of experimental reproduction of enterotoxaemia,[4] it has been possible to observe the post-mortem appearance of carcases at different stages after death. In a fresh carcase the bowels are invariably distended with gas; the kidneys are swollen, hyperaemic and the pericard is distended with clear fluid which may contain a coagulum. Sub-endocardial and -epicardial petechiae are commonly observed but for the rest there is nothing typical. The carcases tend to undergo putrefaction rather rapidly; the kidneys becoming pulpy.

(2) Detection of epsilon toxin in the gut contents.

In a fresh carcase epsilon toxin can be detected readily in the gut contents and the specificity of this toxin can be proved by conventional methods. In a carcase that was left lying at room temperature for 12 hours toxin could still be detected in the intestinal contents.

The intestinal contents from a fresh carcase were collected in a clean container and preserved by the addition of one drop of chloroform to every 10 ml. of liquid and left standing at room temperature. At irregular intervals its toxicity was tested and the specificity of the toxin proved by serum-neutralisation. It was found that this material was still toxic one month after collection.

When, however, a section of intestine containing toxic contents is tied-off at both ends and preserved in 50 per cent glycerine, the toxicity of the contents is lost within 12 hours.

It, therefore, seems advisable to collect intestinal contents from fresh carcases and preserve this material with chloroform for submission to a laboratory for diagnosis. More success is likely to follow this procedure than submitting sections of intestine in 50 per cent glycerine.

The detection of epsilon toxin in the gut contents of sheep is not conclusive evidence that the animal has died of enterotoxaemia, since immune sheep can tolerate high concentrations of toxin in their intestines.

(3) The presence of *Cl. welchii* type D in the gut contents.

Since this organism has been isolated from the intestinal contents of normal sheep,[2] finding it in a specimen of gut contents has no diagnostic significance.

(4) The detection of typical *Cl. welchii* type D in smears of intestinal contents.

Smears should be prepared from the contents of the ileum and stained by Gram's method.

In cases of enterotoxaemia typical *Cl. welchii*, i.e. short, fat, Gram-positive organisms, occur in the smear to the complete or almost complete exclusion of all else. In my own experience this is a very common finding, confirming the views of Rowlands.[10]

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(5) Glycosuria.

Gordon, Stewart, Holman and Taylor[5] report that urine from cases of enterotoxaemia caused by *Cl. welchii* type D contain a great deal of sugar. The sugar content of the blood in these cases was equally high. This upset of sugar metabolism could not be detected in animals injected with the toxins of *Cl. welchii* types B and C.

Bullen and Batty[3] found a well-marked hyperglycaemia and an associated glycosuria in 9 out of 10 experimentally produced cases of enterotoxaemia (1 was not examined). They used this symptom to distinguish between enterotoxaemia and plain acidosis following on over-eating.

The occurrence of glycosuria in natural cases of enterotoxaemia was confirmed by me.

(6) Level of immunity.

Smith and Marsh[6] state: "0.3 units of antitoxin per millilitre suffices to give adequate protection, and it is probable that appreciably lower concentrations are sufficient to give protection against enterotoxaemia".

Thomson and Batty[7] estimate that an antitoxin level in the order of 0.1 unit per ml. is the lowest level which will afford protection against enterotoxaemia.

Jansen[4] challenged immune sheep by injecting culture and dextrin into their duodenum. Before being challenged they were bled and the antitoxin value of their sera determined in terms of Wellcome units. The results are recorded in Table 1.

TABLE I

THE RESULTS OF CHALLENGING SHEEP WITH DIFFERENT LEVELS OF CIRCULATING EPSILON ANTITOXIN

Number challenged	Antitoxin level units ml.	Result
2	0	Both died
7	0.1	3 lived 4 died
4	0.15	4 lived
2	0.3	Both lived

From these results it can be reasonably concluded than an antitoxin level of at least 0.15 units per ml. of circulating antitoxin renders a sheep immune to pulpy kidney.

At times veterinarians and farmers complain that sheep keep on dying of enterotoxaemia in spite of repeated injections of alum-precipitated epsilon toxoid at reasonable intervals. In several such cases I have requested the veterinarians in charge of flocks affected

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to send me serum samples from sheep selected at random. In every instance I established that the serum samples showed a high antitoxin titre (sometimes as high as a mean value of 30 units per ml.). The significance of this finding is that the sheep concerned were fully protected by the injections of vaccine and, therefore, could not have died of enterotoxaemia.

Jansen[4] proved that when sheep were given two injections of Onderstepoort Pulpy Kidney Vaccine as primary and secondary stimulus at an interval of 3 to 4 weeks, they showed a geometric mean titre of 42 units per ml. one week after the second dose.

(7) The detection of *Cl. welchii* type D in liver, kidney and lymphnode specimens.

As mentioned under point (3) above, the isolation of *Cl. welchii* type D from intestinal contents has no diagnostic significance. In the cases of enterotoxaemia artificially produced at Onderstepoort[4] specimens from the liver, kidney and mesenteric lymphnodes were collected at post-mortem with aseptic precautions before the intestines had been opened. Subsequently material from these different organs was transferred separately into Robertson's meat broth tubes and pasteurised at 60°C for 30 minutes. After overnight incubation the contents of the tubes were tested for the presence of epsilon toxin by conventional methods. The results are recorded in Table 2.

From these results it can be seen that out of the 16 liver specimens examined 14 were found positive; all 6 kidney specimens examined were positive, and only 4 out of 16 lymphnode specimens were negative.

Whereas the epsilon toxin is directly responsible for the death of an animal, it seems that the organisms enter the bloodstream in the terminal stages of the disease. While this occurs in the artificially produced cases, there seems to be no reason why it should not take place in the natural disease. Field veterinarians are urged to collect liver and kidney specimens from suspected cases and submit them to the laboratory in 50 per cent glycerine. The specimens should, however, be collected as cleanly as possible before opening the gut.

(8) The occurrence of pulpy kidney after dosing phenothiazine.

Since the introduction of phenothiazine as anthelmintic occasional reports have been received at Onderstepoort of sheep dying of enterotoxaemia subsequent to dosing. No occasion for proving these claims arose until Jansen[4] produced enterotoxaemia in susceptible sheep by dosing them with the therapeutic dose of phenothiazine and immediately afterwards introducing 20 ml. of actively growing culture of *Cl. welchii* type D into their duodenum under local anaesthesia. Control susceptible sheep, receiving the culture only, lived.

This finding emphasises the necessity of ensuring a protective level of immunity before dosing sheep with phenothiazine.

Differential Diagnosis

(1) Overeating predisposes to enterotoxaemaia but by itself can be responsible for death in sheep. When sheep overeat on a carbohydrate rich diet they develop an acidosis. This, however, is likely

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TABLE 2

THE DISTRIBUTION OF *Cl. welchii* TYPE D IN ORGANS COLLECTED AT POST MORTEM

Sheep No.	Liver	Kidney	Lymph-gland
7815	+	NT	+
8107	+	NT	+
8115	+	NT	+
8125	+	NT	+
7902	+	NT	—
8114	—	NT	+
8098	+	NT	+
8097	—	NT	—
8688	+	NT	+
8036	+	NT	+
8053	+	+	+
8708	+	+	+
8705	+	+	—
8069	+	+	+
8711	+	+	+
7984	+	+	—

\+ = positive for Cl. Welchii type D.
— = negative ,, ,, ,, ,, ,,
NT = not tested

to occur only under feedlot conditions where enterotoxaemia is also expected to occur. Bullen and Batty[3] state that enterotoxaemia can be differentiated from acidosis by the fact that hyperglycaemia and glycosuria are not shown by sheep dying of acidosis. This was confirmed by Brown and Jansen[8].

Sheep being fed on a protein rich diet under feedlot conditions may develop an alkalosis due to overeating. Clark[9] established that sheep dying of alkalosis as a result of overdosing with urea show a marked hyperglycaemia and glycosuria. It is furthermore a well established fact that sheep in the terminal stages of certain metabolic diseases e.g. enzootic icterus, domsiekte and geeldikkop show hyperglycaemia and glycosuria.

From this can be concluded that the absence of sugar in the urine collected from a dead sheep has differential diagnostic significance in so far as it points very definitely to the exclusion of enterotoxaemia as the cause of death.

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(2) Rowlands[10] mentions that in North Wales metabolic disorders among sheep associated with improved grassland followed a pattern closely resembling enterotoxaemia.

(3) In South Africa "geilsiekte" which is synonymous with hydrocyanic acid poisoning is very frequently confused with enterotoxaemia.

(4) Fungus intoxication can be responsible for sudden deaths and the active principle contained in some fungi has a severe diuretic effect leading to hyperaemia of the kidneys.

(5) Vegetable poisoning leading to sudden death without typical post mortem lesions.

DISCUSSION

Although the diagnosis of pulpy kidney disease is certainly not easy, it should be possible to reach a decision when the points listed above are considered. In the first instance the flock as a whole or the majority of its members must be susceptible (the susceptibility of sheep can be easily determined by antitoxin titration of the sera of sheep selected at random). There is usually a history of a change of grazing followed by sudden deaths, but sometimes, especially where sheep are kept on rich pasture permanently, a change of grazing is not a prerequisite. The sheep dying of enterotoxaemia show a negative post mortem except for softening and reddening of the kidneys, accumulation of gas in the intestines and hydropericardium. Smears taken from the intestinal contents show typical *Cl. welchii* type D organisms to the exclusion of other bacteria; the presence of epsilon toxin in the gut contents can be demonstrated and the organism can be isolated from kidney and liver specimens.

When, through unforeseen circumstances, it is impossible to obtain fresh carcases for post mortem examination or when it is impossible to reach a conclusion after conducting post mortem examinations, recourse could be taken to vaccinating the flock against pulpy kidney. When sheep which have a basic immunity, i.e. which have had an injection or injections of alum-precipitated pulpy kidney vaccine more than a month previously, receive a booster injection of alum-precipitated pulpy kidney vaccine they should be fully resistant to the disease after about seven days.

SUMMARY

A description of the pathogenesis of enterotoxaemia is given as well as a description of the laboratory and field aids that can be applied to arrive at a diagnosis. The differential diagnosis is discussed.

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A REPORT ON NATURALLY ACQUIRED BESNOITIOSIS IN BOVINES WITH SPECIAL REFERENCE TO ITS PATHOLOGY.

K. C. A. Schulz*

Bovine Besnoitiosis is a protozoal disease caused by *Besnoitia besnoiti* (Marotel, 1912) Henry, 1913. The causal organism has been designated as *Gastrocystis besnoiti* (Marotel) *Sarcocystis besnoiti* and *Globidium besnoiti* in the past. A discussion on the nomenclature of this parasite does not lie in the scope of this paper. It has been dealt with satisfactorily on a previous occasion.[1]

In the acute stage the disease is characterized by fever, a staring hair coat, anorrhexia, respiratory distress, photophobia, lacrimation, a muco-purulent rhinitis, a transient anasarca involving various parts of the body to a variable extent, swelling of the superficial lymphnodes, stiffness and general weakness; a variable degree of depilation and chronic dermatitis crustosa resulting in a markedly thickened, wrinkled, creviced, scurvy and less pliable skin (scleroderma), are manifestations of the chronic stage. In view of this latter change the disease has been referred to as "dikvelsiekte" (thick-skin disease), "olifantvelsiekte" (elephant-skin disease) or "beesskurfte" (bovine mange).

In South Africa typical mature Besnoitia cysts were observed for the first time in skin sections of a horse in the Grahamstown area (1940)[2], and subsequently the first authentic case in bovines, confirmed by histological examination of affected portions of skin, was seen during March 1941 on a farm in the Rustenburg District[3]. Presumably the disease spread from the North and cases occurred in the Rustenburg district four to five years prior to its investigation and diagnosis. Since then, as will be seen later, the affected area reached considerable dimensions involving the whole of the bushveld up to the Eastern Transvaal. In addition, Besnoitiosis is limited to certain other geographical localities. It has been recorded in Europe from Southern France, Besnoit and Robin, 1912, and Portugal, Borges and Franco, 1916, and from Africa in the Sudan(1933)[4], Angola (1949)[5], and Belgian Congo (1952)[6].

Although the condition has been known to occur in horses and bovines for a considerable time, the mode of its transmission has remained obscure. It has been suggested, however, that it may be transmitted by contact[3], or by means of some intermediary host, for instance an arthropod vector[2],[6]. Artificial transmission is possible with material collected during the primary (febrile) stage of the disease, to susceptible cattle. This has been achieved by intravenous injection of infective blood (Cuillé and Chele, 1937, and Barrion, 1938), or by intradermal subcutaneous and intravenous injections of pooled emulsions of spleen and lymphglands[7] and by

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blood transfusion or intraperitoneal, injections[8]. In addition to cattle, sheep, goats and rabbits could be successfully infected by one or other of these means. The rabbit seems to be the most susceptible and suitable for studying the life cycle of the parasite, determination of the host range, and evaluation of chemotherapeutic agents. Subsequently the parasite and its propagation in the rabbit have been described and illustrated[9].

The main object of this publication is to discuss the natural infection in bovines from the pathological rather than the parasitological point of view. The latter, however, is essential in order to appreciate the former more fully and as a means in determining the systematic position of the parasite. It will be seen that the developmental stages are not consistent with or typical of those of globidium nor of sarcosporidium.

The data, to be recorded, were accumulated over a period of 18 years and are based on information compiled from about 100 questionnaires, personal investigations, those accompanying specimens, from the histological examination of tissues, especially skin from various parts of the body of affected animals sent in either by stockowners or colleagues in the field, or by autopsies carried out on several farms, in some abattoirs and at Onderstepoort. The material examined histologically involved that of 150 cases. Numerous serial sections of various tissues, especially of early developing cases, were cut and stained for a more detailed examination. In addition, skin specimens, obtained by biopsy from some introduced, affected bovines at Onderstepoort, were examined at intervals varying from several weeks to months and this procedure was followed up on individual cases over a period lasting from 2 to 6 years. (It may be mentioned here that Dr. A. D. Thomas was responsible for the early research work on this disease and for the drawing up of the form for the questionary, whereby valuable and informative data were obtained.)

Before dealing with the pathology the following points of interest may be briefly considered.

Distribution

Although the disease is widely distributed throughout the entire Bushveld, it is mainly confined to low-lying farms bordering on or traversed by watercourses such as rivers, springs and spruits. However, cattle are liable to become affected on farms where dams or open water during the rainy season and/or boreholes are the only source of drinking water. The types of soil and grass vegetation do not seem to be determining factors as the disease is seen on farms with sandy and/or clayey soil and sweet, sour or broken veld. It often does not occur on a farm/s adjoining or separated by a fence from an infected locality. This phenomenon is rather confusing to farmers. Presumably after it was introduced into the Western Transvaal, it spread from here eastwards and northwards involving the following districts to a variable extent: Marico, Rustenburg, Brits, Pretoria, Waterberg, Warmbaths, Potgietersrust, Pietersburg, Louis Trichardt (Soutpansberg), Lydenburg and Letaba. In these areas Besnoitiosis is enzootic but has not been

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observed to assume epizootic proportions. In fact, *the incidence rate* varied annually from less than 0.1% up to 20% with an average of about 4% visibly affected animals, occasionally up to 40% and in exceptional cases up to 100%, where only a few head of cattle from 20 to 6 respectively had been grazed in a particular area.

The mortality rate is not excessively high, yet the disease may nevertheless be responsible for severe pecuniary losses; their economic significance has, however, not yet been assessed. Badly affected cattle, being in a cachectic and pitiful condition, are usually destroyed for humane reasons. Their hides are valueless for tanning, their carcases are condemned in the abattoirs, milk production is reduced, cows may abort, and bulls often become sterile. Thus, needless to say, the disease is regarded with great concern by the farming community of that area.

A marked *seasonal variation* is clearly indicated, since most cases occur during the summer months (rainy season). Although the peak is reached during mid-summer (January to March), severely affected cases are not necessarily confined to this period as previously stated,[3] but may occur at any time of the season. However, the condition seems to be more prevalent during wet than dry summers. On the contrary some farmers believe the disease to occur the whole year round. This assumption, although in accordance with our records and histological findings to some extent, should be regarded with reservation. The majority of skin lesions, received during the winter and summer months, did not differ clinically and histologically appreciably from one another; in both sets signs of a dermatitis chronica crustosa accompanied only by mature cysts ("dormant forms") of the parasite were evident. In order to determine the period of transmission and consequently the age of an infection more or less accurately, it is essential to consider those cases only in which free (not in groups) and/or progressive developing forms of the parasite are demonstrable, but the rate of their development and the factors influencing it must also be taken into account. This procedure, unfortunately, loses its practical significance on the maturing of the cysts about two months after contraction of the infection. The paucity of such cases placed at our disposal is rather striking. Their relative small number sufficed however, to substantiate a seasonal (summer) incidence, ranging from the end of October to the end of May.

Age. Cattle are prone to contract the disease round about 18 months of age and onwards and even when aged. The peak is reached between 3 to 4 years. Calves appear to be non-susceptible, but affected ones, from the age of six months, have been recorded.[3] Whether young calves show a special resistance to the disease or whether they are less exposed to infection, is not known at present.

Breed. Besnoitia infection occurs indiscriminately in indigenous and imported breeds, and there does not appear to be any one, whatever the colour, with special resistance. The breeds represented on the affected farms and ranches in the Bushveld include principally Africanders and their crosses, to a lesser extent imported breeds

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(Herefords, Sussex, Redpoll, Shorthorn, Ayrshire and Friesland) and native cattle.

Sex. The relatively large number of bulls suffering from besnoitiosis is rather striking. Some farmers claim that males, oxen and bulls are particularly susceptible to the disease. This statement is borne out to some extent by our records. Of the 150 cases dealt with 52 were bulls, 21 oxen, 1 tollie, 36 cows, 2 heifers and of the remaining 38 the sex was not given. On some farms bulls and oxen were reported to be the only infected bovines. It would seem, therefore, that the apparent predominance of males is not merely coincidence, especially when the percentage of diseased bulls is compared with that of the cows in a herd. However, the recorded data by no means reflect the true state of affairs. In addition, any illness afflicting the bulls, usually the most valuable animals, will most likely attract the farmer's attention and invariably will be reported to a veterinarian. Possibly certain characteristic male habits or the local farm management methods employed in regard to these animals may expose them more to the source of infection than is the case with cows and calves.

Theories of transmission

Infectivity of pans and dams. Since the muzzle and the lower extremities often appeared markedly swollen, showing signs of a chronic dermatitis crustosa, it was assumed that cattle contracted the cutaneous affection on entering the water during the act of drinking. The water was, thus, supplied in troughs fed by pipes from the dams, which had been fenced in to prevent free access to them. The results were contrary to expectation and rather disappointing; the measure seemed to have no effect on the incidence of the disease.

Reservoirs and intermediary host. Very suggestive observations, concerning the infective stage of the disease, a probable vector and/or some intermediary host, have been made by some keen observant farmers in this vicinity. Based on their experience they surmized that besnoitiosis is transmitted during the febrile stage from an infected to a susceptible animal. (This observation has been confirmed experimentally.) The responsible vector incriminated is a bloodsucking fly, "die blindevlieg", (blind fly), a species of Tabanidae.

Others emphatically stated that cattle, coming into close contact with the warthog or the saddle-backed jackal suffering from a severe affection of the integumentum, contracted the disease. This statement proved to be fallacious. The dermatitis crustosa concerned was associated with sarcoptic and demodectic mange in the former and with a severe flea infestation in the latter case.

Auto-infection. A primary skin affection was suspected, but how it originated, was not indicated. Grooming and other forms of friction doubtless caused external re-infections. As the most intense infection occurred on the epiglottis and the tip of the soft palate, an alimentary infection was suggested. This particular area presumably became parasitised as a consequence of very little skin biting on the part of the host.[4] This view is untenable.

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By ingestion. In the earlier part of the investigation it was postulated that the superficially placed cysts in the nasal mucous membrane after rupturing formed the main source of infection by contaminating the supply of drinking water. Thus the likelihood of infection is remote at a running stream, proportional to the size of the dam and most favourable at drinking troughs. In the last case, it was concluded that the sporozoites are therefore not only likely to find a host, but what is more, may do so on the day of liberation.[3] On this basis the infectivity of grass and feed soiled with such discharges, though possible, would occur to a lesser extent.

At first sight this theory seems to provide a feasible explanation for the transmission of the disease, but on closer examination it proves to be unacceptable for the following reasons: If the above assumption had been correct, one would have expected to observe young developing forms in susceptible animals in an enzootic area the whole year round. This, as has been indicated, is not the case. The disease could not be transmitted to susceptible bovines in a "healthy" area, although they had been placed in close contact with heavily infected ones for a number of years. The latter were introduced from an affected locality. Undoubtedly, during that period the water supply and feed were exposed to contamination to a variable extent by parasites liberated not only from the surface of the nasal mucous membrane but also from that of the skin of these animals.

On the other hand, healthy stock, moved into an affected area during summer or winter, may contract the disease two to three weeks or several months respectively after their introduction. Again, where the disease is noted for the first time on a bushveld farm, there is usually a history of infected animals either being moved or straying on to such a property. It would seem that the malady spreads very slowly through a particular locality.

There is no doubt that the observations giving rise to the above theory of transmission are correct, but their interpretation and significance have been wrongly assigned. The collected data clearly indicate that the transmission must be ascribed to a chain of factors and is not merely due to direct contact with, or to the ingestion of, the parasite.

A hypothesis explaining the mode of transmission of *Besnoitia besnoiti* is volunteered. Presumably the bovine forms a reservoir for the infection, which is transmitted to other susceptible individuals by means of an intermediary host, possibly an arthropod vector (tick or bloodsucking fly species). Since the parasitic cyst is fairly deeply lodged in the tissues of the skin and the causal organism, either in its free or encysted form, can only be conveniently reached by a tick or insect with a fairly long proboscis, this mode of infection is favoured. Transmission may occur either directly by mechanical means or only after part of its life cycle took place in an intermediary host. The latter procedure is probably necessary to increase the potency and virulence of the organism enabling it to propagate better and to produce a severe natural infection. Striking is that all cases produced experimentally were very mild and could be easily missed, if the necessary pre-

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cautions to prevent this were not adhered to. One may assume thus that the direct mode of transmission, although possible, is not the usual one.

The relatively high incidence of the disease often encountered on farms where boreholes and troughs form the only source of drinking water is rather embarrassing at first. It need not be due to a severely contaminated water supply[3] but to the management methods in vogue on such farms. The development of, and the improvements on, the properties may give rise to local conditions suitable for the propagation of the suspected intermediary host. Cattle either coming to or resting in the vicinity of the water troughs may be exposed to a far denser fly infestation than those in areas where the water supply is not so confined. The incidence of the disease will depend either on the number of infected flies and/or that of diseased bovines. Of interest is that during an outbreak of nagana in the Magut area, Natal, during 1946, cattle became infected under similar conditions.

Pathology

The pathological anatomical changes vary according to their age, intensity and extent of the infection in individual cases. They are dominated by the lesions of the skin, of some of the mucous membranes (nasal, naso-pharynx, larynx and trachea), the cornea, the subcutaneous and interstitial connective tissue of the skeletal musculature and testes. The lymphnodes are generally swollen and the prescapular and the precrural are distinctly visible from a distance. In severe cases signs of cachexia (ascites, hydropericard, hydrothorax and atrophy of the skeletal muscles) and occasionally miliary, disseminated, necrotic myositis are noted. The testes of the bull, in the acute stage, are markedly swollen and turgid, whereas in the chronic obviously atrophied and firm.

The acute stage is characterized by a dull, staring coat, anasarca of a variable degree involving the lips, muzzle, face, ears, neck, brisket, abdomen, flanks, and the limbs. Erosions may occur on the lips and muzzle. Signs of an acute catarrhal or muco-catarrhal to purulent conjunctivitis and rhinitis as well as hyperaemia of the sclera are in evidence. The nares may be partially blocked by a discoloured and inspissated secretion, contributing to the respiratory distress observed in the initial stage of the disease.

In the subacute stage the anasarcous changes have visibly subsided, thickening, depilation, cracking, scurviness and crust formation of the skin are the most salient features. Fissures form especially on the flexor surfaces. A serosanguineous fluid oozes out forming scabs of variable size on drying. These lesions may become fly-blown and myiasis results. Decubitus develops in recumbent animals, if the necessary preventive measures are not adopted. Small greyish, opaque foci from pin-point to 1 mm. in diameter are observed on the cornea and sclera. These elevations are of considerable diagnostic value in determining, clinically, an infection of fairly long standing. In several animals the reddened or cyanotic nasal mucous membrane is studded with numerous similar foci. In addition that of the naso-pharynx,

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larynx and upper part of the trachea are variably infected. Particular attention should, thus, be paid to the eyes and nostrils of animals suspected to be suffering from this disease.

In the chronic stage, although the oedematous swellings have completely subsided, the skin is markedly thickened, wrinkled, scurvy (presence of fine flakes), and covered by scabs to a variable extent (fig. 1). Occasionally, owing to secondary bacterial or fungous infections, there is a pronounced focal hyperplasia of the superficial cutaneous layers, forming dense papilliform structures, often involving the scrotum or perineal region of bulls. In fact, the entire surface of the skin may be intensely affected. However, the eyelids and extremities tend to remain visibly thickened. The size and distribution of the depilated areas vary according to the dimensions and sites of the previous anasarca. Sometimes the surviving hair forms peculiar patterns on the body resembling the markings of a giraffe[3], presumably associated with focal vascular disturbances. The changes in the cutis and hair coat seem to be permanent in most of the severe cases, whereas nothing abnormal, except slight sclerosis of the skin and very little depilation, is noticed after the recovery from mild infections. In the latter case, the presence of the disease can only be determined definitely after portions of the skin have been examined histologically, whereas in the former, numerous parasitic cysts may be seen macroscopically at the periphery of the cut surface of the skin with comparative ease.

Histopathology

Prior to the beginning of 1950 very little headway was made with the histopathologic study because material only of advanced or "mature" infections was received for examination. For this reason, and our being biased too much by previously expressed views[4], the pathogenesis remained obscure. Thereafter, over a period of several years, suitable specimens, enabling one to study the infection from its earliest to its most advanced ("mature" or "dormant") stage in the bovine under natural conditions, were placed at our disposal.

During the febrile stage, after the infection has been contracted, the cutaneous lesions are the most characteristic and striking. Signs of hyperplasia of the epithelium, manifested by an increase of cells of the stratum spinosum, pronounced hyperaemia and oedema, especially of the corium, perivascular cell infiltrations, consisting of lymphocytes, plasma cells, fibroblasts and several large mononuclear cells (fig. 2) appear. The last mentioned cells may be confused easily with an occasional cell of sebaceous glands by the uninitiated. At the same time, after careful and painstaking search of various serial sections, free, crescentic forms can be demonstrated in the arterioles, lymphatics and in the spaces (lymph) between the collagenous connective tissue fibres (fig. 3). They propagate by binary fissure. Incidentally, the large mononuclears are situated in the same sites. After some time, the organisms penetrate the cytoplasm of these enlarged cells, become spherical or ovoid, and give rise to the proliferative form (fig. 4). Schizogony definitely does not occur. The parasites, multiply by multiple binary fissure and are enclosed in a distinct

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vacuole (fig. 5). Subsequently the nucleus of the host cell divides amitotically, giving rise to a variable number of nuclei and a large multinucleate cell results. Shortly after the invasion of the cell a fairly wide light blue homogeneous to vacuolated zone forms around it. All these cells need not necessarily be infected, and some of them degenerate relatively early. Eventually, a homogenous capsule develops and encloses the cell. At first, in a Giemsa stained section, it is basophilic and later eosinophilic, and is surrounded by a cellular, areolar connective tissue capsule (fig. 7). (Note the cell outline is clearly visible.) At this stage only intracellular, proliferative and no free forms of the parasite are noted. Occasionally, more than one parasite may penetrate the cytoplasm of a particular cell. Thus a variable number of parasitic cysts may develop in one host cell at the same time (fig. 6).

In the subacute stage the hyperaemic and oedematous changes in the skin subside but the inflammatory ones become more pronounced. This stage is also characterized by progression of the infection in the organs previously mentioned. The cellular response around the developing cysts remains chiefly mononuclear with a slight sprinkling of neutrophils. The cyst forms increase markedly in size. The rapidly proliferating parasites encroach more and more upon the cytoplasm and the nuclei are pushed toward the periphery of the cell (fig. 8). In addition, a variable degree of hyperkeratosis, crust formation (consisting of a fibrinous cellular exudate), a distinct superficial wrinkling, hyperplasia of the epithelium, an increase of fibroblasts partially replacing the collagenous connective tissue fibres, degenerate sebaceous and sweat glands and loss of hairs are manifestations of the skin lesions. The parasitic cysts seem to have a special affinity for areolar connective tissue, preferably of the superficial layers of the skin, of the mucous membranes (the nasal, naso-pharyngeal, laryngeal and that of the upper part of the trachea), of the male genital tract, of the skeletal musculature and of the lymphnodes, of the lungs, of the vascular system, of the bone and of the perineural tissues to a lesser extent. It was never found in the gastro-intestinal tract, in the central nervous system, and epithelium.

Chronic besnoitiosis refers to the stage in which besnoitia persist mainly in the mature or dormant cyst form. The number of cysts varies considerably, depending on the degree of the infection. In the milder cases, a few cysts, hair follicles and sheaths, some glandular tissue and cellular exudate is present, whereas in the severe cases the corium is completely replaced by enlarged cysts which are only separated by their hyalin capsules (fig. 9). These cysts have often been misnamed as "pseudocysts". They are considerably enlarged structures with a fairly wide capsule consisting of an outer fibro-cellular and an inner homogenous hyalin core enclosing a large parasitised cell. The cytoplasm of the host cell appears as a very thin membrane containing large round, ovoid or fusiform nuclei and a central parasitic portion composed of one (fig. 10) or several colonies separated by linear cytoplasmic septa (fig. 11). The nuclei, presumably, have a

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discoid structure. The structural characteristics of a cyst are similar to those previously described.

The nature of these large, vesicular, round and ovoid nuclei were, at first, not appreciated and, as on other occasions[4], [10], they were believed to be of parasitic rather than of host origin. However, they did not behave like a sporoblast and no chromatin continuity between them and the crescentic cyst forms could be recognized at any time. The conception that the parasite, retaliating the cellular attack of the host, forms a multinucleate capsule around the growing colony[4] is also untenable. *It is accepted now without reserve, that the nuclei originate from host cells and are not of parasitic origin.*

The epithelium of the skin either closely resembles that of the normal, except for increased hyperkeratosis and the presence of small scabs, or secondary micro-organism infection, pronounced epithelial hyperplasia and crust formation are evident. The superficial cutaneous lesions, in which streptothrix organisms can be demonstrated, are consistent with those described for Senkobo disease (streptothricosis) in cattle.[11]

Occasionally, owing to the atrophy of the enclosing epithelium, superficially placed mature cysts in the skin or the nasal mucosa may rupture to the exterior. Either free parasites, or parasites phagocytosed by neutrophils are observed in scabs on the skin or in the nasal secretions respectively. Similarly, cysts may break into hair sheaths resulting in a folliculitis. By these means crescentic organisms may be shed to the exterior. It is striking that intact cysts appear to elicit slight or no chemotactic response and may persist for the life-time of the host.

It will be noted that the intensity of the cellular reaction around the cysts varies considerably. Cellular elements may be absent, slight or fairly pronounced. The latter condition develops in the vicinity of degenerating and/or ruptured cysts. Cyst rupture, depending on the stage it is observed, is followed either by necrotizing inflammatory or mild granulomatous reactions. In the early stage the degenerate hyalin capsule ruptures at the pole with the densest cell accumulation. Numerous parasites are set free and have a tendency to remain clumped together, occasionally free single forms are found in their vicinity, but not in the cellular exudate some distance from the ruptured cysts. The presence of the cellular reaction enables one to differentiate between a burst cyst and an artefact resulting during the sectioning. There is an appreciable increase of eosinophils in this vicinity in addition to lymphocytes, plasma cells, and multinucleated cells which, however, do not reach the size of infected ones. On no occasion have parasites been seen to penetrate or develop in their cytoplasm. Presumeably these organisms are either phagocytosed by neutrophils or destroyed by lysis. Subsequently, cell elements invade the partially vacated cyst cavity through the breach. After the remaining parasites have been cleared up, the hyalin capsule collapses irregularly and is filled either by granulation or scar tissue. The changes in the degenerating cysts, vary according to the stage of disintegration. In some the

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contours of the protozoa become indistinct, their cytoplasm has either a basophilic or eosinophilic tint, and the nuclei are pyknotic. A homogeneous eosinophilic mass is formed in the more advanced stages. Folliculitis, panniculitis, focal disseminated myositis, keratitis, periostitis, endostitis, lymphadenitis, pneumonia, periorchitis, orchitis with epididymitis, arteritis, and perineuritis on this basis have been seen in individuals to a variable extent.

The previously mentioned increase in the number of the eosinophils probably indicates a hyperergic state caused by absorption of the breakdown products of the degenerate and ruptured cysts. Possibly other factors such as infestations by ticks or filaria larvae, or reinfections of besnoitia may contribute in producing such allergic conditions. *Thus the production of lesions is presumably not only determined by the number of cysts, their tendency to degenerate or rupture and their organ distribution, but also by the other contributary causes mentioned.* Sometimes demodectic infestations, involving the sebaceous glands of the eyelids and occasionally those of the skin, produce either only mild or no reactive changes. On account of this, these parasites probably do not contribute to the production of these marked exudative lesions in the skin.

The changes in the testes and the epididymis may be briefly stated. They vary in individual cases. In some of the testes pronounced signs of atrophy, aspermatogenesis, perivascular "cuffing" and a variable number of mature cysts, with or without a cellular infiltration, are evident. In the corresponding epididymis the tubules show catarrhal changes and metaplasia of the epithelial lining to a variable extent. The lumens of the tubules contain no spermatozoa. In other testes spermatogenesis is still in evidence, but reduced, and the sperms are degenerate to a variable degree. The tubules of the epididymis are empty or partially or totally filled with degenerate spermatozoa clumped together or may contain some exudate.

As the condition has been confused with other skin diseases, and unspecific dermatitis, the diagnosis can only be clinched by examining scrapings, smears and sections of skin biopsies.

Course. Besnoitia infection passes through an acute, a subacute and chronic phase, which may or may not be followed by partial or complete recovery, except that lesions involving the limbs and/or muzzle tend to remain permanently. The period elapsing depends on the degree of the infection and ranges from several weeks to years. The rate of its progression varies in individual cases. Lesions accompanying these stages of infection are sometimes asymptomatic. Frequently, however, they are of sufficient magnitude to result in clinical symptoms or even death, ascribed to asphyxia or cachexia, in any of these phases.

Treatment

Up to now all attempts to treat the disease have failed. The claim of some farmers that beneficial results have been obtained by the application of benzene hexachloride preparations and/or used motor oil could not be substantiated. Presumably the condition would only

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be amenable to treatment during its initial stage when the causal organism is still in its free phase.

Preventive Measures

Since the disease is not transmitted by direct contact, the adoption of quarantine measures would prove of no avail in preventing the spread of the disease. Contaminated feed or drinking water presumably need not be incriminated. Since cattle, grazing in a particular area, are apt to become affected, and the disease has a definite seasonal incidence, one may assume the presence of an arthropod transmittor or intermediary host, most likely a biting or bloodsucking insect ("blinde vlieg"). A procedure, eliminating the source of the infection (diseased bovine) and/or the vector, should be adopted. It probably would prove to be the most efficient method of combating the disease successfully. At present it is essential to slaughter all severely affected cattle as soon as possible. On farms where this measure has been adopted and adhered to, the incidence of besnoitiosis in cattle seems to have been reduced considerably. Attempts should be made to determine the suspected vector definitely in order that the remaining link may also be eliminated.

DISCUSSION AND CONCLUSION

As in the case of the horse, although parasites may be liberated from certain cysts and may penetrate some distance into the surrounding tissues and various stages of fibrosed cysts are present, no young developing forms could be found in any of these sections. Presumably, therefore, these organisms degenerate or are phagocytosed and apparently play no rôle in new cyst formation. Possibly a hyperergic condition is produced, giving rise to the cellular reaction and scab formation. The mature cyst in the tissues may be regarded as a "resting" or "dormant" phase of the parasite; the organisms developing further as soon as suitable conditions arise. The possible transmission by an intermediary host (biting or blood sucking arthropods), most likely a blood-sucking species of Tabanidae, must be considered as previously suggested in the case of the horse[2].

An extracellular and an intracellular form of the parasite occurs. Two types of the former are seen in the tissues. The proliferative one, appearing during the initial stage of the disease, is responsible for its spread throughout the animal body, whereas the spores freed from ruptured cysts either degenerate or are phagocytosed, and an inflammatory reaction with an apparent increase of eosinophils results. In this respect it differs from Toxoplasma where the organisms liberated enter other cells and the proliferative cycle is repeated in susceptible, non-immune hosts[10]. Finally, in a besnoitia infection only one particular developmental phase of the intracellular (cyst) organism, depending on the age of the affection, is observed in any one individual. This is rather striking, especially where reinfection could not definitely be excluded. The signs of the chronic dermatitis crustosa are not pathognomonic for *Besnoitii besnoiti* but may probably be evoked by

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other infections, such as a severe infestation with tick larvae, the presence of microfilaria, or a reinfection, occuring concurrently.

The term cyst wall nuclei is rather confusing. These structures are definitely of host but not of parasitic origin as previously stated. They are really the compressed nuclei of the infected multinucleate host cell. The thin membrane, enclosing the mature protozoan colony or colonies, is all that remains of its cytoplasm, and has nothing in common with that of the Coccidian oocyst. This form of parasitism is known as symbiosis.

SUMMARY

The macroscopic and histological changes and the life cycle of besnoitiosis in cattle encountered in the Union of South Africa are described in some detail. The theories of transmission are discussed and a hypothetical mode of transmission is volunteered, part of the life cycle of the parasite being passed in the bovine and the other in an intermediary host. Mechanical transmission, although possible, is not regarded to be the usual mode. The symptom complex, the geographical and topographical distribution, its incidence and factors such as age, sex, and breed, which may have some bearing on it, and the preventive measures are briefly dealt with.

ACKNOWLEDGEMENTS

The writer wishes to express his gratitude to the various colleagues in the field for their kind assistance in supplying the necessary material and information of various cases for these investigations, and to Miss G. E. Laurence who was responsible for the excellent illustrations figs. 2 to 8 inclusive.

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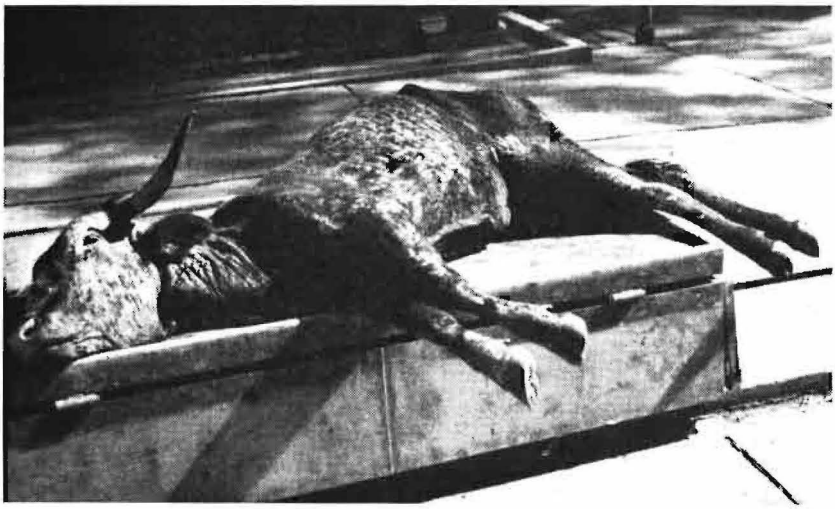


Fig. 1. Severe case of Besnoitiosis.

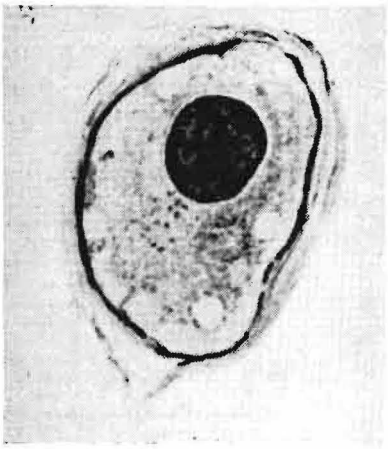


Fig. 2.
Large mononuclear cell.
Path No. 38784.



Fig. 3.
Free parasites in vicinity of large mononuclear cell. Path. No. 38784.

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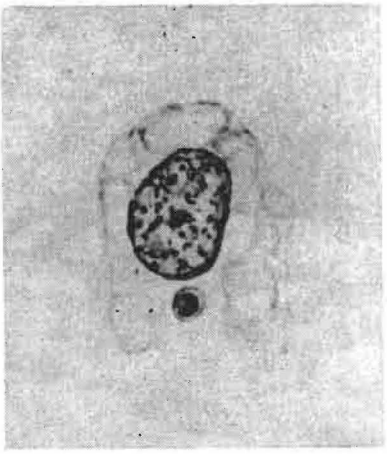


Fig. 4.

Parasite in cytoplasm of large mononuclear cell. Path. No. 38784.

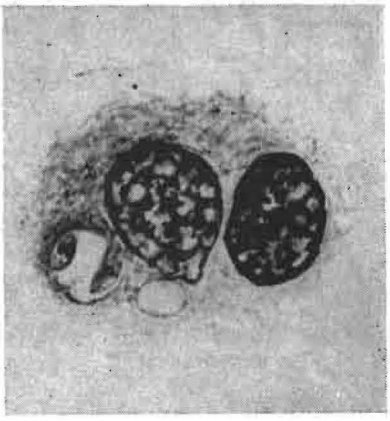


Fig. 5.

Division of parasite and of cell nucleus. Path. No. 38784.

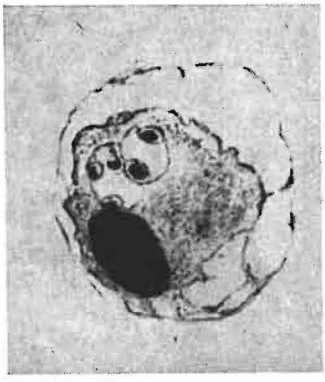


Fig. 6.

Two cysts developing after two separate parasites penetrated into cell.
Path. No. 38784.

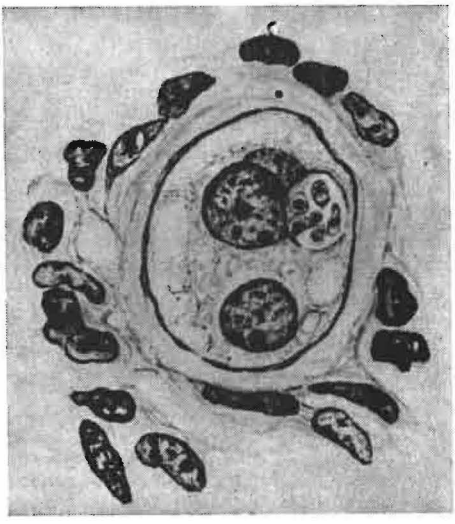


Fig. 7.

Slightly advanced stage of development. Note distinct cellular membrane, hyalin border and areolar connective tissue capsule.
Path. No. 44734.

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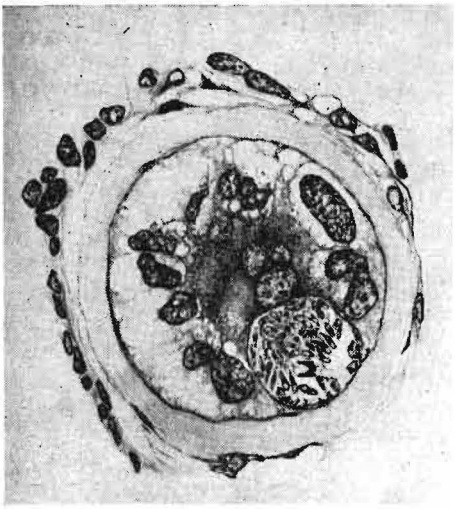


Fig. 8.

More advanced development in subacute stage. Note clear outline of parasite cyst and of multinucleate host cell, and hyalin capsule. Path. No. 44589.

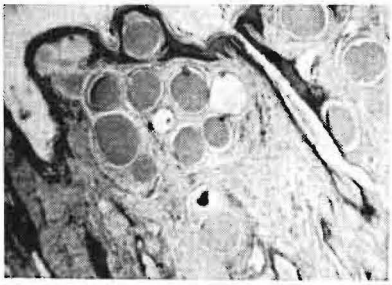


Fig. 9.

Heavy infection in the cutis of mature cysts. Note: no cellular infiltration. Path. No. 44716.



Fig. 10.

Fully developed (mature) cyst. Note: no cellular reaction and hyperkeratosis. Path. No. 44716.

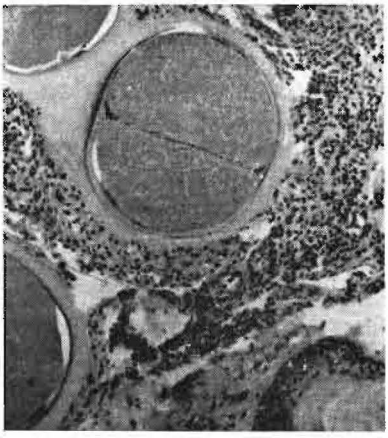


Fig. 11.

Two separate mature cysts in one multinucleate host cell. Path. No. 43540.

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PRELIMINARY OBSERVATIONS ON THE MECHANICAL TRANSMISSION OF CYST ORGANISMS OF *BESNOITIA BESNOITI* (MAROTEL, 1912) FROM A CHRONICALLY INFECTED BULL TO RABBITS BY *GLOSSINA BREVIPALPIS* NEWSTEAD, 1910

R. D. Bigalke,*

During the course of infection with *B. besnoiti* two distinct successive stages in the development of the parasite may be observed in the vertebrate host. Proliferative forms—also referred to as proliferative organisms or trophozoites[14]—are to be found extracellularly or intracellularly, chiefly in monocytes, in the blood and various organs during the febrile stage of the disease. These disappear from the blood and tissues after cessation of the fever reaction, and cysts develop from parasitized hypertrophying histiocytes situated in the dermis of the skin etc.[16], each cyst eventually containing thousands of cyst organisms. These cysts, which were described by Besnoit and Robin[2], remain largely intact as long as the animal lives,[14] thus constituting a chronic infection.

Although Cuillé, Chelle and Berlureau[5] succeeded in transmitting bovine besnoitiosis by subinoculation of blood during the febrile reaction, it was not until 1954 that Pols[14] found that proliferative organisms were responsible for the transmission.

Attempts by Besnoit and Robin[2], Franco and Borges[9], Cuillé and Chelle[6] and Pols[16] to transmit cyst organisms artificially were apparently without avail. Bigalke[2] found that rabbits developed typical besnoitiosis after having received cyst suspensions prepared from infected skin or tissues from chronically infected cattle by parenteral routes. Cattle infected in the same way developed a mild form of the disease. These observations were of particular interest from the epizootological point of view, because evidence was brought forward that the chronically infected animal, harbouring millions of cysts of *B. besnoiti* in the dermis of the skin, nasal mucous membranes, etc., might well prove to be a reservoir of the disease.

Studies on the epizootology of bovine besnoitiosis in Southern France by Cuillé and Chelle[7] led them to believe that an intermediate host was involved in the transmission. They also stated that the introduction of early, and especially chronic cases onto non-infected farms, led to new outbreaks. Barrairon[1] and Herin[11] suggested that the disease was transmitted by an arthropod, on account of its seasonal incidence.

Arthropod vectors may transmit diseases either biologically, mechanically, or by both methods. Only mechanical transmission was investigated in this study. Theoretically this could take place

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during the transient period of parasitaemia or during the life-long stage of cyst involvement of the skin. Pols[16] recorded transmission of proliferative organisms by syringe needle in rabbits, thus indicating that an arthropod might do likewise. However, even if this did take place in nature, he could not explain how the infectious agent survived the interepizootic period. The answer to this question may lie within the cysts of *B. besnoiti*.

Flies of the family Tabanidae are rather common on most farms in the enzootic regions of Transvaal during the summer months. Their ability to transmit diseases mechanically is common knowledge. Although an obvious choice, they were rather difficult to obtain at Onderstepoort in large numbers, and the few that were caught refused to feed. It was therefore decided to use *G. brevipalpis* as an example of a biting fly. They were easily obtainable and, being exceptionally keen feeders, were ideal for the purpose required.

Material and Methods

General

Pupae of *G. brevipalpis* were obtained from Zululand through the kindness of Dr. R. du Toit of Onderstepoort, and Dr. E. B. Kluge of Nongoma. After emerging the tsetse flies were placed individually in 4 oz. glass jars covered with gauze. They were given an opportunity of feeding on the ears of two non-infected rabbits at least every second day when not being used for transmission work. The flies were kept in an insectarium in which the temperature was 26°C and the relative humidity 80%

The experimental rabbits were housed in separate cages and rectal temperatures were taken daily. Bloodsmears were examined daily when a febrile reaction or other sign of disease was noticed.

The "Fuls" and "Schoeman" strains of *B. besnoiti* were used indiscriminately in immunity tests, as complete cross-immunity has been found to exist between all strains of the parasite isolated in South Africa[4].

Experiment I

Attempted transmission with cyst organisms (Table I).

A four year old Afrikander bull (130), that had suffered from acute besnoitiosis two years previously, was used as donor. Cysts were very numerous in the dermis, and it was noticed that cyst organisms were frequently present in blood smears made from the tip of its tail

The hair on the posterior portion of the tail was cut very short with a pair of scissors. Then the gauze-covered end of a glass jar containing a tsetse fly was pressed against it, and when the fly had inserted his proboscis and was apparently feeding, the jar was rapidly transferred to the shaved abdomen (only rabbit 2668) or ears of a recipient rabbit. Considerable probing sometimes took place before the tsetse fly settled down to feed, especially on the tail of bull 130. Undelayed transfer from donor to recipient was repeated until the fly had

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TRANSMISSION OF CYST ORGANISMS FROM A CHRONICALLY INFECTED BULL TO RABBITS

Rabbit	Donor	Tsetse Flies		Reaction						Biological test		Remarks
		Number	Totalno. undelayed alternate feeds	Incubation period in days	Febrile reaction		Trophozoites		Cysts in skin	Rabbit	Result	
					Length in days	Maximum	Blood smears	Organ smears				
2668	Bull. 130	4	17	8	—	—	+++	+++	n.e.	n.c.	—	Died after 2 days; no febrile reaction
2677	do.	4	18	10	3	104.8	Neg.	n.e.	Neg.	2694 2695	Pos. Pos.	Solidly immune
2678	do.	9	17	11	5	104.6	+	n.e.	+	n.c.	—	—
2683	do.	6	20	11	2	104.4	+++	+++	n.e.	n.c.	—	Died after 2 days

Legend: n.e. = not examined
n.c. = not conducted
+ = rare
++ = fairly frequent
+++ = frequent
Pos. = Positive
Neg. = Negative

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engorged (for details see Table I). Smears were made from the drops of blood which frequently exuded from the bite wounds on the tail of bull 130 and examined microscopically for cyst organisms. In the case of rabbit 2678 two flies were fed on the one day and the rest 24 hours later. In all other cases feeding was completed in one session.

5ml. blood from rabbit 2677 was injected subcutaneously into rabbits 2694 and 2695 to prove that the mild febrile reaction shown by the former was due to *B. besnoiti*. 43 days later rabbit 2677 was challenged with infective rabbit blood ("Schoeman" strain).

Skin specimens from rabbits 2677, 2678, 2694 and 2695, which recovered, were examined histologically for the typical thickwalled cysts of *B. besnoiti*.

Experiment 2.

Attempted transmission with proliferative organisms (Table II).

Rabbits showing moderate to fairly marked parasitaemia were used as donors. It must be stressed that proliferative forms are never very frequent in the blood, a single parasite per microscopic field representing a very severe infection.

Rabbit 2683 was infected mechanically by *G. brevipalpis* in Experiment 1. The other donors were all infected artificially with the "Fuls" strain, which is being maintained in rabbits by serial passage of 5ml. blood inoculated intraperitoneally.

Tsetse flies were fed alternately on the outside of the ears of the donor and the recipient as in the first experiment. Blood smears were made from the ears of the donors prior to using them, in order to determine the degree of parasitaemia.

Experiment 3

As it was essential to know if cyst organisms present on the mouthparts of the tsetse flies used in the above experiments remained viable for 48 hours or longer, two rabbits (2685 and 2690) used for routine feeding of these flies, subsequent to their use above, were kept under observation. 39 and 54 days respectively, after the last flies had been fed on them, their immunity was challenged with virulent blood.

Results

Experiment 1 (Table I).

All four rabbits contracted the disease. Two of them died from a peracute form, whereas the other two suffered a mild attack, so mild in fact that only one intracellular proliferative organism could be found in a bloodsmear made from rabbit 2678 on the fifth day of the febrile reaction after searching for half an hour. A few typical cysts of *B. besnoiti* were found in the dermis of the skin of the lips, base of the ears and the vulva, providing further proof of the identity of the parasite that had been transmitted.

In the case of rabbit 2677 no proliferative organisms could be found in bloodsmears, or cysts in skin sections of the lips, but trans-

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TABLE II

ATTEMPTED TRANSMISSION OF TROPHOZOITES FROM RABBIT TO RABBIT

Recipient	Donor			Tsetse flies		Result	Immunity test
	Rabbit No.	Strain and Generation	Parasitaemia	Number	Total No. undelayed alternate feeds		
2679	2652	Fuls Gen. 199	+	3	12	Neg.	Fully susceptible
2680	2652	Fuls Gen. 199	++	3	8	Neg.	do.
2689	2669	Fuls Gen. 200	++	9	28	Neg.	do.
2693	2683	see Table 1	+++	9	37	Neg.	do.

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mission was confirmed by a positive biological test. In addition rabbit 2677 was found to be solidly immune when challenged. Rabbit 2683 developed a moderate oedema of the vulva.

Apart from small haemorrhages which formed immediately after withdrawal of the proboscis, no tissue reaction around the bite wounds was observed ever.

Numerous cyst organisms of *B. besnoiti* were found in the majority of smears made from drops of blood exuding from puncture wounds on the tail of the donor, proving that the haustellum had penetrated the cyst walls without difficutly.

Experiment 2.

All four rabbits failed to contract the disease. On testing their immunity with infective blood, they were found to be fully susceptible (Table II).

Experiment 3

The two rabbits used for routine feeding of tsetse flies did not react to besnoitiosis until they were challenged with infective blood. Although no attempt was made to determine exactly how long cyst organisms of *B. besnoiti* remain viable on the proboscis, it has been shown that tsetse flies may be regarded as clean 48 hours after feeding on a chronically infected bovine.

Discussion

These experiments have shown that G. *brevipalpis* is capable of penetrating the relatively thick (8–35 microns) wall of the *B. besnoiti* cyst by means of its haustellum whilst probing in the skin in search of a blood meal. In this way the haustellum becomes contaminated with cyst organisms which may be transferred mechanically to a susceptible rabbit. Although cattle may be infected in the same way it does not necessarily follow that they will develop the typical symptomatology seen in natural cases. Reactions may be as mild as those observed repeatedly in experiments on the artificial transmission of proliferative[14] and cyst organisms[3] of the parasite.

Tsetse flies do not occur in the bushveld and lowveld regions of Transvaal, where the disease is enzootic. Hence they cannot be involved in the transmission of the disease in South Africa. However, other biting flies such as Tabanidae, Stomoxinae and some biting Muscinae are plentiful in these regions. Of these, the Tabanidae with their stout and sometimes exceptionally long mouthparts (e.g. *Pangonia*) deserve primary consideration.

The feeding mechanism of *Haematopota* has been studied by Dickerson and Lavoipierre[8]. It will suffice to mention that a pair of mandibles and a pair of maxillae are present which effect entry into the skin and laceration of the tissues by means of their cutting and rasping actions. A blood pool is formed as result of the rupture of large and small blood vessels, from which the fly feeds. The same principles probably apply to the other Tabanidae. Mandibles and

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maxillary blades are absent in *Glossina* and penetration of the skin takes place by means of the labellar teeth and rasps[10]. Once the probing haustellum lacerates the walls of one or more capillaries, feeding commences from the stream of blood escaping from the damaged blood vessels. As the haustellum of *Glossina* is quite capable of penetrating cysts of *B. besnoiti*, it is perfectly reasonable to assume that the same applies to the mouthparts of the Tabanidae.

Piercy[13] has remarked on the painful bite-wounds inflicted by Tabanidae when attempting to feed, leading to disturbance of the fly by the host, and making these flies particularly suitable for mechanical transmission of diseases such as anaplasmosis, anthrax, trypanosomiasis, and therefore possibly also besnoitiosis.

Numerical considerations are probably of particular importance in nature when the question of mechanical transmission of a disease arises. In this particular case it is reasonable to assume that the chances of the proboscis encountering a cyst are directly proportional to the number of cysts present in the dermis of the carrier, the number of flies attacking the carrier, the number of carriers present on a farm and the number of times flies are interrupted while feeding on the carrier. Howell *et al*[12] recorded that 13 undelayed transfer feedings from clinical cases of anaplasmosis, was the smallest number that resulted in transmission of the disease by *Tabanus oklahomensis*. This figure is comparable to the number used in these preliminary experiments (Table 1).

If mechanical transmission of bovine besnoitiosis by cyst organisms does take place in nature, it should be possible to control the disease very effectively by eliminating the chronic carriers, on the assumption that no other animal is involved as reservoir in nature. Field experiments on infected farms from which all chronically infected cattle are removed would provide useful epizootological data in this respect.

Although the preliminary experiment on the mechanical transmission of proliferative forms of *B. besnoiti* was negative, further attempts will have to be made before final conclusions can be drawn.

Summary

1. The mechanical transmission of cyst organisms of *B. besnoiti* from a chronically infected bull to four rabbits by undelayed transfer feedings with *G. brevipalpis* is recorded.

2. The failure of mechanical transmission of proliferative organisms of *B. besnoiti* from acutely infected to susceptible rabbits by the same methods used above is recorded.

3. The mouthparts and methods of feeding of *Glossina* and *Haematopota* are compared briefly with reference to penetration of *B. besnoiti* cysts.

4. Control measures, which would provide valuable epizootological information if tried out, are suggested.

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THE RÔLE OF INFESTED DUNG IN THE TRANSMISSION OF NEMATODE PARASITES OF CATTLE

R. K. Reinecke*

With few exceptions the initial free-living stages of parasitic helminths of cattle take place in the dung. This paper draws attention to the important rôle played by dung in the transmission of some of the common nematodes of cattle, viz. *Haemonchus placei*, *Cooperia pectinata* and *C. punctata*, *Oesophagostomum radiatum* and *Bunostomum phlebotomum*. Gordon[5] suggested the format of this paper, the protocols for which were collected at Armoedsvlakte, Vryburg, and are being published elsewhere[7].

Basically two factors are important; the dung pad and animal husbandry. These will be discussed separately; the former from the natural course of events; the latter, the unnatural sequence as a result of husbandry methods.

1. *The Dung Pad*

(*a*) Incubator.
(*b*) Shelter.
(*c*) Reservoir.
(*d*) Prison.
(*e*) Destruction and dispersion.

(*a*) *Incubator*

The dung pad acts as a culture medium in which the eggs hatch; the hatched larvae grow, feed and live in the faeces and after two moults reach the infective stage. During this process three larval stages are recognisable; the first and second or pre-infective stages which grow in and feed on the faeces, and the third stage known as the sheathed or infective larvae. The infective larvae retain the sheath of the second moult and they neither feed nor grow in the dung. Various climatic factors influence egg hatching and the subsequent development to the infective stage.

Oxygen

A certain amount of oxygen is required for egg hatching and larval development, and apparently enough air is trapped in the dung for this to take place. The burrowing habits of dung beetles speed up egg hatching by introducing air, which will be discussed in more detail later.

Temperature

A very profound effect is exerted by the environmental temperatures. From spring to autumn, temperatures occurring at Armoedsvlakte were high enough for egg hatching and larval development to the infective stage. On an average all eggs had hatched within 48 hours; first stage larvae were present in large numbers by the third day; the next stage two days later, and infective larvae by the seventh day.

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In midsummer infective larvae were recovered by the fifth day but in spring and late autumn this was delayed to the eighth or ninth day under conditions of climate experienced at Armoedsvlakte, Vryburg.

On the other hand Durie[3] has shown that the dung absorbs solar radiation and in this respect acts as a "black body". In Queensland temperatures in summer of 122 °F and higher have been recorded in dung pads. This worker has shown that eggs and pre-infective larvae can be destroyed at temperatures of 115°F within four hours. Temperatures frequently exceed 100°F in the shade at Armoedsvlakte and are considerably higher in the sun. Some of the infested dung pads examined for infective larvae in midsummer at Armoedsvlakte were negative. This was probably due to the effects of high temperature as a result of solar radiation.

In midwinter egg hatching and larval development were delayed. Crofton[1] has shown that neither egg hatching nor larval development can occur below 10°C; these processes under field conditions occur in a normal fashion at temperatures of 18°C (65°F) or higher according to Dinaburg[2], Gordon[4] and Roberts, O'Sullivan and Riek[8]. At Armoedsvlakte screen temperatures seldom exceeded 15°C before 10 a.m. or after 4 p.m. in midwinter. In Queensland, Durie and the author showed that temperatures in the dung only exceeded 15°C from 8 a.m. to 6 p.m. in midwinter. Thus optimal conditions for development will only occur for as little as six to ten hours every day in the areas mentioned above, where the trials were carried out. It is not surprising, therefore, that at Armoedsvlakte in midwinter infective larvae were not yet present in dung three weeks after exposure to field conditions.

Evaporation

Under the semi-arid conditions of the North Western Cape dung pads dried out rapidly from spring to autumn. Within 48 hours they had frequently lost half their weight, due to evaporation of moisture. Once the outer crust had formed the process slowed down; however, some dung pads lost 85 per cent of their weight within five days due to evaporation.

As mentioned previously the first and second larval stages showed optimal development on the third and fifth day. These larvae must have moisture for survival. If dung pads lost more than 65 per cent of their weight within the first five days less than 0.27 per cent of the hatched larvae reached the infective stage. This high mortality rate of the pre-infective stages, was largely due to death by desiccation, brought about by evaporation of most of the moisture in the dung pad.

In winter this process was not nearly as rapid nor as marked as it was in summer. However, the low temperatures were more important in larval development.

Rainfall

The moisture present in dung pads is adequate for some of the hatched larvae to develop to the infective stage even in the entire absence of rain. This has been observed by Mönnig[6] and Roberts

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et al.[8] Although confirmed by the writer's observations, a very small percentage (0.2 per cent) only, were able to reach the infective stage. In the critical period, when the pre-infective larvae are present in large numbers, i.e. the second to fifth day, these sensitive larvae were shown to survive in much larger numbers if rain fell. Thus if rain fell at least once, but preferably more frequently, between the second and sixth day as many as 13 per cent of the hatched larvae reached the infective stage.

The climatic factors which have their main affect on the free-living stages of these nematodes in the dung are temperature, evaporation and rainfall. Optimal development will occur where the high temperatures brought about by solar radiation do not apply, e.g. where dung pads lie in the shade. Furthermore, if rain falls at least once but preferably more frequently between the second and fifth or sixth days, it will counteract evaporation and save many larvae which will have died by desiccation. It is, therefore, quite obvious what an important effect the climate has on the dung in its rôle as an incubator for the free-living stages.

(*b*) *Shelter*

As the dung's mass in itself is considerable when compared with the very small mass of eggs and developing larvae, even in the most heavily infested faeces, it acts as a shelter to these developing stages. As evaporation takes place a hard outer crust forms which, particularly if baked by the sun, forms an almost impervious outer covering. This assists in moisture retention, so essential to the developing larvae below the crust. It was found that the largest number of larvae were present beneath the crust and above the surface lying on the soil.

In other words, the crust acts as the roof of a house would, protecting the inhabitants within from the more harmful effects of adverse climatic conditions. Once formed, the hard crust also protects the depth against possible damage by mechanical means (e.g. trampling by livestock, etc.).

(*c*) *Reservoir*

The dung pad acts as a well protected reservoir of infective larvae. Under the hot, frequently dry summer conditions, their life in those dung pads was fairly limited, and their numbers fell sharply 24 days after fresh dung was placed in the veld. In the autumn they survived for long periods, most species being recovered 105 days after dung was placed in the veld. Only the infective larvae of *B. phlebotomum* were unable to survive, dying within a period of 24 days.

Durie,[3] working in Queensland, has stated that the dung pad acts as reservoir of infective larvae for periods of as long as five months, the numbers decreasing with age. We could expect similar conditions to apply to semi-tropical parts of the country with high rainfall, e.g. Eastern Transvaal Lowveld.

(*d*) *Prison*

Although the hard impervious crust protects the larvae, it also imprisons them. To infect the bovine host they must migrate from the dung pad. This migration only occurs when preceded by rain,

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as it is certain that larvae can only migrate when a thin film of moisture is present on the migrating surface.

A short sharp shower is insufficient to cause migration of any note, as it only wets the hard, dry crust where very few larvae are present. If moisture penetrates the crust and reaches the depth where larvae are present, they are unable to migrate through the crust. Thus it was shown that rainfall up to 19 mm. did cause migration but this was downwards onto the surface of the soil below. Rainfall in excess of 20 mm. caused lateral migration to the surrounding grass.

It appears, therefore, that once the moisture has penetrated the dung's crust the larvae below the crust are stimulated to migrate. The impervious crust, although wet, has a physical consistency which prevents the larvae from migrating outwards or upwards through it. The larvae, therefore, take the path of least resistance and leave the dung through the softer more porous layers on the lower or ground surface. If rainfall has been adequate and there is a film of moisture on the soil beneath the dung, the larvae are able to migrate horizontally until they reach the first grass blade. Thereafter they migrate vertically. Horizontally larvae were recovered 80 cm. from the edge of dung pads and more than 20 cm. above the soil surface on grass.

Durie[3] has observed horizontal migrations of more than 6 ft. within 24 hours of suitable rains falling. In the laboratory he has shown that larvae can migrate vertically as much as 10 inches in 4 hours. He has also observed that the dung pad acts as a reservoir of larvae for many months, releasing the larvae in waves as it were, over a period of as long as five months. Each wave is preceded by suitable conditions of rainfall, the number of larvae released decreasing with each succeeding wave of migration.

(*e*) *Destruction and Dispersion*

Dung beetles and other arthropods were very active in dung from summer to late autumn. At times dung pads were completely destroyed by their activities. They facilitated egg hatching by increasing the aeration of dung; the hatched larvae, however, found it more difficult to survive, as the already high rates of evaporation were accelerated in hollowed out dung pads and larvae died off rapidly in the dry remnants of the pad.

Some of the dung beetles removed the dung to the surrounding soil. In these small pieces of dung a few larvae developed. In this way infestation was spread by the mechanical removal of small pieces of infested dung from the pad. Generally it can be stated that these beetles decreased the infestation enormously by virtue of their effect on the dung pad.

Under natural conditions a certain logical sequence of events takes place which has been described briefly above. What is the position, however, where man superimposes husbandry methods on his livestock?

2. *Animal Husbandry*

(*a*) Aggregation of eggs and larvae.
(*b*) Concentration of susceptible animals.

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(*a*) *Aggregation of eggs and larvae*

Due to the concentration of cattle in kraals,* pens, watering places etc. vast quantities of manure are deposited in small areas. This is particularly noticeable in the semi-arid regions without surface water of any kind. The first stage of farming operations is to seek and find water. Windmills and engine driven pumps draw water from great depths. Livestock walk many miles from the grazing to the water every day. Throughout the North Western Cape, with rainfalls varying from 10–20 inches annually, isolated water supplies are vital for survival. From about 8 or 9 a.m. the cattle come to drink; they remain there during the heat of the day, returning to the grazing at about 4–5 p.m. Only after heavy rains will they find sufficient water elsewhere to supply their drinking requirements.

As a source of infestation, kraal manure acts in a different fashion to dung pads in the veld. Under dry conditions normally present in winter and spring, they are practically free of infective larvae. The reason for this is fairly obvious. Fresh dung has hardly fallen before it is trampled underfoot, and mixed with the dry accumulations of dung which form the floor of these kraals. If the eggs are not killed by the high temperatures in the unshaded portions of the kraal, the hatched larvae soon die from desiccation. Repeated examinations of kraal manure, in the winter and spring at Armoedsvlakte, were negative for infective larvae.

An entirely different pattern emerges in the rainy season, particularly if the rain falls every few days. At first the dry surface manure absorbs all the moisture; the subsequent rains soak the lower layers of manure and the following sequence of events takes place. Evaporation from the surface cools the mass of manure. The trampling mixes the fresh and old manure. This creates an ideal culture medium in which eggs hatch rapidly, temperatures and oxygen being adequate; the moisture content is more than adequate for larval development; the soft surface layers which dry first, act as a blanket would and protect the lower parts from evaporation and high temperatures. In addition new infestation is constantly reintroduced because the herd spends a quarter to a third of every day in a very confined area. Millions of infective larvae develop, reaching a very high level within seven to ten days of the first rains. Such areas are highly dangerous and can rightly be claimed to be poison spots on any farm.

(*b*) *Concentration of susceptible animals*

On most dairy ranches calves are kept in pens until the cows are milked and then the calves suckle on their dams in the kraal. At Armoedsvlakte calves kept in this fashion failed to become infested in the dry winter and spring. In the rainy season, particularly in a wet summer, they became heavily infested.

The general practice on dairy ranches is to confine the calves to calf pens in the morning and allow them to suckle after milking the

* Kraals = cattle pens.

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cows in the kraal; overnight they graze in a small calf paddock. In the rainy season these calves become very heavily infested. Every mouthful of food is a potential source of infestation. They suckle on teats contaminated with kraal manure and graze in infested paddocks; in addition they are forced to stay in infested kraals for six to eight hours every day.

In a dry summer and autumn where only 290 mm. (11.5 inches) of rainfall was recorded, calves grazing in the calf paddock and suckling in kraals were by far the most heavily infested group. Calves confined to the paddock and suckling on their dams there acquired less than a third of the infestation of the previous group.

These husbandry methods are artificial methods of rearing calves. The most popular method of calf rearing in the area proved to be the worst method from the point of view of spreading infestation among young stock. The paddock could not really be considered as natural grazing. It was heavily overstocked and covered with a high concentration of dung pads. The previous summer's calves grazed with the newly born calves, not only through the winter, but into the following summer. They carried the infestation from the previous rainy season to the next rainy season and started off the infestation in the paddock, which was promptly acquired by the highly susceptible new-born stock. The kraal being the common meeting place of all age groups, was a highly dangerous area during the limited rainy season for any calf that had the misfortune to be confined there.

Control

It is extremely simple to control parasites on a dairy ranch by applying a few sound principles of animal husbandry. They are briefly as follows:

(1) *Hygienic kraals and calf pens*

The provision of concrete floors, cleaned daily, in all calf pens, kraals and cow byres is all that is necessary to eliminate this source of infestation.

(2) *Grazing paddocks for calves*

At least three but preferably four grazing paddocks should be provided for calves. Any rotation method suitable for the growth of the pasture can be applied in the dry season, i.e. from April to November. In the rainy season calves can be left in a camp for only a fortnight if rain in excess of 20 mm. falls during that period. This is particularly applicable if rain falls on more than one day in any fortnight. As often happens, long dry periods occur even in the rainy season. The grazing will not become infested and calves can be left in a camp during this dry period until the rains start again.

(3) *Separation of age groups*

Calves born in summer or autumn will become infested. They will act as "carriers" for the next season's calf crop. All calves born before the rains cease in April should be weaned by the end of August,

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and placed in weaner paddocks which should be of the highest quality grazing on the farm. The calf paddocks will then be free of "carriers" for at least two months before the rains start. The winterborn calves will not acquire infestation until the rains start at the end of November. They can be run with the new-born summer calf crop and be weaned in February. These weaner calves should have a separate weaner paddock and not graze with older cattle until the following winter.

(4) *Breeding seasons*

Most calves are born in summer (i.e. November–January) under ranching conditions. Serving should take place from 1 February to 30 April for the summer calf crop. Cows not conceiving during the autumn breeding season may again be mated from 15 August to 15 October, to calve from mid-May over the following two months. This will facilitate management of calves particularly; it is also a simple and practical method of picking out sterile or poor breeding cows.

Almost all these recommendations have been standard practice in the herds at Armoedsvlakte for over 30 years. The fact that the animals on this station are almost free from parasitism is the proof that these principles of animal husbandry are very sound and can be applied in practice.

SUMMARY

1. The rôle played by infested dung in the transmission of parasites varies, depending on whether it is undisturbed in the veld or occurs as accumulations of manure in kraals, calf pens, etc.

2. In the veld dung acts as an incubator, shelter, reservoir and prison of infestation. It can be dispersed and disseminated by the activities of dung beetles.

3. Accumulations of dung in kraals are free of infestation in times of drought. On the other hand they are dangerous sources of infestation in the rainy season. The fact that highly susceptible young animals are kept for many hours every day, or constantly, in calf pens and kraals, facilitates the spread of parasites in the rainy season.

4. Control must be based on sound principles of animal management which include:

(*a*) Hygienic calf pens and kraals with concrete floors which are kept clean.
(*b*) The provision of three or four calf paddocks grazed in rotation.
(*c*) Separation of age groups.
(*d*) Set breeding and calving seasons.

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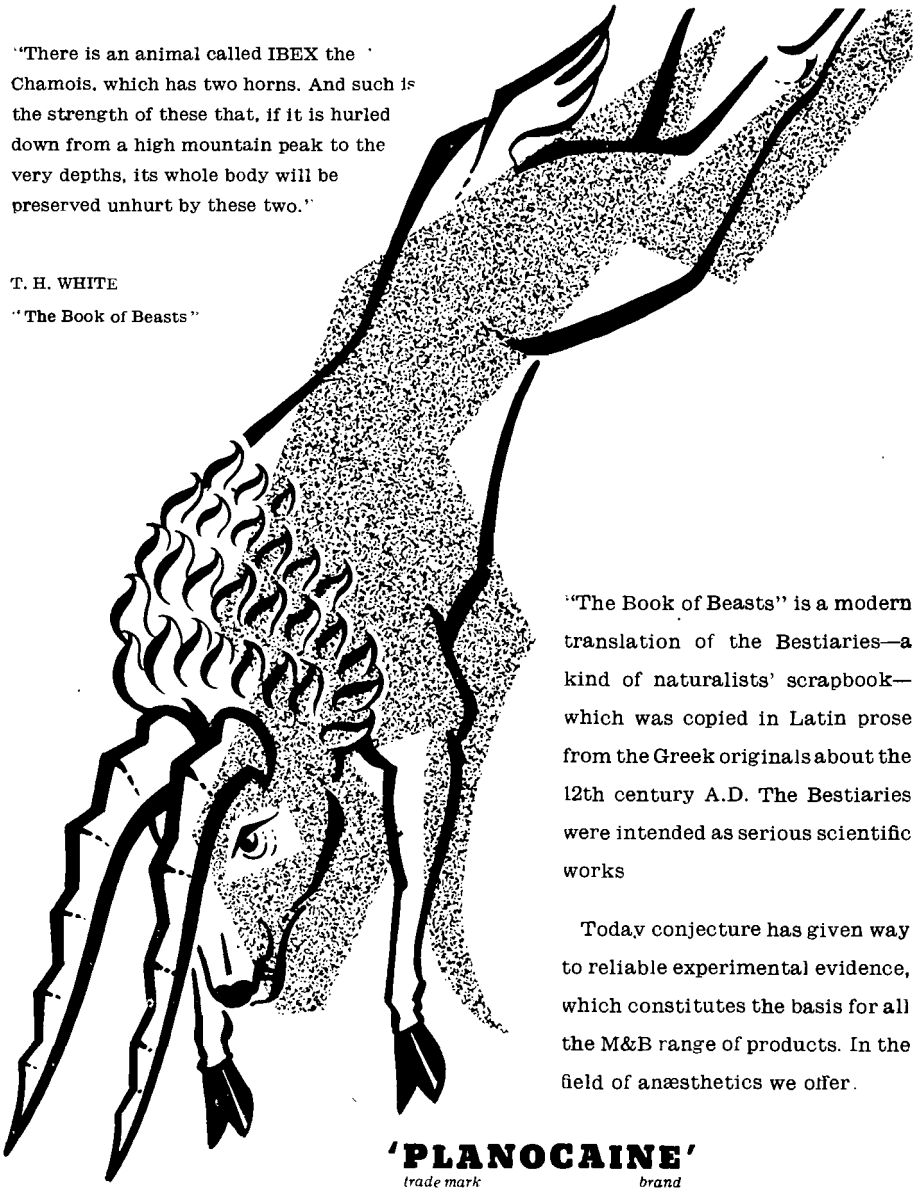
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T. H. WHITE
"The Book of Beasts"



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NOTES ON TRACE ELEMENT REQUIREMENTS ACCORDING TO A RAPID SURVEY OF THE UNION

J. W. Groenewald*

General interest in the possible benefit to be derived from the supplementation of trace-elements is increasing among veterinarians and stockmen in the Union. This is due chiefly to the frequent reports, of improved fertility and general health, especially under intensive stock farming systems, after judicial supplementation of certain trace-elements. Although cases of non-functional infertility in cows and poor condition in calves appear to be reported more frequently from sourveld and coastal regions, the trouble may actually be reported from farms situated in any part of the country. However, considerable doubt and confusion on the value of trace-element supplementation is evident due to the official attitude that there is no experimental evidence available to prove that such application is necessary or beneficial in the Union.

The possibility of a manganese deficiency in poultry fed on grains and greens grown in the Union is readily accepted. It would also be agreed that "slipped tendon" or porosis in chickens is aggravated in the presence of various factors of imbalance such as:—

(i) A high calcium and phosphorus intake; this is in direct relationship to the manganese requirement. For instance if the Ca and P be increased three times, the manganese level has to be increased seven times to prevent porosis.
(ii) Choline,
(iii) Methionine,
(iv) Biotine,
(v) Betain.

Not only is it necessary to pay attention to a sufficient supply of the factors indicated but it is accepted that the heavier breeds of poultry are more susceptible to a manganese deficiency. Although the minimum requirement for manganese in chickens is usually considered to be 55 mgm. per kg. of the ration, local standard specification puts the amount at 70 mgm/kg. As indicated this amount is dependent on various other factors.

In a report on the copper content in 3,702 veld pasture samples drawn from all over the Union, Truter and Louw (1959), found that 10% of the samples fell below 3 p.p.m. Cu and that no less than 31% fell below 5 p.p.m. Actually only 59% of the samples could be classed as "adequate". Work on fertilizer application of copper and also molybdenum is showing promising results at Dohné and the Outeniqua Experimental Stations. Attempts at work on trace-elements in the

* P.O. Box 50, Wadeville. Dr. Groenewald was accompanied by Dr. J. Grashuis on a tour of investigation.

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Union is not complete without reference to Penzhorn's work at Vaalharts and especially that of Perold and Swart at Stellenbosch. However, the technique applied to past work was so inadequate as to cast doubt on the accuracy of analytic data. These researchers, however, drew attention to the worst trace-element deficiency areas in the Union.

During the last few years considerable information has been published in Holland, England, California and Florida which would tend to stress the influence of nutritional and environmental influences on the utilisation of various nutrients, including trace-elements. If considered separately, the work on nutrient interrelationship appears to be confusing. Grashuis from Holland, who possesses the ability of co-ordinating and applying the various influences of nutrition to the animal, visited the Union recently. According to him, intelligent interpretation of available information is possible only by considering the soil-plant and animal relationship. As in the case of porosis in chickens, the animal and not the test tube is the best ultimate measure of animal health. Certain characteristic conformational deviations may be attributed to specific mineral and trace-element deficiencies.

A short description of common conformational deviations as well as the circumstances under which they are likely to occur follow. Of special interest is the fact that several of these deviations are frequently seen in animals in the Union and every effort has been made to correct these weaknesses by breeding. According to Grashuis these are not straightforward inherited defects in certain breeds, but an inherited requirement for a larger amount of the nutrient element for the breed, e.g.:—

1. *A phosphate deficiency* appears to be indicated in cases where the hocks are placed excessively far back in cattle and horses. The end of the tail usually hangs between the hocks and the latter is frequently stained by faeces. This cow- and sickle-hocked condition is seen in all breeds, but probably more frequently in the Jersey. This appears to indicate that the requirement for phosphate is greater in the Jersey breed. So much work has been done locally on calcium and phosphate deficiencies that these minerals are referred to briefly. Actually the ratios of Ca:P greatly influence the availability of certain trace-elements and have frequently to be referred to.

2. *A calcium deficiency* usually occurs in the presence of prolonged high phosphate intake. Cattle then tend to place their hind limbs too far under the body with the result that they stand on the heels and the hoofs tend to grow out too long. The condition can become so bad that sores develop under the heel. The condition of pigs even sitting down and osteo-fibrosis in horses is well known. (Camped under.)

3. *The calcium:phosphorus ratio* is considered ideal when it is 1.5:1 on soils with a pH of 4.8 to 5.5. However, the ratio of Ca:P may be reversed by heavy phosphatic fertilisation. The basis of most of the trouble appears to be an excess of either one or more of the following:

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protein, calcium, phosphorus, potassium especially when applied as fertilizer or when present in soil.

4. *A manganese deficiency* is characterised in young animals by too straight a hock. In some cases the hock joint is almost bent forward with the result that the tail hangs well back of the hocks. The animals are on the tip of their toes and even knuckling over. In older animals bony enlargements on the lateral aspect of the hock may be evident. These enlargements may become bruised by animals lying on cement and lead to galls. Poor fertility is usually evident in such herds. A manganese deficiency is generally present where alkaline fertilizers such as basic slag, lime and potassium have been applied for some time. Should the soil pH be above 6, it would be likely that all animals show symptoms of a manganese deficiency, but below pH 5.1 no symptoms are likely to be seen. However, on the borderline of 5.1 to 6 only certain animals may show symptoms, which again indicates the higher inherited Mn requirement of some animals.

The description of a manganese deficiency is frequently observed in certain breeds such as the Afrikander, and has been assumed to be an inherited breed weakness.

5. *A copper deficiency* can best be seen in young Friesland animals. The indications are a reddish-brown hair coat, long discoloured hair around the rim of the ears, sometimes discoloured tufts of hair on the inside of the ear. Hair may even fall out around the eyes. However, hair discolouration is not specific for a copper deficiency as many other factors may cause discolouration.

A more definite indication of the deficiency appears to be an enlargement of the pastern joint along the epiphyseal line and a slight ridge or unevenness of bone on the medial aspect of the pastern above the hoof. Instead of a normally clean joint there is a fullness which gives the appearance of a "block foot". A spavin is usually noticeable on the medial aspect of the hock as well. Apart from general unthriftiness, milk production is poor and butterfat low. A lowered butterfat of 0.3 to 0.4% may be attributed directly to a copper deficiency. Fertility is lowered and a condition of "silent heat" is prevalent in young cattle on Cu-deficient pastures.

Apart from the possibility of a direct copper deficiency an indirect deficiency is possible on pastures that have been heavily fertilized with nitrogen. In such cases too high a percentage of amines are present in the grass and these tie up with copper in an unavailable form. The same happens on peat soils where the sulphur content may be too high. A high molybdenum or zinc content of feed also proves undesirable for utilization of copper. An indirect copper deficiency is frequently associated with unthrifty animals that often have a diarrhoea and a high blood phosphate level.

6. *A cobalt deficiency* in ruminants is indirect by nature, as there is no proof that animals utilize cobalt directly. However, ruminal bacteria require cobalt to synthesize B , which is an important requirement for the host. Pigs and poultry may obtain their B_{12} requirements from animal protein.

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Cases of cobalt deficiency, or, more correctly, B_{12} deficiency are stunted and small or light for their age. Their heads are usually large for body size and a dull coat is evident. The animals show a strong tendency to lick each other, urine, or eat urine-stained bedding or other material. Abortions may be common or weak calves born and all sexual life eventually ceases.

In the past the combined symptomatology referred to was considered to be rickets. There is sufficient evidence to prove that such classification would be incorrect. In Holland, where phosphate occurs in excess, the condition is still evident. It may also be indicated that the symptoms are not marked in native breeds where growth is slow and stunted. However, in "bone disease" attributed to nutritional inadequacies a very sensitive mechanism must be responsible for the dominating clinical symptom to exert itself. For instance, the symptoms of rickets or osteomalacia appear to be directly opposed to those of a manganese deficiency in the stance of the animal. Analytic evidence would be necessary to confirm or refute the clinical symptoms. However, in the meantime it must be considered a great step forward that clinicians are given some sort of guide and indication as to what to look for in farm animals.

A guide as to the minimum level of intake based on the total ration would be:—

(*a*) At least 0.3% P, provided the Ca:P ratio is not wider than 1.5:1.0. If based on 20 lbs. dry material, for cattle the P requirements would be met by a daily intake of 3 ozs. of bone meal. However, as the Ca:P ratio in mature veld hay is frequently wider than 4:1, optimum conditions are difficult to maintain.

(*b*) As indicated, the manganese requirement would be greater in the presence of a higher calcium. It is estimated that approximately 100 to 150 p.p.m. of the total dry matter of the ration would be necessary. This is equivalent to 100–150 mgm./Kg. of ration which is met by a daily intake of 5 to 7 gm. of manganese sulphate or manganese nitrate daily for mature cattle. The higher amounts would again apply to soils high in lime, potassium and iron as these factors have a depressing influence on the utilization of manganese.

(*c*) The copper content of the total dry matter of the ration should be 10 p.p.m. or 10 mgm/Kg. This would be met by a daily intake of 0.5 gm. copper sulphate or 0.25 gm. copper carbonate. The latter is less toxic and as available to the animal as the sulphate. On highly fertilized pastures it may be necessary to double the above copper levels.

(*d*) The daily allowance of cobalt recommended is 1 p.p.m. of the total dry matter or 1 mgm/Kg. This would be met by giving cattle 0.05 gm. cobalt sulphate or 0.04 gm. cobalt chloride daily.

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At this stage it would not appear to be justified that a special lick containing manganese for alkaline areas, or a lick containing copper for sour areas, be recommended. A general lick to meet all the requirements may be recommended as follows: 64% sterilized bone meal or 48% degelatinised bone flour, or 36% di-calcium phosphate, 10% molasses, 0.20% manganese sulphate, 0.05% copper carbonate and 0.01% cobalt sulphate (total trace-elements of 0.25 lbs.). The balance can be made up with salt. Cattle should each receive approximately 3 oz. of such a lick daily.

As it is difficult to ensure homogenous mixing of trace elements on the farm, the purchase of a premix is recommended.

BOOK REVIEW.

"THE EXAMINATION OF ANIMALS FOR SOUNDNESS"

R. H. Smythe

Published by Crosby Lockwood & Sons Ltd., London. Price 13/6

No branch of veterinary practice offers more pitfalls than the examination of animals for soundness and good health, and the issue of the relative certificates. The omission of a defect, or a wrong interpretation can have repercussions that may lead to litigation and affect adversely the reputation of the practitioner. It is surprising therefore that the veterinary profession has had to wait so long for a reliable guide in this field.

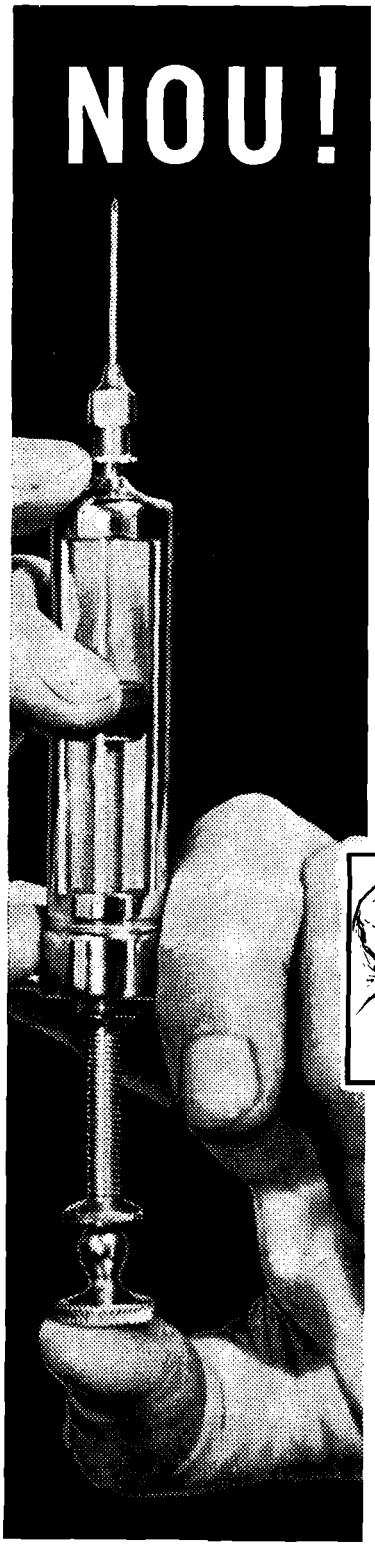
In its 140 pages the above book provides an excellent and detailed description of the methods of examination, the defects to look for and the significance of the various abnormalities that may occur in the horse, bovine, sheep, goat, pig, dog and cat. In addition, one chapter gives a very practical account of the teeth of animals as a guide to age.

The only point on which one can criticise it is that the otherwise very full account of the examination of the cow does not mention rectal palpation of the pelvic genitalia.

The book teems with practical hints and valuable advice, based quite obviously on long experience, keen powers of observation and sound judgment. A noteworthy feature is that the various malpractices of unscrupulous owners which aim at misleading the unsuspecting veterinarian are also mentioned in the appropriate sections.

Every veterinary practitioner, and especially the young and inexperienced, as well as veterinary students, will find this book an invaluable guide.

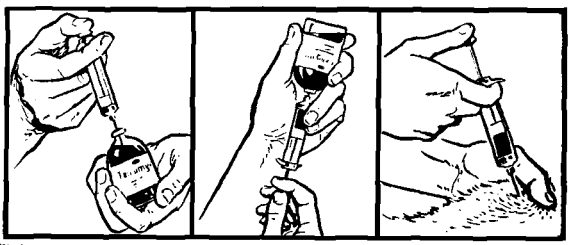
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SOME ASPECTS OF THE NUTRITION OF THE CALF

M. W. Henning*

The economy of calf rearing and the maintenance of the calf's health is a universal problem embracing both the feeding and the husbandry of the calf. In a previous article the author[20] has shown that the incidence of disease and calfhood mortality are influenced as much by the care, management and the housing of the calf as the methods of feeding practised. In the present report the author wishes to confine his discussion mainly to certain nutritional aspects of calf rearing.

Cow's milk, the natural food of the newborn calf, is a perfect food containing all the essential ingredients required for the proper nutrition of the calf. The first postpartum secretion, known as colostrum, an essential product for the survival of the calf, differs entirely in its composition from ordinary milk. Its main component, protein, comprises approximately 16.5 per cent. of its total weight. Of this amount gamma-globulin, the carrier of antibodies, represents about 75 per cent and casein nearly all the balance. No detectable albumin can be demonstrated in cow's colostrum.[39] On the other hand, cow's milk contains only about 4 per cent protein of which barely 0.5 parts is whey protein, composed largely of lactalbumin and a little lacto-globulin. Colostrum is also richer in fat, minerals and Vitamin A and D than milk, but it contains only 2.2 per cent lactose as compared with 4.6 per cent of cow's milk.

The blood serum of the new born calf is characterized by its extremely high albumin and alpha-globulin content and its complete absence of gamma-globulin.[39] Soon after the first colostral meal, gamma-globulin can be demonstrated, increasing in amount during the nursing period, while the albumin levels decrease correspondingly. For 24 hours postpartum the intestinal mucosa of the calf is permeable to both the antibody (gamma-globulin) and the non-antibody fractions of the proteins present in the colostrum. The non-antibody carrying fractions are readily lost through the kidneys, giving rise to a proteinuria, while those proteins carrying antibodes are retained.[9] According to Comline, Roberts and Titchen[8] the colostral globulins do not enter the portal circulation but are carried by the lymph to the peripheral blood. In due course the calf acquires the ability of producing its own globulins, which reaches adult levels from 30 to 60 days after birth.[38]

In agreement with Lovell and Hill[28] and Withers[49] the author has found that calves nursed by their dams during the colostral period are healthier than those that receive their colostrum out of a bucket. Although it is difficult to explain the difference in response by the two groups of calves, there are certain contributing factors which

* Department of Agricultural Technical Services, Transvaal Region, Pretoria.
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no doubt play a rôle. Calves left with their dams suckle at intervals varying from four to six hours and consume anything from 15 to 26 pounds of colostrum during a period of 24 hours without manifesting any evidence of scouring.[46] Those given colostrum out of a bucket are usually fed only two or three times a day and, depending on their body weight, receive only five to ten pounds of colostrum during the same period. If they are given significantly more than ten pounds they are liable to develop digestive upsets.

Observations made by Henning and Johnsen[22] on Jersey calves suckled by their dams during the colostral period showed that calves weighing approximately 50 pounds at birth consumed on an average each 12.25 pounds of colostrum per day for the first four days post-partum. During this period the average gain in body weight amounted to 2.87 pounds per day. A Friesland calf fed in the same way consumed 16.1 pounds per day and gained 4 pounds in body weight daily.

The first three or four weeks of the calf's life is a very critical period for the digestion of carbohydrate, protein and fat. During this period the calf is still a monogastric animal, and can subsist only on colostrum and milk or milk products. Its digestive apparatus must first be adapted to the utilization of roughage, grain and concentrates before it can be fed on the usual ruminant diet.

Carbohydrate digestion

Shaw, Woodward and Norton[45] were probably the first to demonstrate that the calf was not capable of utilizing starch until it was four weeks old. No wonder, therefore, that Maynard and Norris[32] found synthetic milks containing starch entirely unsuitable for the feeding of young calves. According to Larsen, Stoddard, Jacobson and Allen[27] starch and maize administered directly into the abomasum had no effect on the concentration of the blood sugar; and was excreted unaltered in the faeces, whereas glucose given in the same way caused a marked rise.

In a study of the relative efficiency of lactose, glucose, corn-syrup (a crude glucose product) and starch as sources of carbohydrates in milk replacements Flipse *et al*[15], [16] found that lactose was by far the most suitable and best tolerated carbohydrate. Its beneficial effect was reflected by the healthy appearance of the calves, their better growth response, their efficient food utilization and the firmer consistency of their faeces. Blood sugar levels rose rapidly after the ingestion of lactose, glucose and corn-syrup, but there was no change after the feeding of starch.

Dollar and Porter[11] furnished further proof that lactose and glucose were the only suitable sources of carbohydrate for calves during the first four weeks after birth, i.e. during their pre-ruminant life. Carbohydrates fed in any other form, viz. as oat-flour, or maize meal (cooked or uncooked), sucrose or maltose, were not tolerated. Fluid rations in which any of these carbohydrates were incorporated invariably provoked digestive derangement. Whereas either lactose or glucose produced a rapid rise in blood sugar, maltose and sucrose caused merely a slight rise, and starch none.

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A study on the operation of enzymes in the small intestine of young calves by Dollar and Porter[11] revealed that lactase was very active at birth but that its activity gradually waned with advancing age. No detectable maltase, sucrase or amylase activity could be demonstrated at birth, but as the calf grew older these enzymes gradually appeared and progressively increased until their hydrolysing effects were properly established when the calf was about four weeks and ruminating.

In the baby calf the glucose provided by the hydrolysis of lactose is the major source of energy. This is demonstrated by the relatively high glucose values in the blood during the neonatal, pre-ruminant period and the low levels of volatile fatty acids which furnish most of the energy requirements of the ruminating animal.

According to Hodgson, Riddell and Hughes[24] the blood glucose of the newly born calf amounts to 100 mg. per 100 ml. With advancing age the glucose values decrease, eventually reaching a range between 35 and 70 mg. (mean 52.5) in the adult. They observed no appreciable increase after feeding but noticed that the blood sugar might fall to less than 50 mg. during fasting. Excitement caused a marked increase. On the other hand de Villiers and Henning[10] have found that the glucose levels of young calves may rise to 180 or 190 mg.

As soon as the calf is able to consume roughage and concentrates, ruminal activity ensues, as indicated by the rising levels of volatile fatty acids in the ruminal fluids, the decline of blood sugar values and the mounting concentration of volatile fatty acids in the circulation, especially the portal blood. In other words, as the activity of the ruminal micro-organisms increases and the digestion of cellulose and other carbohydrates advances so do the levels of volatile fatty acids in both the ruminal fluid and the blood rise.[29] But since only a relatively small proportion of the fatty acids absorbed in the fore-stomachs actually reaches the general circulation, the concentration of these acids in the peripheral blood cannot reflect the actual amount available for energy.[2]

Absorption studies by Flatt, Warner and Loosli[14] revealed that the rate of absorption of volatile fatty acids from the rumen-reticulum of the young calf amounted to 4.5 g. per hour, the rate being highest for butyric acid, less for propionic acid, and lowest for acetic acid. In the adult the rates recorded were 2.66, 2.26 and 1.69 per hour respectively.

The shift towards adult values and the decline of blood glucose begin as early as one week after birth and gradually continue until the sixth week or later, depending on the availability of milk in the diet, the most rapid fall in sugar usually coinciding with the discontinuation of milk feeding. At six weeks, or sometimes even earlier, the ruminal function is assuming adult status and the volatile fatty acids poured into the circulation are making a considerable contribution to the energy requirements of the calf.

The implication of these observations is that lactose and glucose are the only carbohydrates suitable for incorporation into a synthetic milk. Starch, the cheapest and most common energy-yielding in-

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gredient of stock feed, cannot be utilized by the baby calf. Actually its presence in liquid form is a liability. When fed, it by-passes the rumen-reticulum and travels along the oesophageal groove to the abomasum and intestine. But since no provision has been made for its digestion, it is evacuated unchanged in the faeces. Moreover, during its passage through the gut it irritates the intestinal mucous membranes and in that way causes severe scouring. The proper utilization of starch, cellulose and other carbohydrates can be effected only through the medium of the ruminal micro-organisms which have the power of breaking up food containing these carbohydrates into assimilable factors when given in a dry form. Every inducement, therefore, should be made to initiate ruminal activity and encourage the early consumption of dry feed. This will lead to the utilization by the calf at an early age of hay, grain and concentrates, which are common sources not only of cheap energy-yielding carbohydrates but also of cheap vegetable proteins. As soon as ruminal function and the consumption of good quality hay are properly established the incidence of scours is no longer a problem.

During the fermentation which occurs in the forestomachs polysaccharides and disaccharides in the food are converted into various volatile fatty acids of which butyric, propionic and acetic acids are the most important. As pointed out by Phillipson and McAnally[37] the concentration of these volatile fatty acids is very high in the rumen ingesta but low in the abomasum, indicating that their absorption occurs in the rumen. The amount of fatty acids absorbed in the rumen, reticulum, omasum and also in the caecum provide the ruminant with most of its energy requirements. According to Bancroft, McAnally and Phillipson[2] the absorption surface of the rumen is greatly increased by the ruminal papillae and the amount of fatty acids present in the general circulation represents barely 15 per cent of that found in the venous blood leaving the rumen.

Brownlee[7] reports that the development of the papillae is determined by the nature of the diet, being apparently controlled by such factors as energy value of the food and the rapidity with which it is broken down into assimilable fractions. The papillae remain infantile when the calves are fed on milk alone for two or three months, whereas well developed papillae are formed in the rumens of those receiving an ordinary diet, including hay and concentrates. A ration composed of kale, mangolds and concentrates is apparently a better stimulus than hay and concentrates. But Warner, Flatt and Loosli[47] state that the growth of papillae respond equally to the feeding of hay and concentrates. They postulate that certain chemical agents produced during the fermentation in the rumen rather than the fibrousness of the diet are responsible for the stimulus which leads to the growth of the first stomach.

Brownlee[7] believes that roughage is not the deciding factor in the growth of papillae, yet when concentrates alone are fed, to the complete exclusion of roughage, the mucous membranes of the rumen are easily torn and exhibit areas of erosion. Moreover, every calf has an irresistible urge to eat roughage and in the absence of hay or other dry food,

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will consume straw, peat moss, sawdust, hessian, or any other fibrous material within its reach.

Fat

Butter or milk fat is the natural fat provided by milk for the nutrition of the calf, and cannot be replaced satisfactorily by other fats or oils. It is not only the most valuable and most reliable fat for the nutrition of the calf but it is also the carrier of limited supplies of Vitamin A and D, and it confers on whole milk a valuable costive property whereby it significantly reduces the laxativeness of certain diets.[36]

Many unnatural fats have been employed as substitutes for milk fat from time to time, but the results obtained have been disappointing and their use had to be abandoned in most cases. Before any unnatural fat can be utilized it should first be emulsified in skimmilk or a milk replacement at about 120°F and then properly homogenized, to reduce the size of the fat globules to less than two microns. Homogenization is facilitated by the addition of soya lecithin in an aqueous solution.[26] If the oil or unnatural fat is not homogenized before it is administered the calf will not grow properly and will suffer from severe gastric derangement.[3], [26], [41] Homogenization is, however, impracticable under ordinary farming conditions and cannot be recommended.

In a study of the value of filled milks—skimmilk plus an unnatural oil—Bate, Espe and Cannon[3] found that the feeding of unhomogenized vegetable oil in skimmilk led to shedding of the hair, scours and depressed growth. As soon as the oil was administered in a homogenized state no further loss of hair occurred, and new hair started to grow. Rumen function also prevented the deleterious effect of unhomogenized oils. Soon after rumen activity commenced and the calf was able to consume hay and concentrates no further loss of hair occurred and the health of the calf improved. Growth of new hair and improvement of health continued even when an unhomogenized oil was still incorporated into the ration.

Gullickson, Fountaine and Fitch[19] reported that calves fed on diets in which butter oil was incorporated were less prone to scours than those receiving oils having a lower melting point. Properly homogenized lard or tallow was better tolerated than cocoa-nut oil and peanut oil which in turn were superior to maize oil, cotton-seed oil and soya-bean oil. The feeding of unnatural fats and oils especially in an unhomogenized state, led to indigestion and scours, and loss of hair in patches over various parts of the body. Some of the calves fed on maize oil, cottonseed oil or soya-bean oil did so badly that they had to be sacrificed.

Supplementation of skimmilk or reconstituted dry milk with an unsaturated oil, e.g. cod-liver oil, fish oil, lard, maize oil or cotton seed oil, may lead to serious muscular dystrophy and death of the calf.,[17], [4], [6], According to Garton *et al.*[17] the gross deficiency in tocopherol content of the fats incriminated, rather than the unsaturated fatty acids, is the important consideration in the pathogenesis of this disturbance. In

order to prevent the development of muscular dystrophy in calves reared on filled milks Adams *et al*[1] and Safford *et al*[43] advise the supplementation of the rations with tocopherol, while Blaxter, Brown and MacDonald[5] recommend the use of Vitamin A and D in a concentrated form rather than in cod-liver oil as a source of these vitamins.

For these reasons the author has consistently abstained from supplementing the milk replacements used by him with cod-liver oil, fish-oil or any other unnatural fat. He has, nevertheless, obtained excellent results with calves fed on low fat diets viz., milk replacement, skimmilk or milk replacement plus a small daily ration of whole milk. Vitamin A and D were supplied in concentrated form or by means of good quality lucerne hay. Additional Vitamin D was obtained by exposure to direct sunlight.

Protein

Cow's milk contains approximately 4 per cent protein of which about 3.5 parts are represented by casein, an essential protein for the nutrition of the young calf. For the initial stages of protein digestion the calf is totally dependent on rennin, which acts specifically on casein, coagulating it soon after its arrival in the abomasum. Without prior coagulation the proper digestion and assimilation of the casein cannot occur. The coagulation provides for the retention of the casein curd in the fourth stomach and permits the predigestion of the protein before pepsin-HCl activity sets in.

For the coagulation of milk and the action of rennin calcium is essential. Kastelic, Bentley and Phillips[26] report that milk is not tolerated by the newborn calf when the ratio of calcium to sodium is sufficiently disturbed in favour of sodium to prevent the normal coagulation of the casein by rennin.

The Ca:Na ratio should not be less than 2.5:1 and at least 1.1 g. calcium per litre of milk is essential for the coagulation of the casein by the rennin. The ratio of $CaO:Na_2o$ in ordinary milk is in the vicinity of 3.33:1. Calves reared on Ca-deficient milk suffer from digestive derangement, become unthrifty and are liable to die in the course of about a week. At about two weeks, when the pepsin-HCl activity is established, a calcium deficient milk loses most of its detrimental properties and the calf will tolerate it as well as one containing calcium and sodium in the right proportions.

In South Africa large supplies of butter-milk powder are available at moderate prices for compounding milk replacements. But most of these powders are made from sour cream partly neutralized by sodium bicarbonate, resulting in an excess of sodium in relation to calcium, and invariably produce active scouring when fed to young calves less than three weeks of age, but not when given to older calves. Butter-milk powders prepared from cream in which the acid has been neutralized with calcium hydroxide are much better tolerated by calves, and do not cause the digestive upsets commonly associated with those containing an excess of sodium. The use of butter-milk powders containing an excess of sodium in relation to calcium, therefore cannot be recommended as an ingredient. De Villiers and Henning[10] have

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found some consignments of butter-milk powder to have ratios of Ca:Na as unbalanced as 1:3.

The incorporation of vegetable and unnatural animal proteins in synthetic milks as a substitute for milk proteins invariably leads to serious digestive disturbances and depressed growth when fed to very young calves, and their use had to be abandoned.[21, 35, 41] Only after ruminal activity has been fully established can mixtures containing these proteins be fed with advantage.[23]

It must be evident, therefore, that the only nutrients which can be utilized efficiently when incorporated in a liquid diet of the young calf are casein, butterfat, lactose and glucose.

Dry Food

The increasing demand for milk for human consumption has led to such a marked rise in the price of milk that the rearing of calves for herd replacements and herd improvement has become both uneconomical and unattractive for many dairymen. Although reconstituted milk has found a wide application on farms where fresh milk (whole or skimmed), is not available, it is not the whole answer to the problem. The high price of milk powders required for compounding the milk substitutes is a serious limiting factor to their universal use. Various workers have, therefore been encouraged to explore more economical methods of rearing calves on limited amounts of milk or milk products.

Since the alimentary canal of the young is not capable of utilizing anything else but milk and milk products during the first three or four weeks of its life, its digestive apparatus should be properly conditioned before other foodstuffs can be fed with advantage. Any milk or fluid milk replacement given to a calf by-passes the rumen and is conveyed along the oesophageal groove directly to the abomasum where the digestion of lactose, casein and butter-fat is initiated, to be completed in the intestine. Any other type of food, e.g. grain-meal, or calf-starter, when administered in fluid form, is also carried directly to the fourth stomach and eventually reaches the intestine. But since no provision has yet been made for its utilization it is evacuated unchanged in the faeces. During its passage it irritates the mucous membranes and causes gastro-intestinal derangement. This explains the severe scouring in very young calves commonly following the use of improperly compounded milk replacements, usually of commercial origin.

When cereal-meal or calf-starter, however, is fed in the dry state, with or without roughage, it lands in the rumen-reticulum where it stimulates rumen function and the bacterial fermentation of the food eaten. On this principle several workers have studied the value of dry-feeding in conjunction with the early weaning of calves. Eckles and Gullickson,[13] and Mead, Regan and Bartlett[33] discontinued milk feeding soon after the 30th day and then fed their calves exclusively on good quality calf-starter and water. Jones, Brandt and Wilson[25] and Savage and Crawford[44] continued with milk and milk repacement

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until the seventh week before they switched over completely to dry feed.

Many of the earlier workers incorporated various animal proteins, viz. skimmilk powder, casein, fish-meal and blood-meal, in their starter mixtures. But since the ruminating calf, by virtue of the activity of its ruminal micro-organisms, is capable of utilizing cheaper vegetable proteins, the employment of expensive animal proteins is not essential. Many of the later workers have obtained excellent results with mixtures containing no animal protein but only cheap vegetable protein.[12, 18, 23, 34, 35] The mixtures were in some cases made more attractive by the incorporation of appetising agents such as molasses, lucerne-meal, date meal or molassine meal (containing 36 per cent sugar). Preston[40, 41] claims that the addition of 15 per cent molassine-meal to the basic diet increased the consumption of dry starter during a period of two to eight weeks.

Preston[40] succeeded in rearing calves on as little as 130 pounds of whole milk supplemented with dry starter-meal and good quality hay. Drouliscos, Verbeek and Armstrong[12] reduced the total milk ration to 60 pounds. For the first three weeks some of the starter was incorporated in the milk in the form of a gruel, but since this fluid containing unnatural starches and proteins is conveyed by the oesophagel groove directly to the fourth stomach, the author cannot recommend the feeding of the starter in gruel form.

After studying various methods of dry feeding and early weaning the author eventually decided to employ the following procedure:—

The calf is removed from its dam after the colostral period and placed in a single loose-box or pen where it has free access to prime quality lucerne hay provided in a hay-rack from the first day. It is then fed on either whole milk, skimmilk or milk replacement for a period of about 30 days. The skimmilk or milk replacement is supplemented with 85 pounds of whole milk spread over the entire period of 30 days. The total daily fluid ration of a calf is approximately 10 per cent of its body-weight.

The calf soon starts nibbling at the hay and by the end of the second week has already commenced ruminating. (According to Mathews and Fohrman[31] the calf may start ruminating as early as the sixth day when kept under stabling conditions. On pasture it may ruminate on the seventh day, but rarely later than three weeks[30].) At this stage dry calf-starter, either in the form of a meal or pellets, is offered in a dry milk bucket immediately after a fluid meal. The calf, desirous to obtain more "milk" from the bucket, usually manages to take a few mouthfuls of the starter. The bucket containing the starter is left in an easily accessible spot for the calf to eat more at will. Although Brownlee[7] and Warner, Flatt and Loosli[47] report that ruminal activity can be stimulated by feeding either hay or concentrates all the calves in this experiment were allowed hay *ad libitum*.

The one calf differs markedly in its behaviour from the other. Whereas the one may take to the starter readily, the other is more fastidious and hardly eats any of it. Nevertheless at the end of 30 days most calves have consumed from two to three pounds of hay each,

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and from one to two pounds of starter. On rare occasions a calf may have eaten more or less than this amount.

At thirty days rumen activity has usually advanced so far that the calf is able to obtain a great deal of its energy requirements from the metabolized rumen products. Most calves can then be weaned entirely from milk or milk-replacements. But the time of weaning should be determined by the amount of dry food consumed and the weight of the calf. If a calf is slow to adapt itself properly to the dry rations and its rate of growth is not satisfactory the weaning may have to be delayed for another week or two. But it is superfluous to continue with a milk diet for more than six or seven weeks.[23]

Hunger is the best stimulus for eating the starter, and the complete starvation of the calf for the first 24 hours after weaning is advised; but, in order to prevent dehydration during this period, sufficient water is allowed in a milk bucket at every feeding time. At the end of the 24 hours starvation dry calf-starter is offered in a milk-bucket immediately after water has been given. The noise and sight of the bucket act as a further inducement for the calf to eat the starter. Hay is again provided and the calf is allowed to eat the starter at will, as much as three pounds a day. Few calves, however, will consume as much during the first month after weaning. Some may barely eat a pound a day at the start. During the ensuing month, therefore, while the calf is adapting itself to its new diet, its performance will generally be poor. Nevertheless, as soon as it has accustomed itself to the changed diet, gains in body weight will improve rapidly and at six months its performance will compare favourably with that of a calf fed on a full ration of whole milk for 30 days or even 60 days. (Table 1.)

TABLE 1

Performance of four groups of calves fed for 30 or 60 days either on a full ration of whole milk or on milk replacement plus whole milk for 30 days. All the calves had free access to lucerne hay from the first day, and in due course were allowed calf-starter at will up to the maximum of three pounds a day.

Group	I 60 days on whole milk	I 30 days on whole milk	III 60 days on milk substitute	IV 30 days on milk substitute
No. of calves	6	11	18	17
Average food intake per calf:				
1. Whole milk for 30 days	—	265 lb.	85 lb.	85 lb.
2. Whole milk for 60 days	565 lb.	—	—	—
3. Milk substitute for 30 days	—	—	—	180 lb.
4. Milk substitute for 60 days	—	—	480 lb.	—
5. Calf-starter up to 16 weeks	164 lb.	159 lb.	151 lb.	171 lb.
6. Calf-starter up to 26 weeks	334 lb.	339 lb.	334 lb.	354 lb.
7. Lucerne hay up to 16 weeks.	162 lb.	251 lb.	221 lb.	273 lb.
8. Lucerne hay up to 26 weeks	565 lb.	754 lb.	723 lb.	755 lb.
9. Average weight at 1 week	81 lb.	82 lb.	88.6 lb.	90 lb.
10. Average weight at 16 weeks	241.8 lb.	209.6 lb.	213 lb.	229 lb.
11. Average weight at 26 weeks	306.0 lb.	314.1 lb.	—	342 lb.
12. Average daily gain in pounds at 16 weeks	1.30	1.13	1.11	1.24
13. Average daily gain in pounds at 26 weeks	1.29	1.33	—	1.38

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SUMMARY

It has been shown that the digestive apparatus of the neonatal calf is adapted solely for the digestion of casein, butter-fat, lactose and glucose, and that these are the only nutrients which can be effectively utilized when incorporated in a liquid milk substitute. Alternative sources of protein, fat and carbohydrate are not tolerated and, when administered, are eliminated practically unchanged in the faeces. During their course through the alimentary canal they irritate the mucous membranes and many produce severe scouring.

The utilization of roughage and dry cereal depends entirely on the activity of the ruminal micro-organisms in the fore-stomachs, and these foodstuffs can be digested only when deposited directly into the rumen. When a cereal meal is fed in a fluid form, in milk or water, it by-passes the rumen and is carried along the oesophageal groove directly into the abomasum and intestines where no provisions yet exist for its digestion. If, however, the calf has been trained to take meal in the dry state, the meal will be lodged in the rumen and broken up into assimilable fractions. These will be absorbed to a large extent by the mucous membrane and papillae of the fore-stomachs and utilized by the body.

By introducing the feeding of lucerne hay and dry calfstarter at an early age rumen activity was stimulated and the calves could be successfully weaned at 30 days or soon afterwards. Calves that had received 180 pounds of fluid-milk-replacement plus 85 pounds of whole milk spread over the 30-day period, showed a daily gain in body weight of 1.24 pounds at 16 weeks. Those that had been given 265 pounds of whole milk for a period of 30 days, and no replacement, gained 1.13 pounds a day, and those calves that had been fed on 565 pounds of whole milk during a period of 60 days gained 1.30 pounds daily. Daily weight gains for the three groups at the end of 26 weeks were 1.38, 1.33 and 1.29 pounds respectively. Lucerne hay was given *ad libitum* and calf-starter (20% protein) at will up to a maximum of 3 pounds a day. But it is not necessary to provide more than 16 per cent protein in the starter.

During the first 30 days after weaning few calves consume a full allowance of calf-starter and, therefore, do not perform satisfactorily during this period. But as soon as they take sufficient starter they readily improve and in due course, may compare favourably with calves that have been reared on much more liberal rations of whole milk.

Jersey calves, 50 lbs. in weight at birth and suckled by their dams during the colostral period, consume on an average 12.25 pounds of colostrum per day during the first four days, and gained an average of 2.86 pounds in body-weight per day. A Friesland calf, 87 pounds at birth, treated in the same way, consumed 16.1 pounds of colostrum per day and showed a daily gain of four pounds.

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CARE AND MANAGEMENT OF THOROUGHBRED FOALS

T. Toms*

The expense involved in purchasing bloodstock today, together with the strenuous efforts required of these animals on the racecourse from a very early age, have caused breeders to develop a very intensive system of rearing foals; horse ranching is practised only by those who are prepared to wait until their animals are three or four years old, before asking them to perform any work. The intensive system has naturally brought with it many problems. Man is inclined to forget that animals have certain set patterns of behaviour for performing natural acts such as giving birth etc., and man thinks he is rendering assistance, where in actual fact he is interfering. This is especially true during the birth of the foal, and during the first few weeks of the foal's life.

Period 1: The first two weeks

The foal is usually born completely surrounded by its amnion. It kicks this open as soon as its thorax has emerged from the vulva. The stud groom usually breaks the membranes as soon as the head has emerged. This does not serve any useful purpose, because the thorax is still restricted of all movement, and results only in the foal making unavailing gasping movements if anything at all. (This early rupturing should only be done in cases of protracted stay in the birth canal, where there is a desire to get the foal breathing at the first possible moment.) It will then be noticed that the foal, having ruptured the amnion, lies quietly, taking deep breaths, while its hind legs remain in the birth canal. This might last for about ten minutes, while the mare also recovers from her ordeal. Stud grooms are inclined to remove the hind legs immediately, and tie and sever the cord. When this is done the foal loses some two pints of blood, which would normally pass from the placenta into its own body, and this deprives it of more or less 2/7ths of its blood supply. (There is some evidence to show that this might be the cause of barkers, dummies, and wanderers.)

When the foal is left to lie quietly, it will be found that after plus/minus ten minutes (no pulse will now be felt in the umbilical vessels) it starts kicking with its hind legs in the birth canal. The mare then gets up, and in so doing ruptures the cord, which loses no more than a drop of blood, and shrinks quite amazingly. There is therefore, almost no blood retained in the cord stump, and a possible source of infection is minimised. The mare will pass the afterbirth much more quickly when it has been drained. Some mares will get up very quickly. Even under these circumstances the cord stump is better left alone. A little bit of bleeding might follow, which is of no consequence. Again,

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a hasty mare might have stayed down quietly if nobody were present fussing around her.

When the cord is ruptured the stump should be covered with a disinfectant; tincture of iodine still seems to be the best. At the same time a quick glance at the buccal mucous membrane of the foal may help in the early diagnosis of sleepy foal disease or similar septicaemic conditions where petechiae may be seen, although extreme measures will be necessary to save a foal showing symptoms so very early.

From now on the foal attempts to get on to its feet. This usually takes about twenty minutes, but may take an hour or more. The foal gets on to its feet far better if left unassisted. Human help more often than not, excites it so much that it falls about all the more.

The foal starts looking for the teat as soon as it is up. In the beginning it is exasperatingly stupid, but finds its way around eventually without any help. Stud grooms are inclined to try to guide the infant foal round to the teat, but they find it far quicker when left alone. It has been said that irreversible changes take place in the liver if the foal does not suck within about an hour, but my experience has been that they can go for five hours or more without any ill effect. A word of warning here: if the foal makes no effort to find the teat, and walks round aimlessly getting stuck in corners etc., one may be dealing with a barker.

Usually a barker will be recognised very shortly after birth. They exhibit jerky movements, followed later by convulsions, and make noises which sound like a dog barking. Later the barking stops, and tetanic convulsions take place, triggered off by external stimuli. Several men will be needed to control the foal, and it will have to be given tranquilisers such as Mysoline, and will have to be fed mare's milk by stomach tube. Recovery is slow, and the foal will have to be taught to suck when it has recovered.

Occasionally a mare, especially a first foaler, will be found who objects to the new foal suckling. It is then advisable to slip a bridle on to her, and to back her into a corner, with her hocks jammed up against the wall.

Some old mares will run milk for a long period before foaling (even up to six weeks or more). As the antibody content of the milk will be low when the foal eventually arrives, it is the practice to administer an antibiotic for the first four or five days of the foal's life.

The next thing which the foal has to do, is to get rid of the meconium. Some grooms administer castor oil or liquid paraffin by mouth immediately after birth, or after the first suck. This seems to be unnecessary and fraught with danger. If the foal is left in peace for five or six hours and no meconium has been passed, or only some of a very rubbery consistency, a glycerine suppository seems to be all that is necessary, especially if the mare has been getting an adequate amount of green feed. If, after this, the foal still shows discomfort by swishing its tail about or rolls and breathes rapidly, one will usually get results by administering 3 to 4 ozs. of 50% warm glycerine solution per rectum with a syringe fitted with a piece of soft rubber tubing.

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Occasionally a meconium spoon may be necessary, and on rare occasions even laparotomy has been performed. (A young foal tolerates sodium pentothal very well—the saphenous vein is used for the injection.) The normal foal, after about twenty-four hours, passes a soft yellow stool.

The morning after birth, if the weather is warm, the mare and foal are taken into a small paddock, and provided the mare does not run the foal off its feet, they are left out all day, even if the foal has been born with a bent leg or stiff joint.

It is sometimes the practice to keep mare and foal couped up in the box for four or five days, and even up to two weeks. I feel that the sooner the foal can stretch its limbs the better, especially a bent limb. From the mare's point of view, unless she has a haemorrhage, this early exercise seems to help her clean out more quickly, and any filling of her legs and underline which might have occurred prior to foaling, will subside very quickly.

On the subject of crooked-legged foals, it is sometimes the practice to put on splints and casts, but even the most peculiar looking ones seem to straighten out after a week or so. There are naturally exceptions.

The second morning of its life, the foal should have a small head collar put on, and be coaxed around a loose box with its dam. They learn to lead very quickly at this early age. It is naturally not permissible to fight with them, but an attendant tickling its quarters or its neck when it jibs, will soon make it follow its mother. At one week of age, the foal will usually lead well, is automatically used to being handled, and is therefore much easier to treat, should the occasion arise.

On the second day, on some studs, the foal is injected with tetanus anti-toxin as a routine.

During this time, and right up to weaning, the mare's udder is carefully watched to see that the foal is sucking her out, and any sign of milk on the foal's head and face is regarded with grave suspicion. The foal's temperature should be taken immediately if this is seen. 102.5°F should be regarded as a fever. In the first two or three days of the foal's life, such a temperature with the foal losing interest in sucking, should cause one to suspect, even in the absence of any other symptoms, that the foal is developing sleepy foal disease, and if treatment is started immediately, a cure can be effected within forty-eight hours. Chloromycetin seems to be the drug of choice, both intramuscularly and per os. As the disease progresses, the foal will be seen to take the teat in its mouth, suck a few times, and the teat will slip out of its mouth, and the head will slowly fall down. The milk from the full udder, having been let down, will squirt onto the foal's face. A few hours later the foal will have a diarrhoea, sometimes bloodstained, and petechiae may develop on the mucous membranes. Hyperexcitability and convulsions may set in in the later stages.

Dullness with no fever at about the same age must cause immediate attention in respect of haemolytic icterus or "Yellows". The mucous membranes in this condition become yellow very quickly. The foal

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soon may become too weak to stand, and may develop haemoglobinuria, severe dyspnoea and fast pulse. Treatment once the condition has set in, is a replacement transfusion of compatible blood, and this is rather difficult to arrange in practice.

When a mare has once produced such a foal, a drop of her blood should be mixed with a drop of foal's blood on a glass slide immediately after birth, and if any agglutination is seen, the foal should be muzzled before it has a chance to suck, for thirty-six hours, and the mare should be milked out at frequent intervals. The foal should be given, hourly, a mixture of cow's milk and lime water and placed on antibiotics. As a differential diagnosis, it has been mentioned that intrauterine *Nuttalia equi* infection is possible.

Occasionally round the fourth day, a foal will be seen showing a distended abdomen, although there appears to be no distress, and it will suck normally. This could be due to a ruptured bladder. Surgical repair is quite straightforward, and the animal will normally make an uneventful recovery. This condition, as well as fractured ribs, might again be caused by a hasty stud groom. Fractured ribs normally heal up unnoticed, although this has been mentioned as a cause of barking foals. About this time also, a pervious urachus might be recognised, although this is rare in the thoroughbred. Umbilical hernias are quite common, but usually close up by about the sixth month.

The normal healthy foal, on about the ninth day, will develop a diarrhoea when the mare has a "foal heat". This usually lasts for four or five days and no steps are necessary to curb the diarrhoea unless the foal should go off suck, and develop a temperature, or if the diarrhoea persists. Cremosuxidine four-hourly for about two days, seems to put it back to normal. Usually in this condition all that is noticed is that the foal has a damp tail and smeared quarters.

Period 2: Two weeks to about six months

After the first two weeks, the stud groom feels that he is over a great hurdle.

The mare with a new foal is put into a larger paddock, with some eight or ten mares with foals all about the same age. It is a good practice to put an attendant in with this group, especially at the introduction of a new mare and foal, as the new arrival causes some curiosity among the others and the mother is inclined to attack any foal who comes too close, and a timely shout may prevent a broken leg.

It is also a good practice to have a gate on each side of the paddock, as the foal is inclined to sleep next to a fence, and get up on the wrong side of it. It causes a great panic if the mare has to be led a long way round away from the foal to bring the two together again.

During the early stages of this period, the foal's navel and joints are watched for any swelling. (Navel ill in one case responded very well to Chloromycetin.) *Corynebacterium equi* infection of the lungs has been described which appeared as a chronic broncho-pneumonia. These cases started with a dull cough. Temperature rose steadily over about three weeks to 104 or 105°F, although the foals never went off suck. They became emaciated and were destroyed. On

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post mortem examination abscesses were found in the lungs. Treatment with antibiotics appears to have been ineffective.

From about one month onwards, the foal starts nibbling at food, depending on the milk supply of the mare. At about three months it takes about 1 lb. of grain daily, to about 3 lbs. at six months. At this stage, if it is found necessary to add minerals to the ration, a pound of feed containing the minerals should be put into a corner of the box away from the mare. This can best be achieved by tying the mare up, at an empty manger, and encouraging the foal to its own feed. When it has eaten all or part of this, the mare's ration can be given to her. After a week most foals will clean up their own portion before going to the mare's feed. Occasionally some mares refuse to allow the foal to eat from their manger.

From about one month onwards, the foal's feet should be picked up and and cleaned out daily, oiled if necessary, and at monthly intervals they should receive attention by the farrier.

From about three months onwards the knees, hocks and fetlocks of the foals should receive attention, and abnormal development should be noted. Should any abnormality be seen, the mare's grain ration should be cut by 3–4 lbs. (she would be getting 6–10 lbs. at this stage), and thereby almost cutting off the foal's supply. Growth is apparently slowed down, and one has a little time to make alterations to the diet if necessary.

Occasionally a foal may be found which eats the mare's tail, with the possibility of developing hairball. This can be stopped by applying Stockholm Tar to the tail.

Worming

When the "foal heat" diarrhoea has stopped, that is, at about two weeks after birth, 30 grams of phenothiazine and 90 grams of piperazine adipate are mixed into the mare's ration as one dose. This is repeated at monthly intervals. The foal, as it starts eating, ingests gradually increasing amounts of worm remedy.

Hand Rearing

Hand rearing should only be tried if no foster mother is available for an orphan foal. Two pints cow's milk of low fat content, 1 pint lime water, and a teaspoonful of lactose is a suitable mixture. The foal receives 4 to 5 ozs. hourly of this mixture at blood heat, in a bottle fitted with a large teat. After four or five days, 8–10 ozs. are given two-hourly; at about eight to nine days 16–20 ozs. are given four-hourly; and at about two weeks onwards, the foal can be given 2 pints three times a day.

From the very beginning the orphan foal should have a small quantity of good teff hay in the box with him, and at about four to six weeks a small grain ration (2 lbs.) and a pound of chaffed lucerne hay should be given. The foal can be weaned at three to four months, after which it is treated in the normal way.

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Weaning

Foals are usually weaned at about six months. The best way seems to be to shift the mare and foal to the foal's new quarters, where the mare will stay with the foal for about two weeks. The mare is then removed, and the two are kept out of earshot of each other. It generally only takes about two days for the foal to clean up his ration, and although they may suck ears and sheaths for a few days they seem content to live normally after about a week.

From this time on, if there are serious deficiencies or imbalances in the diet, they will soon show, especially on knees, hocks or fetlocks. Not only has the foal been deprived of the milk, but often has to graze on the poor quality pasture of autumn and winter.

At nine to twelve months of age, the weanling may develop an enlarged submaxillary lymph gland, which may abscessate and rupture. This seems to be connected with the eruption of the molar teeth. Strangles, of course, should be kept in mind. Horsesickness inoculation is done in September, when the animals are approaching twelve months of age.

Worming the weanling

Worming is done at monthly intervals, as with the mares, right up to the age of eighteen months, when the animals will go into a racing stable. The Phenothiazine is dosed at the rate of 3 grams per 100 lbs., divided into three doses, given at daily intervals, and the piperazine adipate is given at the rate of 10 grams per 100 lbs., in one dose with the first dose of phenothiazine. Bot eggs are removed daily with a razor when prevalent, and carbon bisulphide is dosed in liquid paraffin by stomach tube, just before the animals leave the farm, at a rate of 3 ccs per 100 lbs.

Diet

Dieticians seem to agree on the nutritional ratio, digestible protein, and dry matter requirements of horses, but there is great discrepancy on the subject of mineral requirements, especially the Ca:P requirements. Most workers seem to agree that the ratio Ca:P should be 1.2:1, but the amounts within this ratio, vary between 10 grams and 40 grams of calcium daily and 9 grams and 30 grams of phosphorus daily. The trace element picture is also rather hazy. Copper deficiency seems to be causing much trouble in some areas. I would advise a basic ration for all the animals on the farm, to which may be added a mineral and trace element mixture according to local requirements; however such a mixture should be avoided wherever possible. The grain ration could be made up as follows:

Whole oats: 80 parts	Wheaten Bran: 12 parts
Crushed Mealies: 4 parts	Rolled Barley: 4 parts
Linseed Oil Cake Meal: 1 part	Peanut Oil Cake Meal: 1 part

At weaning the animals receive 4–5 lbs. of this mixture, 2 lbs. chaffed lucerne hay, and as much teff hay as they will eat. Green

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feed, (barley, oats or rye) is cut for them and brought to them. 10–15 lbs. daily seems to be adequate. I would not advise grazing winter pastures, firstly because the youngsters seem to fill themselves up so much that they do not eat their rations properly, and secondly the moist conditions underfoot, which often go with winter pastures, cause the feet to soften and crumble.

Grazing during winter on lush green pastures seems to be related to a cause of club feet. Although there is no conclusive evidence, I have observed a definite mild coronitis on a number of foals, and in one case, an acute laminitis affect the youngsters an hour or two after being put on to a lush pasture. This coronitis subsides if they are taken off immediately. If this is repeated, marked laminitic rings will be seen on the walls of the feet after about a month or six weeks, and at the same time a seedy toe will develop. This chronic laminitis might cause some lameness and the feet are very brittle, and break very easily. If there is a severe seedy toe, and separation of the pedal bone from the wall of the foot, the pedal bone will rotate, the sole drop, and a club foot is the eventual result.

In the following spring, on good grazing, the youngsters might stay on 5 lbs. of grain a day and 2 lbs. of lucerne, or the ration might even be reduced if the animals get too fat. In November or December the grain ration will slowly be increased, up to 10 lbs. for the larger animals, with 3–4 lbs. lucerne hay, on which it remains until April or May when the yearling leaves the farm to go into a training establishment. At this stage injuries are very common due to kicks, etc., and it is advisable to run only two or three animals together.

BOOK REVIEW

RICHTER AND GÖTZE'S TIERGEBURTSHILFE

Gustave Rosenberger, Harry Tillman and 18 associated authors. 615 figs. and plates of which 71 in colour.
Pp. XIX 904 2nd Ed. 1960. Paul Parey—Berlin and Hamburg. Price D.M. 178.

This volume has a number of divisions each subdivided into chapters. The divisions include (1) History of Veterinary Obstetrics (2) Normal Pregnancy (3) Normal Parturition (4) Normal Puerpurium (5) Pathology of Pregnancy (6) Pathology of Parturition (7) Pathology of the Puerpurium (8) Pathology of the Foetus, Newborn and Suckling (9) Veterinary Obstetrical Jurisprudence.

The text being written by a number of prominent European Veterinarians in close collaboration, bears the stamp of authority. Most illustrations are reproduced photographs—the drawings are beautifully executed.

Various obstetrical and surgical operations are illustrated.

This book can wholeheartedly be recommended. It will certainly be one of the finest books on the veterinarian's bookshelf.

C. F. B. H.

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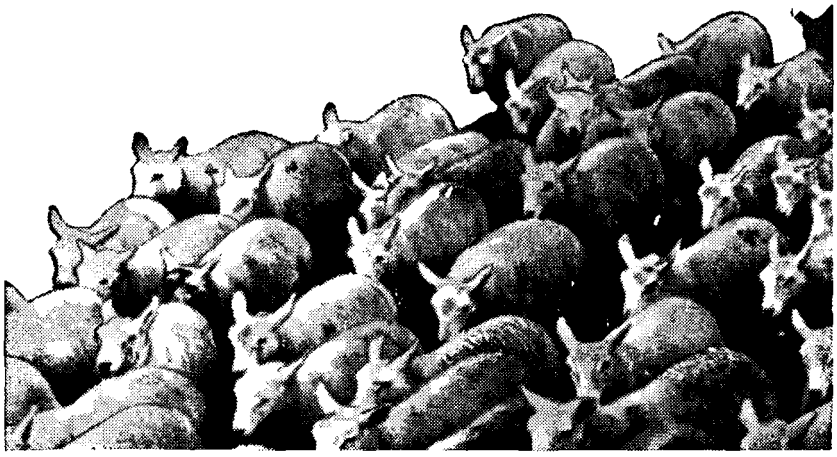
Chloromycetin Tincture 10% — a product of Parke-Davis original research — represents a remarkable advance in the treatment of contagious foot rot in sheep. Stewart, carrying out the first clinical trials in Australia[1], obtained over 80% clinical cure with one application and nearly 100% after the second. Similar success has been reported in England by Sambrook[2].

TREATMENT — Diseased tissue should be carefully pared away and Chloromycetin Tincture 10% applied with a stiff brush. Supplied in bottles of 2 and 16 fluid oz. with brush.

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

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

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MANAGEMENT OF THE NEWBORN PIG

R. K. Loveday*

The lossess occurring among suckling pigs prior to weaning represent what is probably the heaviest drain on profits which the swine industry has to bear. While mortality involving the whole litter occurs on occasion, the sporadic loss of two or three pigs from almost every litter is the usual pattern observed in most commercial herds. Some 80% of these preweaning losses occur within the first week of life, and are most marked among the lightest and least vigorous animals in the litter. That disease plays a minor rôle in these early deaths is evident from the findings of the most recent and comprehensive survey of preweaning mortality in Great Britain[1] where it was found that only 8% of losses during the first week were directly attributable to disease. Thus a study of the physiological peculiarities of the newborn piglet throws considerable light on the basic causes of mortality. The greatest single cause of loss is overlying by the sow.

About 5% of stillbirths occur among pigs, and the following figures have been cited: England: 6.04%[1]; Northern Ireland: 4.9%[10]; and the U.S.A.: 5.5%[21]. Extensive investigations to date have failed to reveal much explanation for this phenomenon, and laboratory examination in the British survey mentioned above provided no solution as to the cause of non-viability at birth in 86% of the material examined. It appears likely that pre-natal influences play a major rôle. Archibald and Hancock[3] considered iron deficiency in sows to be a probable cause of stillbirths, and found that the feeding of iron during pregnancy increased haemoglobin levels in the dams and was followed by the birth of normal living pigs.

In many parts of the world a remarkably constant figure of about 20% of pigs born alive is lost before weaning (Table 1). There are, however, numerous well-run herds where losses not exceeding 9–12% are the order of the day, providing striking testimony to the economic benefits to be gained from superior husbandry.

Physiological Considerations

1. At birth the pig is the most immature of all farm animals, being less than 1% of its mature weight; the comparative figure for the foal is 9%.[12] Lack of vigour and a low birthweight are closely related, and it has been shown that undersized pigs, with their relatively underdeveloped musculature, are more likely to be killed by the sow. In a study of an inbred Large White herd, Pomeroy[26] found that 83% of the piglets in the sample weighing less than 900 g. at birth died within the first three days; of the pigs weighing more than 1,000 g. but less than 1,400 g. at birth, 33.8% died within three days, while of the pigs weighing more than 1,400 g. at birth only 18.5% died

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TABLE 1
ESTIMATED PREWEANING LOSSES IN VARIOUS COUNTRIES

Reference	Country	Average number of pigs per litter			
		Born alive	Weaned	Lost	% Loss
25	Australia, 1953	9.1	7.2	1.9	20.9
31	Canada, Spring 1952	9.2	7.5	1.7	18.5
31	New Zealand, 1950–51	9.1	6.8	2.3	25.2
22	England, 1937	9.4	7.7	1.7	18.1
1	England, 1956–58	10.3	8.1	2.2	21.1
10	Northern Ireland, 1954	11.0	9.1	1.9	18.2
6	South Africa, 1952–54	9.2	6.9	2.3	25.0

within three days. In this strain of pigs there was a critical birthweight of 900 g. "and pigs below this weight had a very poor chance of survival". While improved husbandry (such as the provision of infra-red lamps) and superior mothering ability of sows reduce the environmental hazards and also the critical birthweight, it is always the lightest pigs which are most likely to be lost.

It seems probable that the sum of the genetic and uterine environmental influences of the sow will exert a greater effect on the birthweight of the piglet than the genetic influence of the boar. The low heritability of these factors suggests that selection for this characteristic will result in relatively slow improvement, while attention to sow management and nutrition is of considerably greater practical significance and is under the complete control of the breeder.

2. A peculiarity of the newborn pig is the delayed appearance of satisfactory gluconeogenesis, which becomes established only some three to four days after birth.[29] During this period the liver glycogen reserves are rapidly utilised, whereafter the blood sugar content is extremely unstable and fluctuates widely with the milk intake.[9] Fasting for a relatively short period at this age, whether due to failure to suckle or agalactia in the sow, results in the onset of hypoglycaemia and the appearance of symptoms of a metabolic upset. Unlike other species, the metabolism of the newborn pig appears to be largely governed by the blood sugar content. Weakness, hypothermia, bradycardia, convulsions and eventual coma occur, and such animals are very likely to be crushed by the sow. While hypoglycaemia is generally secondary to agalactia of varying intensity in the sow, "spontaneous hypoglycaemia" of unknown aetiology has been described where an adequate milk supply was available.[13]

Goodwin[9] has pointed out that any physical or morbid disturbance in the piglet, rendering it incapable of maintaining its milk intake, will have the same effect as starvation caused by agalactia, and the animal may die either from the hypoglycaemia or from some other cause, such as crushing or the primary disturbance.

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3. Further evidence of the physiological immaturity of the pig is the absence of satisfactory thermo-regulation at birth.[24] This mechanism only begins to show activity (vaso-constriction of cutaneous vessels) at about the ninth day and is reasonably perfect at twenty days of age.[14] The body temperature falls 3–13°F within the first thirty minutes after birth, dependent upon the temperature of the environment. A gradual return to normal occurs within two days at environmental temperatures of 60–75°F, while under freezing conditions this return to normal may occupy up to ten days.

An undersized piglet not only has a relatively larger body surface area than heavier littermates and thus loses heat more rapidly, but is also handicapped by a relatively underdeveloped musculature with which to generate heat. The lower the ambient air temperature and the smaller the piglet the more rapidly are the animal's glycogen reserves metabolised, the blood sugar level during the first three days thereafter being entirely dependent on milk intake. Chilling, therefore, hastens the onset of hypoglycaemia.

A further interesting observation made by Newland *et al*[24] was that chilling results in haemo-dilution and markedly lower haematocrit values. This is the opposite of what occurs in adult swine with normally functioning thermoregulation.

4. The foetuses of ungulates do not synthesise gammaglobulin in utero, nor does placental transfer from the dam's blood occur. The first normal and immune gamma-globulin is received from the colostrum and contains antibodies specific to the pathogenic bacteria in the sow's environment. Gut permeability to globulin is markedly reduced within twenty-four hours after birth, and no further absorption occurs after the third day.[30] Piglets raised without the benefit of colostrum show limited gamma-globulin synthesis at two weeks of age and reach normal values at about eight to ten weeks of age.

Rutqvist[28] has shown that following colostrum feeding the serum albumin level reaches a maximum at about four weeks, while the globulins decline progressively during the same period to four weeks of age, when they comprise only 5 to 6% of total serum protein. This age is, therefore, a most sensitive period for the animal's health insofar as infection is concerned. After four weeks a gradual increase in globulin content commences until a normal value is reached at about fifteen weeks of age.

5. Pigs are born with the so-called "needle teeth" fully erupted and comprising the central and lateral deciduous incisors and the deciduous canines. The deciduous molars and central incisors erupt during the first month.

6. A knowledge of the gastro-intestinal enzymic changes occurring during the early growth of the young pig is of value when formulating diets for orphan and early-weaned animals. Canadian studies[4, 15] indicate that of the carbohydrate-splitting enzymes, only lactase would appear to be fully active during the first week of life. After ten days of age amylase, sucrase and maltase become increasingly active to

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reach a maximum at about twenty-five days. This confirms the suitability of milk as an artificial feed for very young pigs, while starch, sucrose and maltose may be fed in greater amounts to pigs over one week old. For pigs under seven days old glucose gives better results than sucrose.

7. Piglets normally suckle about once every hour, day and night. There is much individual variation between the yield of the various teats and as each piglet adheres rigidly to its own teat from the third day of life, this variation may account for growth differences between littermates. The front pair of teats usually provide the highest yield with also a higher butterfat content than the rear teats.

Total milk production depends largely on the number of teats which are suckled, and it has been found that the total yield is approximately the same for litters of between five and ten pigs. Above ten in the litter each pig receives progressively less, while below five each pig receives progressively more milk.

Lodge[16] found the average milk yield of eight Wessex Saddleback sows, each recorded over three lactations, to be 993 pounds (or approximately 100 gallons) in eight weeks. Daily yields rose to a peak of 2 to 2½ gallons in the third week of lactation, thereafter usually declining steadily to the eighth week. He found the average milk composition to be as follows: total solids: 20%; protein: 5.7%; lactose: 4.7%; butterfat: 8.6%; and ash: 0.89%.

Sow's milk contains only about 2 p.p.m. of iron and this fraction is not increased by the feeding of extra iron to sows. There is a tendency, especially in liberally fed sows, for the fat percentage of the milk to be particularly high in the third week of lactation and to rise above normal levels again in the fifth week. The reason for these fluctuations is not clear, but it is possible that they may be a contibutory cause of nutritional scours. The composition of colostrum changes rapidly during the first few hours after farrowing, but the change to normal milk is not completed before the fifth to sixth day *post-partum*.

Management in Relation to Physiology

1. The majority of pigs lost during the first week are those of subnormal size and lacking vigour at birth. These losses are accentuated by unfavourable environmental factors such as starvation or chilling, which tend to raise the critical birthweight of a particular litter. In South Africa it is customary to regard around 3 lbs. as a satisfactory birthweight, while the "critical birthweight" for the average herd may be considered to be about 2 lbs.

Development of the mothering instinct varies in individual sows, and poor mothers may crush healthy normal piglets, but even the best mothers can hardly be expected to avoid crushing some of their offspring when they are farrowed in overfat condition under cramped or cold, draughty conditions. Overseas, farrowing crates have been found to provide optimum protection against crushing. During winter in the colder parts of the country measures to prevent chilling,

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such as the provision of brooder boxes, stoves, infra-red lamps or hot-water pipes, will most certainly reduce mortality. In general, farrowing house temperatures for piglets under two weeks old should not be allowed to fall below an absolute minimum of 45°F, if losses are to be avoided. The provision of sufficient dry, short bedding is generally neglected. Wood shavings, derived from timber treated with penta-chlorophenol or other wood preservatives are highly toxic for young pigs. Hypoglycaemia may be treated by the provision of warmth and the intraperitoneal injection of 15 ml. of sterile 5% glucose solution three times daily. Piglets that are able to swallow may be dosed with a glucose solution.

2. The importance of correct sow management and nutrition in relation to the production of heavy, vigorous piglets has been mentioned. The value of exercise for pregnant sows cannot be over-stressed. The ration fed during gestation should be of a high protein, low energy type, containing supplementary minerals and rich in vitamins. It is usual to feed such sows 1 to 1½% of their bodyweight as a daily feed allowance so that good, but not overfat condition is maintained. The most recent American specifications[5] for such a ration are shown in Table 2 below.

The ration should derive not less than one-third of its crude protein content from animal or marine sources to ensure the availability of essential amino acids and also vitamin B_{12}. It is desirable to include not less than 10% high quality lucerne meal in a gestation ration to serve as a source of carotene and also to reduce the digestibility and consequently the energy value of the ration.

TABLE 2

NUTRIENT REQUIREMENTS OF BREEDING STOCK[5]
(Expressed in percentage or amount per pound of total ration)

	Pregnant	
	Gilts	Sows
Liveweight, lb.	300	500
Expected daily gain, lb.	1.0	0.7
Protein and energy:		
Crude protein, %	15	13
T.D.N. %	70	70
Digestible energy, Kcal.*	1400	1400
Inorganic nutrients:	same for gilts and sows	
Calcium %	0.6	
Phosphorus %	0.4	
Salt (NaCl) %	0.5	
Vitamins:		
Carotene, mg.	2.5	
Vit. A, I.U.	1200	
Vit. D, I.U.	60	
Thiamine, mg.	0.5	
Riboflavin, mg.	1.5	
Niacin, mg.	5.0	
Pantothenic acid, mg.	6.0	
Vit. B_{12}, mcg.	5.0	

* Digestible energy calculated on the assumption that one pound of TDN has 2,000 Kcal. of digestible energy.

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The overfeeding of sows during the first week of lactation may lead to overproduction of milk or udder disturbance, and may be followed by digestive upset in the litter. The sow should not reach full ration until five to seven days after farrowing. Lodge[17] found that neither the milk yield nor the protein output in the milk appeared to be increased over the limited period of the study by the feeding of rations with a crude protein content in excess of 15%. He also found that there was no advantage to be gained from feeding in excess of the oft-quoted daily maximum allowance of 2 lbs. of meal for the sow plus 1 lb. for each piglet in the litter.[18]

Overlarge litters in excess of functional teats should be reduced by the transfer of the surplus pigs as soon as possible to another sow in a comparable stage of lactation. These piglets should be well mixed in a box with their new fellows for at least an hour before being presented to the foster-mother.

3. Agalactia or hypo-galactia, with subsequent loss of the entire litter, is a much-feared disaster at farrowing time. The most usual and destructive form of this syndrome has been conveniently named "*agalactia toxaemica*" by Ringarp[27] He includes under this title all the various names used in the literature to describe a very similar syndrome, viz. post-parturient or puerperal fever, puerperal septicaemia, milk fever syndrome, hypopituitarism, Bact. coli infection, mastitis, metritis or simply "agalactia".

The onset of *agalactia toxaemica* is usually within twenty-four to forty-eight hours after farrowing. A sow which has hitherto suckled her litter quite normally suddenly exhibits high fever, sluggishness, sometimes inability to rise, lack of appetite, usually a swollen udder and very often a white vaginal discharge. All interest in the litter is lost and where suckling is allowed it is found that milk secretion is very scanty or entirely absent.

Ringarp relates this syndrome to stresses resulting from poor sow management and nutrition, particularly lack of exercise and overfeeding just prior to parturition. In addition to endocrine and metabolic factors, he states that gastrointestinal intoxications and/or infections clearly form an integral portion of the aetiology. It is recommended that feed intake be reduced some five to six days prior to the expected farrowing date, and the occurrence of constipation be avoided by the substitution of half the reduced ration by wheaten bran.

Treatment with oxytocin, laxatives, antibacterial drugs and corticosteroids is prescribed. Steroid therapy has proved particularly valuable in promoting the rapid restoration of lactation and thus saving the litter. Since gram negative organisms (coliforms) are commonly encountered in the bacteriological investigation of such cases the use of penicillin alone is not recommended as an antibacterial measure.

4. It is the practice in some commercial herds to clip the piglet's needle teeth during the first twelve hours after birth. It is claimed that this measure reduces udder soreness in the sow and fight wounds

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among the litter. More harm than good can result from the careless performance of this operation if lacerated gums or broken tooth fragments are produced.

5. Cracked, worn farrowing sty floors often harbour pathogenic agents and helminth ova. It is possible that such conditions may expose a newborn litter to strains of pathogenic agents against which the colostrum has provided little or no immunity. Unsanitary conditions are largely responsible for the frequent losses from enteritis, coliform or streptococcal septicaemia, navel-ill, streptococcal meningitis, joint ill, spirochaetosis and oral necrobacillosis occurring in two to three weeks old pigs.

It is usual to wash down sows before admitting them to a disinfected farrowing pen in order to remove ascarid eggs from the udders, feet and quarters. It is safe to deworm sows with piperazine up to seven days before farrowing.

6. Microcytic, hypochromic anaemia is a man-made disease progressing with age and resulting from iron deficiency among litters raised on concrete without access to soil. Venn *et al*[32] have estimated that the piglet must retain about 7 mgm. of iron daily for normal needs and that sows' milk will supply not more than 1 mgm. daily. A piglet is born with a body store of about 50mgm. of iron and if reared on concrete requires early iron supplementation within the first week of life. The absorption of iron from the gut is very limited and where oral iron in the form of soil, paste, iron solution, etc., is utilised for piglets, it appears advisable to make these products available not less than once weekly for the first four weeks of life. Failure to appreciate the need for the repeated administration of oral iron has resulted in many litters suffering from sub-clinical anaemia and uneven growth.

Judged on the experimental results obtained here[20] and elsewhere,[7 11] iron-dextran intramuscular injections constitute a far more efficacious method of iron supplementation. The intestinal absorption of iron ceases when haemoglobin levels reach normal levels, although the iron-containing enzyme systems may still be deficient in iron. No such disadvantage attaches to iron-dextran injections, which are able to supply sufficient iron both for haemoglobin synthesis and enzyme systems. It is evident that the local sarcomata described in rats and mice following multiple injections of massive amounts of iron-dextran[2] will not develop in pigs as a result of the administration of two relatively small intramuscular injections.[8]

The iron content of S. African cereals is reasonably high, and for this reason the addition of iron salts to creep mixtures is not only unnecessary but exerts a harmful effect on the vitamins present in the ration. Beeson *et al*[5] have recommended for baby pigs that the creep ration contain 45 mgm. of iron and 4.5 mgm. of copper per pound.

7. After the third week of lactation the sow's milk yield begins to decrease and very soon supplies insufficient energy for the continued optimal growth of the litter. It is usual to commence the feeding of a high protein (18—20%), high energy (80% TDN), low fibre creep

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feed at this stage, espedially where high weaning weights at 8 weeks are desired. Lodge and McDonald[19] found that by far the greatest influence on weaning weight was exerted by supplementary feed consumption, and the influence of sows' milk consumption declined rapidly from 3 weeks to 8 weeks of age.

8. The handrearing of newborn piglets requires methods rather similar to those employed in brooding chicks, viz. heat, hygiene and a suitable diet. Where colostrum has not been available, the feeding of serum obtained from adult animals forms a useful substitute, but requires to be fed within the first 24 to 36 hours of life.

Piglets have been removed from the sow after suckling at 4 hours of age and successfully raised at Onderstepoort under an infra-red lamp (70°F floor temperature) using plain cow's milk fed cold and ad lib. in a trough. The milk may be improved nutritionally by the addition of one raw egg per quart. The piglets were starved for 4 hours after removal from the sow and were forcibly introduced to the milk once only. All groups learnt to drink within twelve hours and no digestive upsets were encountered. No water is offered until the commencement of dry feeding. A gradual change over from milk to a commercial calf milk substitute ration is begun at about 10 days of age and completed in 3—4 days. On the attainment of a liveweight of 25 pounds a change to a creep ration is carried out in a similar fashion. Utensils used required to be boiled once daily, and the sty was washed out once daily using a quarternary ammonium disinfectant and detergent. Iron-dextran injections were administered at 3 and 10 days of age.

The raising of colostrum-free piglets for the production of completely disease-free animals has been described by Young and Underdahl[33],[34]. This technique involves the procurement, utilising a special aseptic hood, of pigs by hysterectomy some 2 to 4 days before term, and their rearing in separate isolation incubators until one week old. During this period they are fed cows' milk fortified with egg and a mineral mixture. From one week to four weeks of age they are housed in isolation brooders in groups of eight to twelve animals. This method is now being used commercially in Iowa, U.S.A. for the production of disease-free pigs for sale to local farmers.[23]

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BOOK REVIEW

THE VETERINARY ANNUAL

Editor: W. A. Pool, M.R.C.V.S.
Bristol: John Wright & Sons Ltd.

Research has made tremendous strides during the post-war years and this progress has been accompanied by the appearance of infinite numbers of publications giving essential information about current progress. Research workers find difficulty in keeping abreast with the literature of interest in their own fields but especially veterinary practitioners have neither the time nor the library facilities to remain well-informed on matters beyond the scope of a few journals. I, therefore, believe that practitioners will find the Veterinary Annual a particularly useful book. It gives essential information about current progress on virtually every aspect of veterinary science. The most important work done during the last two or three years is summarised briefly, although on some aspects more details are provided.

In the first part of the book an account is given of the current trends in veterinary activities in different countries. The rest of the book is devoted to reviews of subjects such as the following:

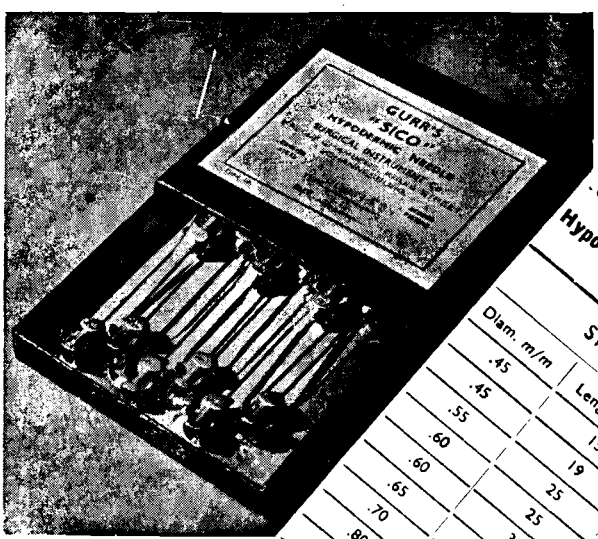
Bacterial, fungal, protozoal and virus diseases; parasitology; metabolic and reproductive disorders; radiation and radiation hazards.

Very useful references are given at the end of each chapter and at the end of the book are listed short descriptions of recent pharmaceutical preparations and appliances.

B. C. Jansen.

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REARING OF PUPPIES

J. R. Frean.*

Pups are among the easiest domestic animals to rear. A bitch in good health and condition and fed reasonably well during pregnancy will produce her pups and rear them with the minimum of trouble up to near weaning when it becomes necessary to gradually augment her waning supply and the pups' increasing demands as they grow larger and become more active. Care after weaning demands little attention beyond regular feeding.

The Mother

The health and robustness of the pup starts before it is born. The bitch should be in good health and condition and receive moderate exercise right up to the time of whelping.

At the Hound Breeding Station at Robertson all dogs receive a ration of meat and pumpkin stew and steamed yellow maize meal fortified by fish meal, ground, dried seaweed, dicalcium phosphate, brewer's yeast and cod liver oil.

Pregnant bitches receive cow's milk *ad lib.* as well.

The daily allowance per dog is:

Meat	¼lb. to ½lb.
Maizemeal	2lb.
Pumpkin.....	6oz.
Fish meal	1oz.
Dicalcium phosphate	1/2 oz.
Ground seaweed.....	1/16th oz.
Brewer's yeast.....	1/16th oz.
Cod liver oil.....	1/16th oz. (on alternate days)
Salt	1/3rd oz.

The food is cooked in fifty feed lots. A stew of meat, pumpkin, dicalcium phosphate, salt and water is made separately and the maize meal steamed so that when cooked it is crumbly like the Zulu's "putu" and not as porridge. When cool the fish meal, seaweed, brewer's yeast and cod liver oil are stirred into the meal.

At feeding time the meal is placed in flat troughs and the stew and gravy mixed into it. The hounds eat this with relish and do well on it, despite the meagre meat ration.

When pumpkin is not available, carrots or beet and sometimes even cabbage, green beans or onions are used.

A secluded spot for whelping should be provided and should be easily accessible for inspection. The floor is dusted with 2% D.D.T. powder and covered with grain bags or other suitable material to protect the pups from contact with the powder. The bitch's bed should be of grain bags or blanket rather than straw or hay in which

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pups are liable to get entangled and which also render cleaning by their mother more difficult.

She should not be disturbed during whelping, beyond receiving an occasional unobtrusive inspection by some well-known person to note progress. After whelping she requires little attention beyond ensuring a liberal supply of milk additional to her ordinary ration as well as a constant supply of clean water.

The Pups

Up to the third week the normal bitch will provide all they require in food and warmth. She will "change their nappies" and so prevent soiling by faeces and urine. At four weeks her supply of milk will start decreasing while the pups' requirements will steadily increase by reason of their added size and energy. One hears and reads of many special milk substitutes for supplementing her supply but it is doubtful whether anything better than ordinary cow's milk is required. This is given in small repeated feeds which pups soon learn to lap. They should be fed in a wide shallow dish with perpendicular walls to permit easy access and equal distribution with a minimum of splash and soiling. A pup will go on feeding almost to bursting point if allowed, in which case diarrhoea and even gastro-enteritis could ensue. A sound practice is to give small repeated meals rather than occasional large ones. The size of the meals is increased as the pups grow and by the sixth week they can be reduced in number to three of four per day, giving enough for each pup to have a reasonably good meal without undue distension of the abdomen. A healthy pup is naturally greedy and will overeat if given the opportunity.

Fom five weeks a little oatmeal porridge should be stirred into the milk and the amount gradually increased so that at seven to eight weeks the feed consists of equal parts of milk and porridge. It is possible to wean at six weeks but, if allowed, the bitch will continue feeding them up to eight or nine. Apart from saving in food and attention thus effected, it is desirable to have her educate and discipline as well as exercise them. She should, however, be provided with an "escape" in the form of a shelf or platform out of their reach for respite from their constant vigorous demands for milk, which are rendered all the more unpopular because of the needle-like teeth and claws which often produce small but painful lacerations of her mammary glands.

From the fourth week a small quantity of raw meat can be given, starting with a lump of mince half the size of an acorn and gradually increasing until at eight weeks each pup receives about 2 oz. per day. At this stage it can simply be sprinkled over one feed of porridge and milk.

The pups are weaned at eight weeks but it is desirable to keep them another ten to fourteen days before distribution.

All along the size of the meal is increased daily. The meat can be diced into one-inch cubes and as their teeth and jaws develop it is desirable to give raw bones with meat attached for them to tear off and

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gnaw. Hereafter, they can receive portions of their parents' rations but should continue receiving milk up to six months.

Disciplining of pups should start as soon as they are weaned and not before. As they normally are always hungry when awake, their disciplining should be at meal times, being allowed to feed as reward for obedience.

A pup urinates upon awakening and he defecates as soon as he has had a meal. Knowledge of these fundamentals will help considerably in house training. Guided in a friendly manner in the early stages he soon learns to go outside to "do his business". And if he makes a wet patch by mistake, drop a page of newspaper over it, and do not punish him.

Docking and the removal of the dew claws should be done at about the seventh day—an ideal instrument is a pair of carpenter's pincers—and at twelve weeks anti-distemper vaccination is performed.

The Orphan Litter

The veterinarian is consulted more often in respect of the orphaned litter than about healthy pups with a good mother. Except in the case of very valuable animals it is doubtful whether the successful rearing of such a litter will adequately reward the time, trouble and patience it entails. Unless one knows the owner to be steadfast and painstaking, or the value of the litter to be such as to compensate adequately, it is best to advise destruction at the time of the mother's death, all the more if she has died before the pups have been able to take a feed of colostrum. In this case the pups will most probably die in a few days despite every care and attention. It has been reported that blood-serum can substitute for colostrum.

Rearing an orphan requires a substitute for dog's milk; provision for the constant warmth that would ordinarily have come from the mother; a three to four-hourly feeding routine throughout the day AND night for two full weeks; and constant attention to clean up the pups after soiling with faeces and urine.

Milk Substitutes.

According to Smith, dog's milk contains about twice as much fat and thrice as much protein as cow's milk and one-tenth less water.

In a personal communication Dr. M. de Lange told me of the successful rearing of a litter of Boxers with powdered milk added to ordinary milk. Groenewald recommends 20 parts dried whole milk to 80 parts water, but his first choice is apparently: $\frac{1}{4}$ oz. cream and 2 oz. casein added to 4 oz. whole milk. He also states that "success has been claimed with rearing on condensed milk from the fourth day". The mixture the writer has recommended as successful is: the albumen of one egg and $\frac{1}{2}$ oz. cream added to 8 oz. whole milk.

If the pup has had a feed of colostrum it is probably here also quite unnecessary to "augment" good cow's milk or, say, "top milk". The secret of success lies in the manner in which it is fed, viz. in small frequent feeds at body temperature regularly throughout the 24 hours for two weeks. This is best given with a fountain pen filler or a

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doll's feeding bottle. Thereafter the feeds, and the intervals between them, can be increased gradually. For the first week no feed should exceed a teaspoonful and increased very gradually up to two teaspoonsful by the end of the second week; then up to four teaspoonsful by the end of the third week and continued until the end of the fourth week. Thereafter the survivors are treated as in the case of pups with mothers.

It is most important that the pup BE KEPT WARM. Otherwise feed and pamper as you will, he will die! And lastly, a healthy pup defecates and urinates often and he must therefore be kept constantly clean and dry. This calls to mind Punch's advice to those about to get married, viz. "Don't!".

Deworming

Anyone acquainted with articles on the rearing of pups will have noted that thus far no mention has been made of the administration of anthelmintics. Almost without exception one is told to "deworm at weaning" and again at 12 weeks and thereafter at regular intervals varying in length according to the fancy of the writer . . . but invariably one must deworm!

The writer has kept dogs since childhood but cannot recollect ever having had to administer an anthelmintic to one of his dogs either as an adult or a pup. But he has frequently had to attend to unfortunate pups suffering from the misguided ministrations of the owners on the advice of "doggy" people and chemists. That a heavily infested pup requires anthelmintic treatment is a matter of common sense, but to assume that because a pup has reached a certain age indicates that he must be subjected to the administration of the drastic nauseating drugs is not.

Acknowledgements

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Books Consulted: Veterinary Physiology, Sir F. Smith; Fauna and Flora No. 5, Transvaal, 1954, D. Compion; Dogs and their Management, Alfred Sewell, revised by Hamilton Kirk; Handleiding oor Dierevoeding, J. W. Groenewald; The Popular Alsatian, Jos. Schwabacher; To Own a Dog,Eric Harrison.

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NAME OF ANIMAL F. VETALENTA 2ND

DATE	REMARKS
7·10·52	Calved (Heifer calf)
1·1·53	Served (B. Flash Prince)
9·10·53	Calved (Bull calf)
27·12·53	Served (B. Flash Prince)
17·1·54	" " " "
12·2·54	" " " "
	Injected 'Lutormone' — 1500 I.U. I/V.
28·2 54	Injected 'Protormone' — 10 c.c. I/m.
19·11·54	Calved (Bull calf)



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ARTIFICIAL INSEMINATION UNDER RANCHING CONDITIONS

I. G. Horak*

Before dealing with the subject of insemination it is necessary to give some background as to the ranch itself. The ranch is approximately 30,000 acres in extent and is situated in Northern Zululand twenty miles South of the Swaziland border. The rainfall varies between 12 and 20 inches per annum and the grass types are mainly Rooigras and Panicum species, the carrying capacity varies between 7 and 9 acres per beast. The ranch is divided into two sections, the southern section is used for summer grazing and A.I. paddocks and is situated in flat country, the other section is situated along the Mkuze River and in the hills and is used for all-year-round rotational grazing for pregnant cows, and heifers and oxen. The present cattle population of the ranch consists of 1,400 cows, 250 heifers ready for breeding, 350 younger heifers, 1,000 oxen, 20 bulls and 650 calves.

The subject may be divided into the following sub-headings:— The Cows, The Bulls, The Semen, The Inseminations, The Calves and Labour, a factor which cannot be underestimated in any ranch A.I. scheme.

The Cows

When starting off on a programme like this, one has to decide between running small or large herds of cows. In the light of American experience, where large herds of cows have to be run due to labour shortage and the ranch A.I. conception figures are low, it was decided to run small herds of cows consisting of approximately 70 to 100 cows per herd. Each of these herds is grazed in a paddock of 300 to 800 acres in extent for the duration of the A.I. season. Each paddock is served by only one watering point to facilitate collection of the cows and each paddock also has a collecting pen, an insemination pen, and a crush-pen. Where possible one drinking point supplies more than one paddock and in this way one crush-pen suffices for as many as four herds.

The cow types present in the programme are Afrikanders, Drakensbergers, Zulus and a few Frieslands, there are also crosses between bulls of Hereford, Shorthorn, Drakensberger, Aberdeen Angus, and Sussex breeds and Afrikander cows. To assist in the identification of cows for insemination record purposes each cow is branded on the right ribs with a number ranging between 1 and 1500. Each successive year the heifers, put into A.I. paddocks, are branded with a letter preceding the number on the ribs, so that the various age groups can be recognised readily by their letters. Each cow also has a correspondingly numbered plastic disc in her left ear. The cows running with calves carry a coloured plastic disc in their right ears indicating the 40 day period during which their calves were born. Their calves carry a similarly coloured disc in their right ears. All cows with calves born in a particular 40 day period are run in the same herd,

* "Quest Ranch", Mkuze, Zululand.

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with their calves, hence if a calf becomes lost one can easily identify its herd of origin by looking at the colour of the disc in its ear.

Nymphomaniac cows, created by repeated injections of stilboestrol, were at first used to assist in the identification of on-heat cows, but were found to be unsatisfactory, for once having located an on-heat cow they followed her only and failed to indicate any other cows. A number of young bulls have now been vasectomised for use as teasers, but have not been used as yet. After a few weeks' experience with an A.I. herd under the supervision of a more experienced boy, the normal herd boy becomes quite efficient in identifying cows on heat, and this is the only method of identification used on the ranch at present.

The Bulls

In 1958, the first season of A.I on the ranch, one Hereford, one Drakensberger, one Sussex, one Africander and three Brahman bulls were used for A.I. purposes. In the 1958-1959 season semen from one Simmenthaler, five Brahmans and a Charollais bull was used, the semen from the latter bull was flown from Pretoria. During the 1959–60 season two Brahmans, one Africander, one Limousine and two Charollais were used, the semen from the latter four bulls was flown from Pretoria.

The ranch A.I. bulls are kept at the ranch A.I. station; some of the Brahmans prefer to be penned together as they are by nature gregarious. For insemination purposes each bull has a code number, this facilitates record work considerably.

The Semen

Three methods of semen collection have been used. The Simmenthaler bull needed no training to use the artificial vagina and semen from this bull was collected by this method. The Brahman bulls refused to mount a cow in a crushpen and consequently the artificial vagina cannot be used with them. Two of the Brahmans have been trained to ejaculate into the artificial vagina after hand massage of the penis through the skin of the prepuce and then directing the erect penis into the artificial vagina. However the results obtained from using semen collected by this method have been poor.

The third method of collecting semen is by electrical stimulation of the ampullae of the vas deferens, and this method is used exclusively on the ranch at present. The electrical current is applied to the ampullae via the rectum by means of electrodes for five to ten second periods, with a 5 to 10 second rest between applications. The clear fluid, which is practically invariably secreted at first, is discarded, and then the semen is collected. Some bulls give better semen if the electrical current is re-applied after a five to ten minute rest after all the clear fluid has been drawn off.

The semen obtained by this method varies between 2.5 ml. and 24 ml. in volume, and the sperm density varies between 100 million and 700 million spermatozoa per ml. Some bulls give better results when worked once a week and others when worked twice a week.

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For instance bull No. 4B gave a conception rate per insemination of 48.6% when used once a week, when used twice a week, the conception rate for the same bull was 62.3%.

The semen is diluted in a 50:50 mixture of egg yolk and sodium citrate buffer solution so that the final dilution does not contain less than 20 million live spermatozoa per ml. The semen is then poured into 5 ml. test tubes and cooled in thermos flasks containing ice. This semen is suitable for use within the next three days after which its viability falls off rapidly.

Inseminations

At the start of the first A.I. season on the ranch in February 1958, each cow was inseminated once per heat-period at the time of heat, and only one insemination-round a day was made. The first pregnancy test, however, revealed a very alarming state of affairs with only 15 to 20% of the cows inseminated having conceived. It was then decided to make two insemination rounds a day and inseminate each cow three times per heat period, should delayed ovulation be the cause of the low conception rate. Individual cows were then examined for delayed ovulation and it was found that some cows ovulated as late as 60 hours after they were seen on heat. The three inseminations per heat period were performed at 12 hour intervals, the first insemination being performed when the cow was seen on heat, the second twelve hours later, and the third twelve hours later again.

A further difficulty which occurs when such a diversity of breeds is used for A.I. on a ranch is that the length of the heat-period of the various breeds varies considerably, ranging from two to eight hours in the Zulus to 12 to 24 hours in the Frieslands. Three inseminations per heat-period also eliminates this difficulty, for even though a cow may not ovulate late she may have a long heat-period characteristic of her breed, and hence she will be more likely to conceive to the third insemination.

Each herd has two herd boys, excepting herds drinking at the same drinking trough, where one boy per herd suffices, unless the paddock is exceptionally large. Herds are collected at half-hourly intervals in the morning and afternoon, this allows time for insemination and travelling between herds. The cows to be inseminated are sorted out and put into the crushpen and the other cows are allowed to go. The morning round of inseminations commences at about 7.30 a.m. and the afternoon round at 4 p.m. Semen is collected before the morning round.

The following results are based on figures obtained during the 1958–59 and the 1959–60 season. Unfortunately the results for the two seasons cannot be compared, as during the 1959–60 season semen from one of the bulls used gave very variable results and hence there is a considerable difference between the results obtained in the two seasons. Various methods of insemination have been tried experimentally and the following results were obtained.

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1958–59 *Season*

51 heat periods had only one insemination per heat period and this insemination was performed at the time the cow was seen on heat: only 11 of the cows thus inseminated conceived, a percentage of 21.6.

127 heat periods had two inseminations per heat period, the first insemination being performed when the cow was first seen on heat and the second twelve hours later: 66 cows conceived to this method, a percentage of 52.

97 heat periods had two inseminations per heat period, the first being performed when the cow was first seen on heat, the second twenty-four hours later; 48 cows conceived, a percentage of 49.5.

693 heat periods had three inseminations at 12 hourly intervals per heat-period and 332 cows conceived, a percentage of 47.9.

From these results it can be seen that two or three inseminations per heat period give far better results than a single insemination performed at the time of heat. This can be ascribed to the delayed ovulation present in the herd. On the strength of this result the first insemination was omitted during the 1959–60 season and during this season the cows were inseminated twelve hours after being seen on heat and again twelve hours later.

1959–60 *Season*

37 heat periods had two inseminations per heat-period, the first being performed when the cow was seen on heat and the second twenty-four hours later: eight cows conceived, a percentage of 21.6.

68 heat periods had a single insemination twenty-four hours after the cow was seen on heat: 24 cows conceived, a percentage of 35.3.

194 heat-periods had a single insemination twelve hours after the cow was first seen on heat: 56 cows conceived, a percentage of 28.9.

675 heat-periods had two inseminations, one twelve hours after first seen on heat and the second twelve hours later: 249 cows conceived, a percentage of 36.9.

From these results it appears as if the third insemination, namely the one performed twenty-four hours after the cow was first seen on heat, is the most important and the most likely to bring about conception because of the delayed ovulation present.

The following first insemination results were obtained during the 1958–59 season by placing the semen in various parts of the genital tract of the cow.

370 first inseminations into the cervix and entrance of the uterus yielded 181 takes, equal to 48.9 percent.

There were 60 first inseminations into one or both horns of the uterus for every insemination during that particular heat-period and 35 cows conceived, a percentage of 58.3.

57 first inseminations into the cervix only yielded 36 pregnancies, a percentage of 63.2.

This latter figure is consistent with the fact that semen deposited in the cervix is more likely to bring about pregnancy than any other

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method of insemination, whereas if the other figures are correct the horns of the uterus are a better place to deposit semen than in the body of the uterus.

The following observations were made in connection with the correlation of conception and the length of the oestrous cycle.

137 oestrous cycles were 19 to 22 days long, which is considered normal; of these cycles 52 preceded conception after artificial insemination, a percentage of 37.9.

73 oestrous cycles lasted 10 to 18 days or 23–31 days; 18 of these cycles preceded pregnancy, a percentage of 24.7.

26 oestrous cycles were 21 days long and 16 of these preceded pregnancy, a percentage of 61.5.

From these results it appears that abnormally long or short oestrous cycles are not as favourable for ensuring pregnancy as cycles of a normal duration.

The conception rates for the various months during the 1958–59 seasons were as follows:—

December 1958:—129 inseminations resulted in 58 conceptions, a percentage of 45.1.

January, 1959:—230 inseminations, 125 takes, equal to 54.3%.
February 1959:—163 inseminations, 92 takes, equal to 56.4%.
March, 1959:—253 inseminations, 111 takes, equal to 43.9%
April 1959:—151 inseminations, 70 takes, equal to 46.4%

From these results it would appear that February was the most fertile month of the season.

The conception rates for the various months during the 1959–60 season were as follows:—

November 1959:—88 inseminations, 33 takes, equal to 37.5%.
December 1959:—144 inseminations, 68 takes, equal to 47.2%.
January 1960:—258 inseminations, 69 takes, equal to 26.7%.
February 1960:—276 inseminations, 91 takes, equal to 33%.
March 1960:—216 inseminations, 62 takes, equal to 28.7%

It must be stressed, however, that during January, February and March semen from the aforementioned bull, which gave poor results, was used more regularly than during November and December, taking this into consideration it again appears that February was a fertile month.

The following observations were made on the average number of cows coming on heat daily during the various months of the 1959–60 season as compared to the average daily ideal of 1/21st. of the non-pregnant cows of the herd. (As the oestrus cycle of the cow is 21 days long theoretically an average of 1/21st. of the non-pregnant cows in a herd should come on heat every day.)

On January 11th and February 7th more cows were brought into the A.I. herds, hence the increase in the number of non-pregnant cows in the herd on these two dates.

From these findings January and February are the months during which the greatest number of cows comes on heat daily, and tying this up with the previous results in which February proved to be the most

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TABLE 1

Month	No. of non-pregnant cows	No. of cows coming on heat	No. of insemination days	Average daily no. of cows coming on heat	Ideal 1/21st of herd	Average daily %age coming on heat compared to the ideal
Nov. 59.....	456	91	21	4.333	21.714	19.9
Dec. 59.....	423	138	21	6.571	20.143	32.6
Jan. 1–10, 1960	355	88	10	8.80	16.905	52.1
Jan. 11–31, 1960	427	160	21	8.048	20.333	39.5
Feb. 1–6, 1960	382	50	6	8.333	18.190	49.0
Feb. 7–29, 1960	395	211	21	10.048	18.810	53.4
Mar. 1–21, 1960	322	141	21	6.714	15.333	43.8

fertile month, it seems obvious that the A.I. season, in the particular part of Zululand in which the ranch is situated, must be concentrated around the month of February.

The following relationships between left and right ovary ovulation and pregnancy in the left or right horn of the uterus were established during the 1959–60 season.

449 ovulations occurred from the right ovary, 331 from the left, hence 57.5% of the ovulations occurred from the right whereas 42.5% occurred from the left.

Of the 449 right ovary ovulations 147 preceded conception, a percentage of 32.8.

Of the 331 left ovary ovulations 112 preceded pregnancy, a percentage of 33.8.

Hence left or right ovary ovulation does not significantly affect the conception rate one way or the other.

In 15 cases where the follicle was diagnosed as being in the left ovary the foetus was later found in the right horn of the uterus.

In 6 cases where the follicle was diagnosed as being in the right ovary the foetus was later found in the left horn.

A possible explanation for the greater number of foetal migrations from the left to the right side than vice versa is the proximity of the rumen to the uterus on the left side and hence the ruminal movements could influence foetal migration.

A comparison of first insemination conceptions between herds of cows running with their calves and herds of cows without calves, the latter group of cows having calved at least 9 months previously, gave the following results:

201 cows running with calves were inseminated and 114 took on the first insemination, a percentage of 56.7.

223 cows without calves were inseminated and 80 took on the first insemination, a percentage of 35.9.

From these results the conclusion can be drawn that cows which have had a long sexual rest do not conceive as easily as cows which have recently given birth.

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A comparison was made of insemination results obtained from using semen from two Brahman bulls kept under identical conditions. No microscopic difference could be found in the semen of these two bulls excepting that Bull No. 5B had a slightly higher motility rating. During the 1958–59 season Bull No. 4B's semen had an average motility of 4.5, whereas Bull No. 5B had an average motility of 4.6. The average ml. of Bull 4B's diluted semen contained 34 million live sperm, whereas 5B's diluted semen contained 34.4 million live sperm per ml., and yet 4B gave an insemination conception percentage of 52.2 whereas 5B's conception rate was 43.2%. This seems to be quite a common phenomenon for which there is not always a ready explanation.

Pregnancy tests are started after 2½ months of insemination and one herd per day is tested. The pregnant cows are moved to a different paddock so that they and their calves do not have to go through the herding necessary twice daily for A.I. After the initial test pregnancy tests are performed every four to six weeks and the newly pregnant cows are moved to different paddocks.

Insemination results were as follows during the 1958–59 season:

612 cows were inseminated and 345 conceived to the first insemination, a percentage of 55.5; overall there were 474 conceptions irrespective of the number of inseminations, a percentage of 77.5.

Insemination results during the 1959–60 season, up to and including March:

501 cows were inseminated; 230 conceived to the first insemination, a percentage of 45.9.

337 cows conceived irrespective of the number of inseminations out of the total of 501, an overall percentage of 67.3.

After the A.I. season the other bulls which have not been used in the A.I. programme are put with the cows to serve those which were not inseminated or did not conceive to insemination.

The Calves

When a calf is born a plastic disc with a letter, indicating its year of birth, and a number is affixed to its left ear; an identical letter and number is also tattooed in its left ear. In its right ear a coloured plastic disc indicates the 40 day period during which it was born. The calf's number, date of birth, sex, type, sire's and dam's numbers are entered into the calf performance testing records and on its mother's performance card.

When a group of calves born during a particular 40 day period reaches an average age of 225 days, the whole group is weighed, dehorned, branded, dosed for worms, inoculated for quarter evil and the heifers are inoculated with C.A. vaccine. The calves' weights are converted to a mean 205 day weight and the calves are then divided into three groups according to their 205 day weights. All bull calves not falling in the top weight group are castrated.

At the start of the A.I. season the replacement heifers are branded with their numbers, dosed for worms and inoculated again with C.A. vaccine as well as being weighed again. The older cows are tested for pregnancy and the non-pregnant ones are sorted into herds and

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sent to the A.I. paddocks. Cows with young calves are not put into the A.I. paddocks until their calves are approximately 2 months old.

Labour

The herd boys are housed as closely to their herds as possible and are responsible for dipping their herds, treating maggot infested sores and seeing that the cows have water. To demonstrate the importance of the herd boy in an A.I. programme, the following conception figures of two adjoining herds of cows during the 1958–59 season are enlightening. The one herd, herded by a boy with extensive experience had an overall A.I. conception rate of 84.7%, whereas the neighbouring herd had a conception rate of 75.2 percent. In addition to their normal wages, the A.I. herd boys are paid a bonus for each cow in their particular herd that conceives to insemination.

Finally it must be stressed that the A.I. programme is only secondary to a performance testing programme.

BOOK REVIEW

"THE INSPECTION OF FOOD"

H. Thornton,

Chief Veterinary Officer, City & County of New Castle 2nd Edition: 1960. Published by Bailliere, Tindall and Cox, London.

This publication, described as "A Handbook for Students of Public Health, Agriculture and Meat Technology" is a revised edition of a wellknown handbook. It is not primarily intended for veterinary students (both graduate and undergraduate) and cannot replace the accepted textbooks on meat and milk inspection written by Dr. Thornton himself and others and in which the subjects are dealt with more thoroughly. It is however, a most excellent "ready reference" and covers a wide field in a concise, precise and clear manner. The subjects dealt with are Anatomy of Food Animals, Slaughter and Dressing, Bacterial and Parasitic diseases, Physiological and Pathological conditions, Prepared meats, Preservation of Foods, Post-mortem changes, Poultry, Eggs, Game, Inspection of other Foods, Fish & Shellfish, Milk & Milk products and finally a list of questions for examination purposes.

The book covers some 200 pages, is printed on good paper, the type and illustrations are clear, and costs about 15/-.

L. W. v .d. H.

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SOME REMARKS ON THE PROBLEM OF POOR FERTILITY IN DAIRY HERDS SERVED BY THE NATAL ARTIFICIAL INSEMINATION CO-OPERATIVE

D. J. le Roux,*

As indicated by the heading of this paper, it is not intended or possible to give a learned scientific discussion on the great many factors which may influence the fertility of cattle. An endeavour will be made to reiterate some of the most common and practical problems which the author has come across, in the course of his duties when dealing with herd fertility work.

The problem is basically one of poor or wrong general management, which directly or indirectly affects the fertility of individual animals, or that of a herd as a whole.

The following are the most important factors to be considered:—

1. *The keeping of records*

The lack of a proper record keeping system reflecting the history of all individual animals in a herd, gives rise to a lot of management errors, especially as regards the maintenance of the reproductive health of the herd or individual animal, in the following respects:

(a) Heifers are allowed to become too old before they are first bred, giving rise to trouble with delayed breeding.

(b) The length and regularity of the oestrus cycles of individual animals are not known.

Irregularities in this respect give a very good indication of the normality or otherwise of the animal, and may give an early indication of breeding trouble arising in a herd.

(c) Calving dates are not known, so farmers are not able to breed back their cows at their optimum fertility period, plus/minus 75 days after calving.

(d) Since the number of services required to settle a cow is not known, no timely steps are taken to investigate or remedy a problem arising. The inherent fertility of different cows or families is not known, so very little culling is done on the basis of fertility.

(e) Records of specific or general conditions which may influence fertility, for instance retained afterbirth, metritis, and febrile conditions are not kept or taken into consideration to ensure that a cow is fit to conceive within three months after calving.

2. *Feeding*

The influence exerted by nutrition on the fertility of dairy herds, cannot be overstressed. Essentially, farmers do not seem to be able to maintain an even supply of the essential nutrients for the maintenance

* Natal A.I. Co-operative, Howick.

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of both production and reproduction in the mature animals, and to maintain an even growth and development of their young stock.

The most important considerations seem to be:

(a) *Protein.*—Concentrate rations are usually mixed on the farm, and consist of 2 or 3 ingredients only, mostly too low in protein content. This is not fed on the basis of milk produced, with the result that especially the higher producers, either suffer from anoestrus for a long time after calving or, if and when they come on heat, are difficult to get into calf. An example of a herd of very high producing cows may be quoted. The conception rate in this herd was very poor, and the farmer could not afford a higher plane of nutrition. It was decided, instead of breeding cows at the accepted 2½ to 3 months after calving, to start inseminating at the second heat period, which is plus/minus 6 to 9 weeks after calving, and before the animals were in the peak of production. Since this policy has been followed, a considerable improvement in the conception rate has been obtained.

(b) *Supplementation of Phosphates.*—Although most herds are in fairly high rainfall areas where a deficiency of phosphate is known to exist, it is surprising how few farmers give any phosphate supplementation to their stock. Excellent results have been obtained with the supplementation of phosphates in the form of a lick, mixed with molasses.

(c) *Cultivated Pastures.*—The intensive and indiscriminate use of pastures in certain areas, seems to play a role in depressing the fertility of herds.

Two factors are to be considered:

(1) When clovers predominate, symptoms of a hormonal disturbance (excess of oestrogen?) may appear, which definitely depresses fertility. Symptoms shown are the following: Long, intense heat periods, often at short intervals. On internal examination, excessive follicular development; the uterus feels engorged and tense, as though the animal is on heat even during di-oestrus. On speculum examination, the mucous membranes are abnormally engorged, and during oestrus copious amounts of mucus, often containing specks of blood, are excreted.

(2) (i) Where cows have to exist mainly on rich, lush pastures for a considerable time, a protein/carbohydrate imbalance seems to arise. The animals milk well, but are extremely thin, and great difficulty is experienced in getting them into calf. The supplementation of some source of carbohydrate, for instance yellow mealie meal, usually improves the condition. A ration on which cows produce well, does not necessarily ensure a satisfactory breeding ability.

(ii) Continuous feeding on such pastures to the exclusion of all other forms of roughage, often leads to such animals continuously suffering from what could be termed a diarrhoea. This probably is a contributing factor to their general poor condition.

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Where such animals also have access to some form of a coarser rouhage, for example veld hay, this abnormal physiological manifestation, is much less prevalent.

(d) *Feeding of Heifers.*—Heifers between the age of weaning and breeding, are usually the most neglected animals on any farm.

Delayed puberty and breeding, due to poor nutrition, is the rule rather than the exception.

Since these heifers are the replacements and the future foundation of a herd, many instances of poor herd fertility can be traced to this period of neglect in the development of the animals.

3. *Genital Infections*

Since the great majority of farmers using A.I. in Natal and East Griqualand, do so because of the presence of one of the venereal diseases in their herds, this constitutes one of the major breeding problems.

Considering the method of transmission of these conditions, as well as general farming methods, for instance regular stock sales, the small number of self contained herds, poor fences, communal dipping tanks, etc., it is evident why this is the case. Vibriosis is especially rife. The following interesting and practical points about this condition may be mentioned. The symptoms vary tremendously, and apart from a possible variation in the pathogenicity of different strains, seem to be essentially influenced by the standard of herd management and nutrition, and the length of time which animals have been exposed to the infection.

The condition seems to be self-limiting to a certain extent, but cases of flare-ups, usually with a 2 to 3 year interval, when natural breeding is persisted with, are not uncommon. Often these new outbreaks seem to be predisposed by some adverse conditions entering the herd, for instance: a period of poor nutritional or climatic conditions or debilitating diseases.

A further aspect of this condition, which is very well borne out by the use of bulls on heifers only, is the following: Apparently, although heifers become infected at the time of service by an infected bull, they very often conceive and calve down quite normally. (If they do not conceive to the first service, a vaginitis and cervicitis may set in, which affect their breeding ability severely.) After calving, however, the infection seems to exert its effects. Apparently the infection lies dormant in the posterior genital tract, till the cervix opens for calving. Subsequently, a forward spread of the infection into the anterior genitalia takes place, often leading to complications such as cervicitis, metritis and even salpingitis and oöphoritis. An interesting observation has also been, that this process may take place after each calving, and be responsible for a progressive reduction in the fertility of an animal. Especially in herds where no treatment has been practised, one finds an abnormally large number of cows developing such lesions, although their past 2 or 3 calvings have been to A.I. Although these symptoms are usually associated with infectious epidydimitis and

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epivaginitis, no other proof or symptoms of this condition could be found in these herds, whereas vibriosis had definitely been diagnosed.

Evidence of transmission of this condition other than by mating, has been found in several severely infected herds, where a large number of virgin heifers showed symptoms of the infection.

Very good results have been obtained in combating this condition with the combination of A.I. and intensive herd treatment. An intra-uterine infusion with an appropriate antibiotic (for example Pen-Strep or Terramycin) immediately after calving or at the first heat period, or both is given. The animal is then inseminated during a subsequent heat period. With this method, a very good conception rate is obtained, and the progressive reduction in fertility previously mentioned, seems to be curbed.

4. *Functional Infertility*

(a) *Anoestrus.*—This is very prevalent in both cows and heifers during certain times of the year, especially when the nutrition is poor. Excellent results have been obtained with the supplementation of Vitamin A, to ensure a regular breeding cycle throughout the year. The practice of "steaming up" cows before calving, and heifers before breeding, usually ensures that the animals will not be suffering from this condition when they are due to be inseminated or served.

(b) *Delayed ovulation.*—The role played by delayed or anovulation in depressing the fertility of dairy cattle must not be under-estimated. Heifers and cows that have been allowed to have many heat periods without being bred, are prone to ovarian disfunction and especially delayed ovulation. It is recommended that heifers be bred when they have reached an adequate stage of development, rather than at a certain age. The fact that the fertility of cows deteriorates beyond plus/minus 75 days after calving, may also be ascribed to this condition. A very high incidence of delayed ovulation has been found in herds, or individual cows, where for some reason or other, the animals had had several heat periods without getting into calf. This is especially true of Jersey cows and heifers, and we are now following a policy of inseminating Jersey cows the second heat period after calving (six to nine weeks) in an effort to overcome this problem.

In an experiment which we are now conducting, the excellent results so far obtained with repeat inseminations during the same heat period, supports the view that delayed ovulation is responsible for lowering the breeding potential in many instances.

5. *Climate and season*

The overall conception rate of the Natal A.I. Co-Operative indicates a definite seasonal variation in the breeding potential of herds. The worst periods for conception are late summer and autumn, and spring to a certain extent. In late summer, many parts experience conditions of extreme heat and high humidity, which definitely lower the conception rate. Even relatively short periods of drought and heat, are also immediately reflected by a lowered conception rate. In autumn,

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natural grazing and pastures deteriorate rapidly, and usually farmers do not embark on their winter feeding programme until early winter, leaving a gap of very poor nutritional conditions. As soon as winter feeding is in full swing, the fertility is restored. The same condition arises when winter feed supplies are depleted, and spring rains delayed.

6. *Internal Parasites*

Although conditions of high rainfall and intensive use of pastures, under which most of the herds are kept, are ideal for internal parasites, very little is done about their control in cattle. A spectacular response in improved production and reproduction may be obtained, when this aspect is attended to.

In one particular herd, suffering from very poor fertility, the problem was eventually diagnosed as a complication of a severe lung worm infestation.

7. *Veterinary Control*

The lack of veterinary supervision over the health of most herds, contributes a great deal to breeding problems continuously arising and developing without check.

There are two aspects to this problem: *Firstly*, farmers must be convinced of the necessity of regular veterinary supervision and examination of their herds, instead of the accepted practice of calling in the veterinarian only for individual sick animals or pregnancy tests. *Secondly*, the basic approach of veterinarians should be one of supervision, advice and guidance on the general management, feeding, breeding and health, *of the herd as a whole.* Very often herds suffer from a lowered breeding potential due mainly to ignorance on the part of the owners, which it is our duty to rectify.

This problem is aggravated by the fact that in some of the most densely populated cattle breeding areas, the services of neither a private nor a state veterinarian are available.

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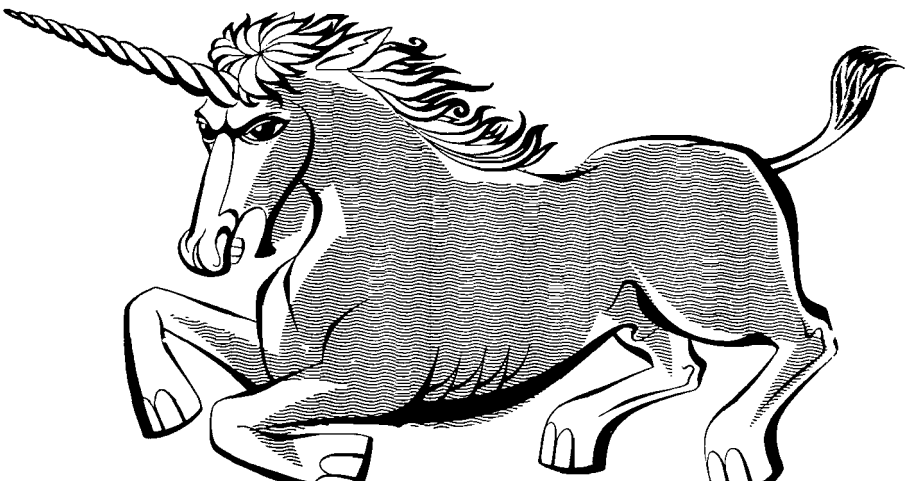
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T. H. WHITE *"The Book of Beasts"*

The "Book of Beasts" is a modern translation of the Bestiaries which were copied in Latin prose from the Greek originals about the 12th century A.D. The Bestiaries were intended as serious scientific works

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THE PHYSIOLOGY OF REPRODUCTION IN THE MARE WITH SPECIAL REFERENCE TO THE THOROUGHBRED

C. W. A. Belonje*

Thoroughbred breeding is an established industry in all civilised countries in the world and will no doubt remain as the solid core of equine breeding of the future. For racing purposes an age limit had to be drawn which in South Africa dates from the 1st August. This in turn contracts a breeding season into an artificial period from approximately 10th September to about the 15th December i.e. a period of 97 days.

The amount of work done on reproductive physiology is quite considerable and most confusing by its cross-references to the other domestic animals and man, until it becomes nearly impossible to obtain a clear overall picture of the process of reproductive function and disfunction of this breed. For this purpose it is necessary to refer to the origin of the horse: how long ago it was a small marsh-living animal, about the size of a dog. Its natural evolution consisted of its taking to drier ground and of the development of speed by increasing the length of leg in proportion to the length of body.[1] The animal became domesticated and was used for work, war and play. The mares still remained useful for this purpose even though she bred and produced a foal only occasionally or not at all. As a result there has not been the tendency to improve fertility by selective breeding and variations have been allowed to increase through this lack of selection. The result is a relatively low fertility rate as compared with the other classes of livestock, a fact reported by several research workers in the world.[2], [3], [4], [5] Selective breeding was performed, but only to create an animal for speed and endurance and to this day we are left with highly specialized animals but in which the reproductive mechanism still follows the primitive pattern of the past where in some breeds additional variations have been added by more attention to selection. This selection to greater fecundity is more noticeable in the heavy breeds and there we usually find a more stable pattern of recurring oestrum and dioestrum. This difference between the heavy or draft horse and the light or riding horse has been emphasized in the literature and has been the subject of some attention. In the Thoroughbred an animal was specially selected for speed which clearly represents an advance in neuro-muscular development and perfection. This development is such that it forms a decided additional factor for consideration in the reproductive function of this breed.

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The Breeding Season

It is generally accepted that the breeding season occurs during the spring and summer months i.e. during the period of ever increasing hours of daylight.[20] When mares are transported from the Northern to the Southern hemisphere they change over the times of the breeding seasons to fit the seasons in their new surroundings. Near the Poles the breeding season becomes constricted so that all the foals are dropped within a short period of time, but as one passes to the Tropics, however, there tend to be two breeding seasons in the year, for unlike the temperate zone, the sun here is overhead twice during the year instead of only once[1]. Even in this transportation of animals from one region to another marked differences are shown indicating the decided variations that occur in the acclimatisation of horses of the different imported breeds[7]. By exposing barren anoestral mares to the effects of artificial lighting during the winter in England it was possible to bring them into season[8].

Oestrum and the Interoestral Period

Oestrum in the barren mare is detected by the presence of the stallion and it is extremely rare to find mares giving oestral indications by themselves. On Thoroughbred stud farms after the breeding season is over and the mares are no longer subjected to regular teasing, one does not encounter any mares on heat.. This is a basic fact. Further it is possible to force a mare into a heat period. An old method was to place the stallion in a loose box and put the mare in the adjoining box so that both could see each other over or through the partition and for the stud groom to insert his arm into the vagina and gently massage the cervix. This cervical sensitivity has been noted recently by two research workers[9], [10].

The more modern method is to twitch and if necessary hobble the mare in a padded crush and to allow the teaser stallion to manhandle her and even mount her but not serve her. In both methods oestrum will appear in the majority of cases within a week, depending on the time of the season, but the oestrum so provoked may be completely disassociated from any ovarian development. This external manifestation of heat is now called 'Psychological Heat' which is correct as it is an outcome of a mental stimulus, whereas the ovariogenital tract development is called 'Physiological Heat'. This distinction has been observed and commented upon by many workers. It makes the critical analysis of much work very difficult, because the psychological heat may be artifically induced by excessive teasing and is essentially an artefact.

According to the work of Satoh & Hoshi the semi-wild native mare in the Saislin Island in Korea is monoestrus, its appearance occuring in a definite breeding season extending from May to July. Kupfer[12] working on mares kept under ordinary veld conditions in South Africa concludes from his observations that the appearance of ovulation and oestrus in donkeys and horses is seasonal, occurring only at certain times of the year. For the rest of the year ovulation and symptoms of heat are entirely absent. He states that this season

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will vary from year to year in as much as both the beginning and the end of the active season are subjected to influences determining the condition of the animal. This breeding season extends from the end of October to the end of March, but he failed to establish any definite cyclic periodicity in the recurrence of the heat periods. Some cycles were prolonged, other extremely short (1–2 days) and sometimes such a short cycle was followed by a few day's interval and then heat reappeared. Young mares usually ovulate only once, while with age there occured two or three ovulations. Ovulation may occur without heat and heat without ovulation, the latter being more frequent. In an interrupted heat period ovulation will occur during the second part.

Quinlan and others[13] carried out observations on mares stabled at night at Onderstepoort. The mares were never let out to graze but received uniform rations throughout the year with the exception that green feed was given during the summer months but not during the winter. These mares were tested for oestrus daily by a teaser stallion and it was observed that oestrus occurred irregularly throughout the year but that the months of April, May, June and July were found to be barren in so far as none of the mares that were mated then became pregnant. With one exception all cases of anoestrus commenced during the first 7 months of the year. There was an increase in the mean duration of the oestrous cycle in January, indicating the end of the optimum breeding season. Oestrus tended to be longer in the early part of the breeding season.

Hammond[1] states that in a cold dry spring and especially in very young or very old mares in poor condition the heats may last a long time (10–15 days or more) but as the breeding season advances, however, the heats tend to get shorter so that by June-July they last only 5 days on the average. Day[14] mentions that while some mares continue to come into oestrus throughout the year, others go into an anoestrus condition in winter and do not come into oestrus from November to February.

Although the heat periods and service dates are recorded in all well regulated Thoroughbred breeding establishments it is extraordinary to find very little data published in any scientific publication on this aspect. The data on other breeds of horses are numerous and have been subjected to various statistical analyses and indicate an extremely wide variation. Later work has indicated further differences between draft and light weight breeds of horses[6], [29].

The desire to subject these phenomona to the rule of averages came from observations on the human, and on laboratory and other domestic animals where reproductive function has evolved into a more or less stable and rhythmic pattern. No such defined periodicity occurs in the mare, more often than not the only constant feature of the oestrous cycle is its irregularity, and we feel that we must agree with Lagerlof[15] that the variations in the oestrous behaviour of the mare represents a transition from the monoestrous to the polyoestrous confined within a definite season, the outer limits of which vary according to environmental factors.

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The Mature Barren Mare

Taking as a basis for discussion the mature barren Thoroughbred we find that she is put out into the veld during the winter months. On many Karroo farms a specially spared camp or vlei is used for wintering them out, in other cases a small amount of supplementary feed, usually lucerne hay, is given. The mare grows a thick winter coat and usually becomes lean. On rectal palpation the ovaries are small, firm, and non-functioning: the typical ovaries of the deep anoestral stage[17].

With the advent of spring and the increase in the length and intensity of daylight the sexual development commences. The anterior pituitary is no doubt stimulated by several factors, chief of which are

(1) the stimulus of light on the retina transmitted to the hypophysis

(2) the oestrogenic content of the actively growing vegetation and

(3) the presence (i.e. sight, hearing, smell) of the stallion.

It is possible that many more factors are involved in this process which primarily is one of ever increasing body metabolism and that the changes that do occur cannot be ascribed to any single factor alone.

The early heats are frequently prolonged[16], [13], the mare has not shed its coat and when served does not conceive. The reason for this is that ovarian development has either not kept pace with the changes occurring in the genital tract or the tract has lagged behind ovarian growth. In the first instance we would have an anovular heat, in the second, insemination by the stallion would most probably be intravaginally and hence lost, but even should it occur into the uterus, the destruction of the spermatozoa would be so rapid in the adverse environment that fertilization of the ovum would not take place.

The advice that the heat period of the mare should be checked by examination of the cervix by means of a vaginal speculum[18] is only to determine the state of development of the genital tract. When there is oestrus, but a dry, pale, constricted cervix is encountered, service is invariably barren, even if ovulation should occur.

It was noted by Caslick[18] that the Thoroughbred mare required two or three oestral periods before conceiving. It seems as if early in the season the anterior pituitary, after adequate stimulation, commences to pour out ever increasing quantities of gonadotropic hormones into the bloodstream which in turn stimulate the ovaries. These begin to soften and swell through infiltration of fluid into the interstitial tissue[8], [19] to enlarge greatly in size through swelling of the secondary and tertiary follicles. The increasing output of anterior pitutiary hormones stimulate the formation of oestrogen; it is clear that in the mare this hormone is not predominantly formed in the ovary, if at all.[50]

The stallion excretes in his urine about 1,700,000 M.U. of oestrogenic sex hormone per diem, equivalent to 62 gm. of hormone in a year, a much larger amount than does the pregnant mare. Mass excretion of oestrogenic hormone in the urine of the male animal is considered a peculiarity of equines[20].

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The pregnant mare again[21] excretes small quantities of oestrogen in the urine during early pregnancy but this amount increases rapidly after the disappearance of circulating gonadotropin. Maximum excretion occurs between the 7th and 8th month after which the output gradually declines. During this phase the ovaries are without visible follicles, contain no trace of a corpus luteum, are completely contracted and it is highly probable that they play no part in the maintenance of pregnancy or oestrogen secretion.

Seaborn[22] has noted that during oestrus the adrenals may in some cases double their weight and later endocrinological work indicates that the cortex of this organ may be intimately associated with oestrogen production[23].

The function of oestrogen, apart from any direct action it may possess on the germinal epithelium of the ovary itself and on secondary sex characteristics, is to promote the growth and development of the genital tract, increasing the blood supply, stimulating uterine contractions[24] and increasing motility of the Fallopian tubes[25]. In addition it appears to be the most important hormone factor in the control of mating. The doses of oestrogens necessary to induce mating are however much larger than those which will induce morphological changes in the secondary sex organs.[23]

The fact that 2 to 3 heat periods are normally required before a Thoroughbred mare will conceive[18] may be due to subjection of the genital tract to prolonged oestrogen stimulation before it will reach that stage of perfection when successful fertilisation, implantation and gestation can be carried through. Although ovarian activity is usually marked during these early heats, the ovaries becoming larger, softer and containing many follicles, it is very rare to find any rupturing, until apparently both activities have reached the same degree of optimum development. Such follicles usually shrink and either degenerate or become atretic[25].

This position is clearly recognised in the filly when she first comes into heat at about 10–11 months of age. The ovaries are active but the tract is still undeveloped. True puberty, i.e. the point where sexual maturity is reached and these two processes synchronise to make reproduction possible, may be very much delayed and postponed up to the 5th year of life. This "immaturity" in maiden mares is responsible for more cases of barrenness than is generally recognised[17].

Under our Karroo conditions the barren mare reaches the peak of reproductive performance during late October and November. The rains usually fall late September or during October-November and the natural grasses are then actively growing. It is interesting to note that actively growing grass has been found to contain appreciable amounts of oestrogen, but that lucerne does not contain any at all[26]. The latter is extensively grown in this area as a hay and grazing crop. Later in the season, the heat periods shorten to about 4–5 days and the interoestral period to about 16 days. By this time ovulation normally occurs during the heat period.

Symptoms of heat is shown in the presence of the stallion[50]. If the mare is free, she will approach him, allow him to muzzle her,

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raises tail and clitoris and ejects mucus and urine, and gives every indication of being receptive. The vulva is enlarged, relaxed, soft and swollen, the vestibule pink to red, covered by a thin glistening mucus. Vaginal examination by means of speculum is easy, the thin mucus facilitating insertion of the instrument. The cervix is swollen, congested, relaxed, lying usually on the floor of the vagina and will easily admit the passage of 2–5 fingers[16].

Service

Insemination by the stallion at this stage is invariably intra-uterine[16], the semen being ejaculated directly into the uterus assisted by a strong suction-like action of the womb during coitus[27]. The semen is ejaculated mainly in two portions, the first consisting approximately of 20–70 ml. sperm fluid containing the spermatozoa mixed with prostatic liquid, followed by about 50–200 ml. of a mixture from the accessory glands[28]. The latter provides bulk and forces the sperm fluid along the horns to the *papillae uterinae* without causing thorough mixing. Sperm motility plays little or no role in this approximation to the uterine opening of the Fallopian tube[29].

As the genital tract of the mare is still under influence of its own oestrogens the defensive mechanism is highly organised[24]. The sperms in the uterus are stimulated to greater activity, then die and start agglutinating. Leucocytes enter the uterus and attack the living and dead spermatozoa and phagocyte them. Slight reddening and swelling with desquamation of the upper epithelium, resembling a light inflammation, takes place within 8 hours of insemination. Antigens are produced against the sperms (sperma-toxins), which, if in high concentration, kill off the sperms[29]. This is the normal defensive reaction to prevent disease through the introduction of dirt, smegma and organisms by the intra-uterine insemination of the stallion and constitutes a reason why repeated matings at short intervals during the same heat period are to be avoided[29].

The *ostium uterinum tubae* is usually tightly closed but opens in a similar fashion to the cervix during heat and remains open until the fertilised ovum reaches the womb[30]. The spermatozoa swim up the Fallopian tube against the mucus stream and ciliary motility to reach the egg and cause fertilisation.

This positive rheotaxis, i.e. movement against the mucus stream is characteristic of all mammalian spermatozoa. Yamane and Ito[51] estimated equine sperm motility in standing fluid to be approximately 5.2 m.m. per minute. In moving fluid this speed reaches its maximum of 6.4 m.m per minute where there is a weak stream flowing at about 1.2 m.m. per minute.

The longevity of the sperm in the mare's genital tract appears considerably longer than in the cow and sheep and intervals of 48 hrs[1] to 138 hrs[52], [39] are given. One reason for this may be that the testicles of the stallion are carried very close to the body, so that sperm production cannot be at a very much lower level than the body temperature[31] and secondly that the *ostium uterinum tubae* may function as a reservoir in a similar fashion as has been described for sheep and cattle[29].

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Ovum

The spontaneous rupture of the Graafian follicle takes place about 24–48 hours before cessation of heat[14] and the ovum enclosed in a clot of *liquor folliculi* passes into the funnel-shaped opening, which with its fimbricated end approximates the ovulation groove during heat and ovulation.[29] The egg moves down the Fallopian tube assisted by ciliary movement, contractions of the tube as well as outside movements of the bowel, respiration and the body. During its passage it gradually loses its *zona radiata*. Its viability is a matter of conjecture but in mares appears to be longer than in cows and sheep, and it is possible that the ovum continues to maturate in its downward passage.

On grounds of theoretical considerations, service at or just before ovulation is regarded by many[32], [33], [34], [53] to be optimum but evidence is accumulating that the percentage fertility is considerably increased if insemination is performed up to 12 hours after ovulation. After 24 hours however the chance of fertilisation becomes very remote. Under natural mating conditions, it is likely that the mare will only accept the stallion at the height of the oestral period which normally coincides with or just after ovulation. Normally heat continues from 1–2 days after ovulation[14], [25].

The transport of the ovum through the Fallopian tube becomes delayed and this coincides with the change over from the oestrogen to the luteal phase of the ovary. It is estimated that it takes the ovum from 95 hours[35], [36] to about 6 days[29], [37] to reach the uterus and it is claimed that the defensive mechanism can be supported by vaginal and uterine irrigation after mating while the fertilised egg is still in the Fallopian tube[29].

Clinical Diagnosis

The experienced equine specialist is fortunate in that the changes in the genitalia can be followed with a great deal of accuracy. The appearance of the vestibule, anterior vagina and cervix give him an excellent indication of the development of the tract. Rectally the ovarian changes can be followed closely from the time that the infiltration of the stroma and puffing-up of the secondary and tertiary follicles occur and the organ changes from a firmly contracted to an elastic, vital, functioning gland. The follicles, at first indistinct and firm, take shape by protrusion, until one or more can be distinctly felt. The size of the follicles is subject to great variation but at the commencement of heat one usually becomes more prominent and about 1–3.5 cm. in size[38]. In the prolonged heats early in the season, this follicle usually recedes either by degeneration or atresia, but when all factors are operating in unison it takes about 3–4 days to mature, reaches suddenly or gradually about 3.5–6.5 cm. in diameter before rupturing[38]. Impending ovulation is usually indicated by a sudden drop in intrafollicular pressure and fluctuation of its content may be noticed.[14]

Ovulation is recognised by the sudden collapse of the follicle and the formation of a very conspicuous crater. Within 8 hours [40] this becomes partly occupied by a blood clot which in turn is invaded by

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bloodvessels and luteinized. The size of the corpus luteum is usually below that of the Graafian follicle[25] but sometimes approximates it[41]. Clinically it is palpable for about three days after which it sinks into the ovarian tissue. It feels soft, elastic[40], there are no fluctuations, is not compressible and feels like the crunching of a snowball[29]. The mare at this stage gives indications of a low grade painfulness when the ovary is handled.

From this course of events all kinds of aberrations can be expected and are actually encountered within a single mating season on a Thoroughbred stud. One feels that they cannot be regarded as pathological, but merely as evidence of a rather primitive reproductive system struggling to achieve the synchronisation of all responsible organs to reach that unity of effect conducive to successful reproduction, something which we take so much for granted in our other domestic animals. It is rather difficult to visualise a breeding season on a Thoroughbred stud farm without the full grown maiden mare with normal heats but infantile genitalia; the mare with normal heat and ovulation but no implantation; the mare with the development of the tract during heat but no ovulation; and even the "shy" mare showing no external sexual desire but having achieved unity between ovarian development and the genital tract. All these are normal phenomena, noticeable particularly in the early part of the breeding season.

Within 6 hours of ovulation the oestrogen level disappears[29] and the rapidly developing corpus luteum takes over and secretes progesterone. This inhibits further pituitary action, and sexual desire normally disappears within 1–3 days after its formation. The functional activity of the temporary corpus luteum is short and estimated at 10–-12 days[42] which is then followed by a short dioestrous period of 5–6 days during which there is an interplay between the progesterone and anterior pituitary hormones. Tertiary follicles develop slowly during this time until oestrus begins again, when the pituitary is completely released from ovarian control. In general the length of the interoestral period tends to be more uniform than the length of oestrus[43]. The corpus luteum is not completely autonomous, but is definitely under the influence of the inner secretions of the uterine mucosa. When fertilisation has taken place the rapidly growing egg stimulates the endometrium which in turn prolongs the life of the corpus luteum of pregnancy. This body in the horse, however, has only a span of 30–35 days[44], its function being taken over by other corpora lutea and after the 5th month by the co-operation of the foetal placenta and the endometrium.

From the literature[45] it appears that the implantation of the mare occurs between the 40–70th day and this period coincides with the appearance of gonadotropic hormone in the bloodstream. Up to about the 6th week the foetus is attached to the uterus through the "yolk-sac" only—that is a primitive mode of attachment especially characteristic of the lowest mammals, but between this time and the 9th week this is replaced by the more permanent allantois, which is one of the foetal membranes which does not fully develop until this stage[2]. The so-called serum gonadotropin (P.M.S.) occurs from

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the 40th to 120th day of pregnancy and is at its highest potency at about the 60–70th day[21].

It is elaborated in special structures called "endometrial cups" in the fertile horn of the uterus[47] and the sudden fall in the potency of this hormone in a case of abortion, and the maintenance of a high level in another, in which resorption had occurred, suggests that the conceptus may be necessary for the preservation of the functional activity of these glandular structures in the uterus[37].

The usual pattern of ovarian behaviour during early pregnancy as we know it in cattle, sheep, etc., is disturbed in the mare by the presence of this P.M.S. but it does not cross the placental barrier to affect the gonadal development of the foetus.

In the mare the ovarian changes of pregnancy may be divided into 4 periods[42].

(1) From 1–40 days after conception follicles are present in the ovary but there is no evidence of the serum gonadotropic hormone in the blood.

(2) From 40–150 days there is progressive activity in the ovary manifested by continuous formation of follicles and marked luteinisation. The presence of serum gonadotropic hormone may be necessary to maintain the functional activity of the corpus luteum of pregnancy. The additional corpus luteum produced at this time may be a factor of safety in ensuring implantation. Ovulation quite frequently occurs prior to luteinisation.[9, 37, 42].

(3) is the period of regression of the corpus luteum. Large follicles are absent. This period begins at the end of the 5th month and may end towards the end of the 7th month. The corpora lutea shrink and this coincides with the disappearance of the serum gonadotropin.

(4) The ovary at the end of pregnancy is entirely lacking in corpora lutea . A mare ovariotomised at 200 days after conception maintained her pregnancy[42]. This is the period during which oestrin appears in ever increasing quantities[42] reaching its maximum excretion between the 7th and 8th month. Thereafter the output declines gradually during the remaining three months. During this period the ovaries become completely inactive, are without visible follicles and contain no corpora lutea. Histologically the cortex is very fibrous and contains only a few primordial follicles.

The foetal gonads, however, show remarkable growth during this oestrogenic phase, caused by a prodigious increase in the interstitial cells. Regression subsequently sets in at about the 8th month due to the disintegration of the interstitial cell mass and this coincides with the fall of the oestrogen level in the urine.

Clinical Observations

A. A very noticeable ability to contract occurs in the uterus of the pregnant mare from about the 20th until the 40th day. One has to

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accept that during this period the uterine musculature is so influenced and sensitized by the hormones (oestrogens) that on the stimulus of rectal palpation a powerful contraction occurs which lasts for a certain time. This action is no doubt due to protect the unattached egg and to retain it in situ. In a relaxed womb there is danger that external influences such as galloping, manual manipulation, may move the small egg to and fro and unsettle it[29].

The uterus on handling becomes erected, feels like sponge rubber with no palpable lumen. This change in consistency is so different from the non-pregnant horn that it cannot pass detection. The conceptus is roughly:—

(a) at 18–25 days the size of a pigeon's egg.

(b) at 25–35 days the size of a golf ball. Several large, soft, flabby folicles may be present in the ovary during these periods. The vestibulum and anterior vagina are usually pale, the cervix tightly contracted and a small amount of sticky glue-like mucus is found in the canal and anterior vagina.

B. After about 5–6 weeks the sensitivity and hence the contractibility of the uterus decreases and diagnosis can be made on the size and position of the foetus. From 35–50 days it is from a fowl's egg to a goose egg size. The swelling is unilaterally pronounced, is carried slightly downwards, becomes soft and fluctuating and thin-walled. The cervix and anterior vagina are pale, the cervix contracted and closed and the sticky glue-like mucus more pronounced. To feel the double layer as in cattle is contra-indicated as the foetus lies loose and the danger of abortion exists. In 80–90% of cases a definite diagnosis can be made during this period. In other cases the foetus has already reached the body of the uterus and creates in this manner diagnostic difficulties especially in old barren mares where there might be bilateral or more frequently unilateral dilatation of the womb. The vaginal smear can be employed to confirm the diagnosis and, after the 45th day, the blood test.

C. From 2–3 months the foetus enlarges from about an orange to grape-fruit size, is thin-walled, lies next to the bladder and covers the anterior edge of the pelvic inlet.

D. From 3–4 months it is the size of a football, the suspensory ligaments are taut and hang straight down. The uterus completely covers the anterior edge.

E. From 4–5 months the uterus sinks to the ventral abdominal wall and cannot always be reached by hand. The signs are:—

(a) That the pregnant horn cannot be found.

(b) The pubis is covered by the cervix.

(c) The utero-ovarian ligament is tense, taut and perpendicular.

(d) The ovaries become small, are without follicles, and are drawn downwards.

(e) The *arteria uterina media* enlarges and gives a characteristic thrill.

F. From about the 7th month foetal parts can be detected.

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Parturition

The length of gestation in the Thoroughbred mare in South Africa is given most commonly as 330–340 days with a variation from 300 to 349 days[48]. About two months before the mare is due to foal the udder swells behind and the skin becomes soft. The factors influencing parturition are unknown, but it may be mentioned that the maximum excretion of oestrogens seems to occur some considerable time before parturition and there is in this species no increase in the excretion of free oestrogens at parturition (blood oestrogen content unknown).

Impending parturition is usually signalled by the mare forming wax-like or transparent scales on the tips of the teats within about 1–3 days before foaling. The sacro-iliac ligaments are relaxed and the vulva is soft and swollen. Birth is invariably accomplished lying down, although in a few exceptional cases the mare may start foaling standing up. The act is of short duration and the placenta is usually expelled within about ½ hour.

Foal Heat

This is the interval between parturition and the first oestrus following foaling, including the day of foaling, and extends to the onset of oestrus.

Williams[49] found that the mare is commonly in heat at not later than the 8th or 9th day after foaling and states most appropriately:—

"According to tradition this is definitely the best time to breed her, and the tradition is so ancient that it is well-nigh sacrosanct."

Caslick[18] working on Thoroughbreds in Kentucky, U.S.A., mated 301 mares on the 9th day postpartum—of these 111 (37%) conceived. Two hundred and thirty-two mares were mated on the 10th day and 84 (36%) conceived, twenty mares were mated between the 11th–21st day after foaling and 8 (40%) conceived.

Mahaffy[50] found that in a Thoroughbred stud of 27 mares in Australia only 59% came in heat on the 9th day during the 1948 breeding season of which 7.4% failed to ovulate. During the 1949 breeding season 34% of the mares came in heat on the 9th day but, 20% did not ovulate. Foal heat occurred at intervals ranging from 7–50 days with oestrus of 1–12 days' duration; where ovulation occurred it usually took place towards the termination of the foal-heat.

Andrew and McKenzie[6] record that only 54% of the mares were actually on heat on the 9th day and of these 12.5% did not ovulate. They also observed that at foal-heat involution may be incomplete and that at that time in 8 out of 9 mares studied the surface of the uterine mucosa was not completely covered by epithelium, the subepithelial area of degenerating glandular epithelium being highly disorganised and containing large numbers of leucocytes. They conclude by considering that under such circumstances the uterus is not in a perfectly favourable state for sperm transport and viability.

Mahaffy[50] states that with appropriate selection of mares following ovarian palpation and examination of the genital tract, some 70% of those served on foal-heat will conceive. He concludes that foal-heat should be made use of in a Thoroughbred stud, where no time

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can be lost if high final percentage fertility is to be achieved during the short season.

Clinical Observation

Rectally the uterus on the 9th day is still large, conical, firm and very heavy. Failure to involute is recognised by the uterine horns remaining elongated; the walls are soft, and can be squeezed against each other, the lumen being clearly detectable. There is little or no tendency for the organ to contract into the typical conical shape. The ovaries are as a rule large, soft, containing several follicles of which one or more may be prominent and fluctuating.

Summary

The purpose of this paper is to give a general survey of the reproductive function of an animal, which although highly specialised in its performance, has still retained, to a very great extent, all the characteristics of a primitive pattern of reproduction.

In the cow and sheep a highly specialised reproductive system functions with almost clockwork regularity. Where its pattern is disturbed the underlying cause is invariably pathological. In the mare this specialisation, this intercorrelation between the component parts, is frequently absent and each of its component parts may act independently resulting in the disharmony so frequently observed. These phenomena are not abnormalities but merely expressions of a reproductive system reflecting the genetical, nutritional and climatic circumstance of the mare, and should be recognised as such.

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CONTROL OF BRUCELLOSIS IN SHEEP AND GOATS BY MEANS OF VACCINATION

G. C. van Drimmelen.*

Intensified methods of husbandry and increasing knowledge of small stock diseases have revealed extensive Brucella infection in most sheep breeding countries such as Australia[1], New Zealand[2], the U.S.S.R.[3], South Africa[4], [5], [6], Tunis[7], and the Argentine[8].

Brucellosis of sheep and goats, well known in Mediterranean countries, Greece, Italy, Malta, Spain and France, also spread to Germany as the result of post-war movements of stock from France[9]. It is being eradicated by slaughter of infected flocks in some countries and Malta itself is virtually free of Brucella infection today.

The test and slaughter policy is not successful in the absence of facilities for isolation. In these circumstances immunity and resistance to infection have to be relied upon. Natural immunity of variable degree has been found present in endemic areas and in nomadic flocks.

Vaccination with Strain 19 *Brucella abortus* was attempted but the results were of no value because the challenge was too great. Natural infection in sheep is not as violent as in cattle but they do not develop a higher resistance to infection during immunization. The difference between natural levels of immunity and acquired levels of immunity is more difficult to determine.

Artificial immunization of small stock with Strain 19 has been extensively practised in Russia[3] with results of great value to the farming industry. Following the lead given by Lisbonne, Roman and Renoux[10] who immunized sheep with simultaneous inoculation of Strain 19 vaccine and killed culture of *Br. melitensis*, Buddle and his co-workers in New Zealand partially protected sheep with such a vaccine[11].

Rams and ewes vaccinated with live Strain 19 vaccine and simultaneously inoculated with killed culture of the local sheep infection, were protected from infection responsible for genital lesions in rams and a low rate of abortion in ewes. This type of Brucella infection spreads rapidly amongst rams, and is relatively mild in its effect on ewes and guinea pigs.

Elberg and his associates in California evolved a variant of *Br. melitensis* which did not produce permanent carriers and yet caused an infection of much longer duration than Strain 19. Its virulence may be said to be higher than Strain 19, but not high enough to produce the clinical disease or the carrier state. When tested as vaccine in goats it imparted statistically highly significant increased resistance against artificial exposure to graded doses of virulent infection[12].

Renoux and his co-workers in North Africa have demonstrated good immunity in sheep, goats, guinea pigs and mice after vaccination

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with formol-killed *Br. melitensis*, suspended in specially prepared adjuvants[13].

By "all-or-nothing" tests a Brucella vaccine can appear worthless with a large test dose and only insignificantly protective with a small test dose. Yet the same vaccine may be capable of producing a tenfold or even a hundredfold increase in the 50% infective dose: "ID_{50}". The newer techniques of measuring the degree of resistance to infection in a host-parasite relationship system of the kind found in brucellosis have, however, led to a better understanding of the problem.

The percentage of animals infected by exposure to increasing doses of virulent organisms, as other biological phenomena of similar nature, fits an asymptotic curve. The 50% infective dose (ID_{50}) can be calculated by the method of Reed and Muench[14], or by plotting a straight line on log probability paper[15], or by means of the more accurate method of probit analysis. The immune response to vaccination can then be measured in terms of increase in ID_{50}.

Thus Renoux[16] reported a 140– to 193– fold increase of the ID_{50} from vaccination with formol-killed *Br. melitensis* suspension containing 2.0×10^{11} cells plus adjuvants "Aracel A" and "Mayoline 2214' in experiments which showed an ID_{50} of 2.0 to 6.0×10^4 virulent organisms in control goats and an ID_{50} of 4.0 to 5.5×10^7 in goats vaccinated with live Elberg Rev. 1. *Br. melitensis* (i.e. an 89– to 110–fold increased ID_{50}).

Jones, Thomson and Alton[17] in similar experiments at Weybridge reported a 62-fold increase of the ID_{50} after vaccination with live Elberg Rev.1. *Br. melitensis* when the ID_{50} in control goats was 4×10^5 virulent organisms and the ID_{50} in goats vaccinated with 10^{11} heat killed *Br. melitensis* in liquid paraffin and Falba was 2×10^7 (i.e. 50–fold increase of ID_{50}).

Elberg[18] demonstrated in Spain an increase in the ID_{50} of "Rev.1" immunized goats, which varied from 340– to 12,000–fold by the graphical method and which by the more accurate method of probit analysis showed a 2,000–fold increase [from 2.5 to 5×10^4 cells to 6.14×10^8 (with 95% fiducial limits of 1.03×10^8 and 2.9×10^{22})].

TABLE 1

THE ID_{50} OF VACCINATED AND NON-VACCINATED GOATS

Vaccine	Results		
	Renoux North Africa	Jones Thomson & Alton England	Elberg Spain
Nil	6.0×10^4 (53H38)	4.0×10^5 (53H38)	3×10^4 (6015)
Killed Brucella orgs..... (53H38)	9.0×10^7 (i.e. 190×)	2×10^7 (i.e. 50×)	———
Live Brucella orgs. ..... Elberg Rev. 1	5.5×10^7 (i.e. 110×)	2.5×10^7 (i.e. 60×)	6.14×10^8 (i.e. 2000×)

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Unfortunately there is no comparable evidence of the effect of Strain 19 vaccine in similar experiments, but Renoux[19] found the degree of immunity derived from killed culture better than from S19 in guinea pigs.

An aspect that has been forcibly brought forward by the Russian work, which includes the vaccination of some 35 million sheep and more than 7 million human beings with live Strain 19 *Br. abortus* vaccine, is, that immunologically all types or so-called "species" of true Brucella organisms are closely related. The immunity against infection by *Br. suis* or *Br. melitensis* derived from Strain 19 vaccination is as effective as that against a strain of *Br. abortus* of equal virulence.

The immunity in brucellosis, being mainly cellular, is, surprisingly, not very specific and some degree of immunity to Brucella infection can even be produced by B.C.G. vaccination.[20] Serum of immune animals, however, also plays a rôle in enhancing the resistance of the cells of immunized animals to the necrotizing action of virulent Brucella organisms that have invaded the "immune" phagocytic cells.[12]

The best immunity to natural Brucella infection appears to result from a massive stimulus of long duration brought about by a vigorous invasive infection of a virulence just insufficient to produce the chronic carrier state.[21]

The purpose of the present report is to make available existing data on the immunity to be expected in Merino sheep in South Africa against the types of Brucella infection encountered in this country.

In the western districts of the Union and in S.W.A., outbreaks of abortion in flocks of sheep and goats have been observed frequently. An abortion storm is usually self-limiting, being followed by a fairly high flock immunity. Nevertheless, veterinarians and stock owners have observed favourable results following Strain 19 vaccination in small stock.[5] A preliminary test with Strain 5711, a variant of *Br. melitensis var. karakul*, showed that, when used as a vaccine, it possessed suitable safety and conferred immunity in a few ewes.

In New Zealand Strain 19 vaccine and killed, adjuvant vaccine, inoculated simultaneously at weaning into ram lambs, or at one year old into ewe lambs, protected against artificial infection with cultures of the local Brucella strains causing abortion, stillborn lambs and genital infection of rams. Rams mated to a flock of 26 ewes all of which had grossly diseased foetal membranes at their first lambing following experimental infection, failed to develop active brucellosis. Yet rams even as young as eight weeks of age are susceptible to experimental infection and transmission occurs readily when rams run together, separated from the ewes. A high percentage of rams secrete infected semen for periods in excess of four years.[22]

The results of vaccination experiments in New Zealand are summarised in Tables 2 and 3.

In Australia[23] transmission of the ram infection to ewes is less evident. British breeds, especially Dorset Horns, seem to be more susceptible than Merinos. Stud breeders have to eliminate all infection

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TABLE 2
EWES EXPOSED TO INFECTED RAMS

Vaccination	No. infected	% lambs
1957 controls not vaccinated	4 / 22	—
1957 S19 + adjuvant vaccine	1 / 22	—
1958 controls not vaccinated	7 / 50	56
1958 only adjuvant vaccine	2 / 50	76
1958 S19 + adjuvant vaccine	1 / 50	80

TABLE 3
RAMS EXPOSED TO INFECTION

Vaccination	Exposure	Infected	%
1957 controls	i.v.	10 of 11	91
1957 S19 only	i.v.	9 of 12	75
1957 only adjuvant	i.v.	4 of 12	38
1957 S19 adjuvant	i.v.	2 of 12	17
1958 controls	field	76 of 176	43
1958 only adjuvant	field	21 of 131	16
1958 S19 adjuvant	field	8 of 146	6

because they wish to sell healthy rams. The complement fixation test has been applied to clean up many studs. No elimination of infection in wool growers' flocks is carried out. Provided 3% rams are put to the ewes, including at least one young ram in three, the lambing results are satisfactory. Young rams are not permitted contact with older rams at all before mating.[23]

MATERIALS AND METHODS

Merino ewes one to two years old and negative to all tests were kept in open camps where they were tested, vaccinated or left as controls, then served and exposed by depositing suspensions of virulent culture simultaneously in the conjunctival sac and in the vagina. Regular serological tests were carried out until after lambing.

Merino ram lambs, three to four months old, negative to all tests, and born at a station free from brucellosis, were vaccinated or left as controls on the farm of origin. They were brought to the laboratory at the age of six to eight months and exposed by depositing a few drops of infected semen on the glans penis on one or two occasions during one year, while repeated examinations of semen and clinical examinations of the genital organs were carried out.

The sites selected for inoculation were the ear or the ribs behind the shoulder.

Serological tests were continued at intervals during the period of observation.

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Tests for complete agglutinating antibodies were done in 0.85% and in 5.0% saline. Incomplete antibodies were investigated by means of blocking and antiglobulin tests. Complement fixation tests were also carried out.

The products of abortion of ewes and the semen of rams were examined microscopically, culturally and biologically.

The vaccines used were:—

1. live Strain 19, *Br. abortus* Batches 2884, 2940, 3077.
2. live Strain 5711 *Br. melitensis* Batch 3.
3. live Strain E.Rev.1. *Br. melitensis.* Elberg vaccine.
4. killed Strains 5716 and 588.

About 12,000 doses of Elberg Rev.1. vaccine have been used in field tests which are still in progress.

RESULTS

(1) Effects of vaccination.

(*a*) Temperature reaction of the animals under close supervision and vaccinated at Onderstepoort showed an immediate rise in temperature in some cases. Others show rises later and sometimes repeatedly. (See Table 4.)

(*b*) Swelling at the site of inoculation was often evident but serious lesions were recorded only with killed vaccine.

(*c*) Clinical symptoms due to the vaccinal reaction did not affect feeding or the general condition.

(*d*) Infertility due to vaccination was not found.

(*e*) Titres found after vaccination showed great variation.

TABLE 4

EFFECTS OF BRUCELLA VACCINATION IN SHEEP

Vaccine	Sex	Number	Swelling	Temperature	Titre
5711	Ewes	15	+	—	2560
Rev. 1	Ewes	10	+	105	1280
S.19	Ewes	6	++	106	80
Killed	Ewes	6	++++	105	1280
Rev. 1	Rams	25	+	108	320
Rev. 1 & S19 & K	Rams	6	++++*	—	80
S19	Rams	13	++	—	20
S19 & K	Rams	5	++++	—	80
K	Rams	14	++++	—	2560

* Two cases of large necrotic cold abscess

(2) Effects of exposure.

(*a*) Abortion occurred in some ewes; all yielded Brucella organisms. The highest dose employed barely reached the 50% abortion rate in the non-vaccinated control ewes. (See Table 5.)

(*b*) Semen infection occurred in a few vaccinated and in most non-vaccinated control rams. The dose of exposure could

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not be controlled but a very definite protection in the vaccinated groups was demonstrated as shown in Table 6, *q.v.*

(*c*) Clinically detectable lesions were observed in eight infected rams: five out of the sixteen infected non-vaccinated and three out of the four infected rams which had been vaccinated with Strain 19 showed epididymitis. (See Table 7.)

Comments received on several field tests which are still in progress are given in Table 8, *q.v.*

TABLE 5

MERINO EWES EXPOSED TO ARTIFICIAL INFECTION

Group	Number of virulent organisms	Result			
		Lambed	Aborted	Infertile	Total
Vaccinated 5419 (S19)	10^{10}	1	—	—	6 (—)(84% lambs)
	10^{7}	1	—	1	
	10^{1}	3	—	—	
Vaccinated 5711	10^{10}	1	—	—	4 (—)(75% lambs)
	10^{7}	1	—	—	
	10^{1}	1	—	1	
Vaccinated .... Elberg Rev. 1	10^{10}	2	1	—)	9 (ID_{50} 10^{10})? (67% lambs)
	10^{7}	3	—	—)	
	10^{1}	3	—	—)	
Not vaccinated	10^{10}	1	2	—)	11 (ID_{50} 10^{10})? (38% lambs)
	10^{7}	3	—	—)	
	10^{4}	2	—	—)	
	10^{1}	3	—	—)	

TABLE 6

RAMS EXPOSED TO ARTIFICIAL INFECTION WITH SEMEN

Vaccination			Exposure			Result
Number of organisms			No. of sheep	No. died	No. of times challenged	No. infected / No. tested
Rev. 1	S19	Killed				
1.25×10^{6}	20×10^{9}	100×10^{9}	1	.	1	0 / 1
2.5×10^{6}	20×10^{9}	100×10^{9}	3	.	1	1 / 3
5.0×10^{6}	20×10^{9}	100×10^{9}	2	.	1	0 / 2
—	20×10^{9}	—	6	1	1	2 / 5
—	—	—	6	.	1	3 / 6
5.0×10^{6}	—	—	10	2	2	0 / 8
5.0×10^{6}	—	—	5	.	2	0 / 5
50.0×10^{6}	—	—	4	.	2	0 / 4
—	20×10^{9}	—	9	1	2	2 / 8
—	2×10^{9}	100×10^{9}	8	3	2	0 / 5
—	—	—	9	1	2	7 / 8
0.5×10^{6}	—	—	5	1	2	0 / 4
50.0×10^{6}	—	—	5	1	2	0 / 4
—	20×10^{9}	—	9	1	2	2 / 8
—	—	—	8	1	2	6 / 7

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TABLE 7

EFFECTS OF ARTIFICIAL EXPOSURE TO BRUCELLA INFECTION (RAMS)

Vaccine	Number	Titre	Semen smears	Epididymitis
Rev. 1	25	5	—	—
Rev. 1 & S19 & K	6	80	1 pos.	—
S19	13	10	4 pos.	3
S19 & K.....	5	—	—	—
—	21	2.5	16 pos.	5

TABLE 8

COMMENTS ON FIELD TESTS STILL IN PROGRESS

Breed	No. of doses used	Diseased condition	Owner's view	Veterinarian's finding
Karakul	1504	Epididymitis: Infertility: Abortion:	1 case post-vaccination No improvement Several cases post-vaccination	Believes vaccine unsafe
Merino	2700	Epididymitis: Infertility:	Fewer cases Young rams fertility improved	Results very good
Suffolk Downs	310	Abortion:	Full protection	Results excellent
Merino	800	Epididymitis: Infertility:	(Full protection in (young rams	Satisfactory results
Merino	4400	Epididymitis: Infertility:	Young rams fully protected Improved fertility	Good results
Goats	400	Abortion:	Confident	Not yet received

DISCUSSION

Even at this preliminary stage it is possible to state that vaccination of sheep and goats in South Africa is a promising and practical method of combating brucellosis.

The results reported from North Africa, Weybridge, New Zealand and Spain are supported by our findings.

Our massive artificial exposure, augmented with close contact with rams secreting infected semen, revealed gross results showing vaccination of rams as a promising method of combating this type of Brucella infection.

Out of 49 vaccinated rams 5 became infected, leaving 90% of rams protected; whereas amongst 21 non-vaccinated rams, 16 became infected, i.e. only 19% of rams were naturally not susceptible.

Concerning the different types of vaccine to be recommended the analysis of the results as given in Table 10 favours the living Rev.1. vaccine.

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TABLE 10
ANALYSIS OF BRUCELLA VACCINE IN RAMS

Vaccine group	No. infected/No. protected	Per cent
Elberg Rev. 1 alone	0 / 25	100
Rev. 1 with others	1 / 31	97
Strain 19 plus killed cells with others	1 / 11	91
Strain 19 with others	5 / 24	80
Strain 19 alone	4 / 13	69
Not vaccinated	16 / 21	19

The different manifestations of brucellosis in this country are apparently due to infection with different types or varieties of Brucella organisms. For instance strains isolated from bovine foetuses have been typical *Br. abortus* except two aerobic variants from Pietersburg and Maseru. At Brits a typical *Br. abortus* strain was isolated from an outbreak of abortion in goats. Atypical *Br. abortus* strains (*Br. abortus* type III)[24] have been isolated from sheep in the Cape Province and appear to be present in the O.F.S. and Natal.

Br. melitensis was found in S.W.A. in human beings, cattle and sheep and proved to be the cause of abortion storms in Karakul flocks.[4]

The semen infection in rams in South Africa, which has been likened to the New Zealand and Australian "*Br. ovis*" infection is caused by organisms producing few agglutinins for *Br. melitensis* and *Br. abortus*. It is, peculiarly, more sensitive to Basic Fuchsin than to Thionin (rather like the American and European *Br. suis* strains or like the organism of Hare brucellosis in Switzerland, France, Germany and Denmark). It causes no abortion in ewes and shows no virulence for guinea pigs.

Natural immunity to all these forms of brucellosis is permanent but not solid or complete. Artificial immunity can be derived from transient infections due to live vaccines. Although Strain 19 is believed to protect against abortion in ewes[25] it appears to be inferior to strain Elberg Rev. I. in protecting rams against semen infection.

The latter is to be favoured also because of the smaller dose required which facilitates freeze drying and distribution.

At this stage it is not possible to evaluate the killed Brucella vaccine. Experiences with killed vaccine have been good in Tunis but inferior to live S.19 vaccine in Russia and to Rev.1. vaccine at Weybridge. The use of killed vaccine in the double vaccination method with Strain 19 in sheep in New Zealand, suggests that a strong antigenic stimulus producing high humoral antibody of transient nature given simultaneously with a mild invasive stimulus producing a permanent cellular immunity may have satisfactory results.

However, there are ample indications that an infection of longer duration, such as that produced by Elberg Rev.1 Brucella Vaccine, which produces a high degree of cellular immunity with, obviously, also a long continued and highly effective antigenic stimulus, will give better results. Such a vaccine is more economically produced, more

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conveniently administered and less likely to cause inflammatory reactions.

There is no longer any doubt that a useful protection against all forms of Brucellosis in sheep and goats can be obtained by vaccination with live vaccine. Although much of the nature of Brucella immunity still remains to be discovered, it can already be stated that killed vaccines of increasing efficacy are being developed. The amount and composition of the adjuvants employed play an important rôle, which is not sufficiently understood. This is particularly important in connection with the vaccination of adult sheep and goats including pregnant females.

SUMMARY

The measure of immunity attainable in sheep and goats against Brucella infection is investigated with special reference to active artificial immunization.

Preliminary results are given on the enhanced resistance to artificial exposure to be found in Merino ewes and rams after having been vaccinated with live and killed Brucella vaccines, viz:

Strain 19, *Brucella abortus*,
Strain G5711, Op, *Br. melitensis*,
Strain Elberg Rev.1, *Br. melitensis*,
and Killed Brucella organisms inoculated with adjuvant.

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SHY BREEDERS IN CANINES

J. L. Doré*

When asked to read a paper on "Shy Breeders in Canines", I was apprehensive, but on second thoughts felt I would like to set the ball rolling and gain more knowledge from the experience of others when the subject is discussed.

Strictly speaking, shy breeding should include difficulties in mating, conception, gestation, parturition and the rearing of the neonatal, but for the purposes of this paper, I have confined myself to the difficulties of mating only, because this in itself forms a complex mechanism dependent on numerous factors of the male and the female.

Failure of the male to play his part in breeding may be due to one or more factors:

(*a*) Pathological.
(*b*) Physiological.
(*c*) Psychological.

Pathological conditions embrace inflammations, tumours and malformations, and ectopic positions of the genital system. Most of these conditions are comparatively easily diagnosed, and their significance can be assessed in accordance with their severity. Whether a dog is sterile or not can very often be determined by a semen test.

The second group of abnormalities, the Physiological Group, are more baffling, and the symptoms usually to be found here are those of eunuchoidism, lack of libido, and feminism. These are caused by some imbalance of the hormone endocrine system. Exactly where to trace the cause is most difficult, and usually the practitioner will attempt to correct the imbalance by testosterone and chorionic gonadotropic hormones, with generally indifferent results. Although many of these imbalances may be brought about by trauma, infections or intoxications, probably the majority of these effeminate, obese and eunuchoidal dogs are hereditarily so disposed, and in any case undesiderable as stud. The obvious cases are easy to spot and the owner can appreciate their significance, but the borderline cases are usually difficult at stud, and the owner feels insulted when told that his dog is a "sissy" and should not be used for breeding purposes. One point, however, must be borne in mind, and that is, some dogs mature sexually far later than others, and therefore, a young dog should not be condemned too soon.

Finally in the male there is the psychological factor. Where there is no pathological or physiological deterrent, the male may not mate because he is inexperienced, and it is for this reason that many breeders will not mate an inexperienced dog to a virgin bitch. On the other hand he may be highly strung and nervous and will require some form of sedation, although more often than not, it is the bitch

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which requires the sedation. Some dogs have a fear of unfamiliar surroundings or strange people and will not mate unless they are in their usual environment and supervised by people they know. Distraction caused by the close proximity of other dogs and bitches may hamper his efforts to mate, and it is therefore advisable to keep other animals well away from the pair about to be mated. A fear of the bitch herself is a great deterrent to even good stud dogs, especially if they have already been bitten by her or other bitches, and it is important to exercise control over the bitch in many cases, to avoid the future of a good stud dog being spoilt by this fear.

With regard to the bitch, the same three factors governing shy breeders pertain, namely the pathological, the physiological, and the psychological factors.

Pathological conditions again include inflammations, tumours, and malformations. Accuracy of diagnosis is not an easy matter here, and it is usually only when symptoms of metritis or tumours or some other abnormality become apparent, that it is realised why the bitch will not breed.

It is with physiological conditions that the practitioner experiences the greatest difficulties. The ovaries cannot be palpated, and while the semen of the male can be tested, there is no such test available to establish whether the bitch will mate and conceive. Irregularities of the oestrous cycle are ascribed to hormone imbalances, and the poor practitioner in ignorance and by guesswork, takes chances with oestrogens, serum gonadotropins, and luteinising hormones. It is here that I seek assistance and knowledge and leave it to others to discuss.

The psychological factors responsible for shy breeding in bitches, still require much study of animal behaviour. She will refuse mating in the same psychological circumstances as the male, except that she does not seem to fear a bite from the male. There are, however, other reasons in addition which will cause her to refuse the male. Firstly, she will usually only accept the dog during the oestrous stage, and not during pro-oestrum or metoestrum, but there are probably numerous exceptions to this rule, making it very difficult to calculate the day of ovulation. Secondly, a bitch will very often show individual choice of her partner. She will usually indicate her willingness to stand for the male by turning her tail to the side when the base of the tail is scratched. There is many a bitch that will show this acquiescence, yet when the dog is produced she will fight him off vigorously, but will mate with another of her own choice. Thirdly, there are bitches that have been so mollycoddled by human beings that they will refuse mating in spite of the normal sexual urge.

When a bitch refuses submission because she is not in true or full oestrum, it is positively wrong to try to mate her. In many cases the bitch does require a certain amount of control, either by holding, or by the administration of tranquilizer. There are many bitches, especially if they are strange to the male, that wish to indulge in a certain amount of "love romping" prior to submission, and it is usually advantageous to allow this because it is her prerogative to

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accept or reject the male, and this romp gives her the opportunity to sum him up.

The time of the day does not have much influence on mating, but it is advisable to avoid the heat of the day, especially in brachycephalic dogs that find it very exhausting. Other external influences may be slippery or highly polished floors, which may make mating difficult, and care should be paid to this aspect of mating. So often owners seeking privacy do not take slippery floors into account.

Many owners find it difficult to assess the start of the pro-oestrum which usually lasts about seven to twelve days. This is not very necessary, provided they can recognise the oestrous stage which lasts anything from three days up to ten days, the average being about five days. As ovulation takes place about forty-eight hours after the first acceptance of the male by the female, it is advisable to defer the first mating for about forty-eight hours from the time she will stand. A second mating can be done forty-eight hours later, and if the oestrus period lasts longer than the average five days, a third mating can be done forty-eight hours after the second. Three services for one bitch by a busy stud dog is heavy, and may be one of the factors that cause sterility in show dogs. Owners who know their bitches well, can obtain good litters from only one mating at a time—this usually requires careful observation and recording of the oestrous cycle. The tendency among most owners is to try to mate too early. The dog becomes worn out, and eventually disinterested through frequent "try outs".

It must also be remembered that as the bitch becomes older, her seasons shorten, and under these circumstances it is wise to mate her earlier than the younger bitch.

Practitioners are often asked whether it is necessary to have a "tie" or "lock" at a mating in order for the bitch to conceive. This is a difficult question to answer because on asking the owner how long they were mated before "breaking", the owner is unable to state that it was half a minute, or one or two minutes or longer. The first fraction of the ejaculate—the clear fluid from the mucosal glands of the urethra—contains no spermatozoa, and takes about half a minute to be voided. Obviously if the mating has taken less than half a minute no conception could take place. The second fraction containing the spermatozoa takes about one or two minutes to be ejaculated, and the third fraction of the ejaculate from the prostate gland which contains no spermatozoa, but is an activator of the spermatozoa, and supplies the greater volume of the ejaculate, may take from five to thirty minutes to be voided. It is not necessary to have any of the third fraction present for conception, it is probably useful partly as an activator, and partly as forming bulk to be washed forward into the uterus by the rhythmical action of the vagina. It can be appreciated therefore that if the first two fractions have been deposited well forward in the vagina, there is quite a good chance of conception, even though the mating may have lasted about one and a half minutes.

At this juncture it may be advantageous to consider artificial insemination. Theoretically speaking there should be no necessity

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for it, but certain circumstances are found when it can be applied, although the South African Kennel Union frowns on the practice. Successful conceptions have taken place up to five days after the ejaculation of the male, and semen has been flown over vast distances with successful results. The keeping qualities of canine semen is poor, however, and most of the requests for artificial insemination are made because the owner has not been able to mate the dog and bitch naturally, and consequently the keeping of semen does not enter into the picture.

Not many dogs are good donors, and as a rule a dog will only ejaculate if he has been teased by a bitch. Collection can be done by gently and rhythmically squeezing the penis through the prepuce immediately behind the bulb. The ejaculate can then be caught in a clean glass vessel of a suitable size. Alternatively, Harrop has devised a good artificial vagina with a warm water jacket which can be rhythmically pumped by a bulb. The semen amounting to 2–25 m.l. is then drawn up into a glass syringe and a plastic catheter is attached to the syringe nozzle. It is then injected well forward into the vagina, and the bitch is held with her hind quarters well up to allow the semen to run as far forward as possible. It should be routine in all cases to examine a drop of the semen microscopically; it does not take long and is worth while.

I trust I have laid the foundation for a general discussion and hope that others will contribute towards more knowledge on this subject.

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A CLOSED CIRCUIT METHOD OF ANAESTHESIA IN SMALL ANIMAL PRACTICE

P. H. le Roux*

The long-acting barbiturates have several disadvantages when used as general anaesthetics on small animals. The main ones are: the erratic response to the computed dose, the long recovery phase and the severe postoperative depression in weakened patients. These unwanted effects may be lessened by various means, but never entirely eliminated.

In an attempt to overcome these problems a closed circuit method of administering ether was investigated. In experimental thoracic surgery on dogs, wide use is made here and abroad of closed or semi-closed systems of artificial respiration with muscle relaxants and nitrous oxide as the anaesthetic. In veterinary literature, references to these methods are not very common, and I am therefore grateful to Dr. Vetten of Johannesburg General Hospital and the Department of Surgery, Faculty of Veterinary Science at the Onderstepoort Veterinary Research Institute, for their help and encouragement.

The system finally arrived at was built up as follows from used equipment: a rubber bag serves as an air reservoir and allows space for the respiratory movement of the gases. A Ruben valve sends the gases round in one direction through the circuit. A soda lime cannister removes the carbon dioxide from the exhaled air. An ether vapouriser supplies additional anaesthetic as needed. A Heidbrinck valve serves as a safety valve. Two T-pieces and two lengths of corrugated rubber tubing complete the circuit. Additional oxygen, nitrous oxide and carbon dioxide can be let into the circuit in the proportions required through a Bobbin-type flow meter. The patient is attached to the circuit by means of a cuffed or plain endotracheal tube connected to the Ruben valve. The total "dead space" is approximately 5 cc.

With this apparatus spontaneous respiration or positive pressure artificial respiration is possible as a closed circuit, semiclosed or open system. When used as a closed system, the flow of oxygen is intermittent and only sufficient to make good the loss to the animal. When a semiclosed method is needed a portion of the expired air is allowed to escape through the half-open Heidbrinck valve and a portion is left for rebreathing. If an open system is required, the soda lime absorber is eliminated and all the exhaled air escapes at the expiratory end of the Ruben valve. The explosive property of ether makes it advisable to use it in a closed circuit only, while the large gas flow required with nitrous oxide and oxygen makes it easier to use as a semiclosed system. An open system necessitates such large volumes of gas that it becomes impractical.

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Preoperative sedation deep enough for the patient to be intubated can be produced by various drugs. In dogs thiopentone sodium, thiambutine hydrochloride and chlorpromazine have been used satisfactorily. If succinyl choline chloride is used, very little additional anaesthetic is required, but as it makes artificial respiration necessary it is only practical in thoracic operations. If chlorpromazine is used, the animal must first be anaesthetised using a face mask before a cuffed Magill tube can be inserted through the glottis.

In cats spasm of the glottis makes it very difficult to pass a tube even though the animal has been deeply anaesthetised with thiopentone sodium intravenously. Tetracaine hydrochloride swabbed on to the epiglottis and its surroundings suppresses this reflex. In cases where thiopentone is contraindicated, largactil intravenously and ether administered by means of a face mask is reasonably satisfactory.

This apparatus is in continuous use for all cases requiring general anaesthesia for more than about ten minutes and has been found perfectly satisfactory and simple to use.

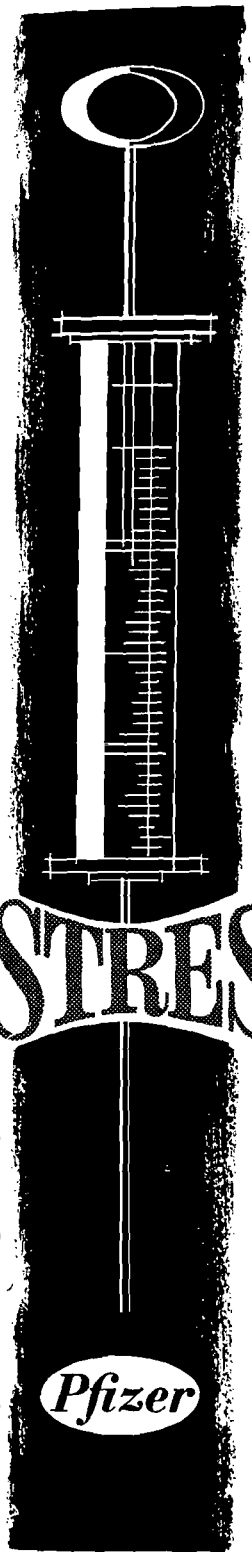
Deep anaesthesia is produced on demand and maintained for as long as required and no longer. The recovery is smooth and rapid, and there is little danger of even severely shocked patients dying during the recovery stage due to respiratory depression. Positive pressure is available for the occasional thoracic operation and if a patient should get into difficulties during the operation. A free airway is always available so that vomitus, mucus and bloodclots cannot be aspirated into the lungs. The cost of operating the machine is negligible if ether is used as the anaesthetic.

The only disadvantage in this method lies in the fact that it takes longer to perform a batch of operations, as each patient must be anaesthetised immediately before the operation. The wisdom of having a few deeply anaesthetised animals lying about while you operate on another is in any event questionable.

The time wasted in scrubbing up between operations is more than compensated for by the fact that when the last operation is completed, all the animals are awake and capable of caring for themselves until they are fetched by their owners.

I am convinced that this method of anaesthesia is the safest and most satisfactory method now available. I should like to see this method become more generally used, especially in small animals where it is completely practical at the moment, and perhaps later in the larger species as well.

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OORWEGINGS TEN OPSIGTE VAN SKOK EN DIE SWAK-PROGNOSE OPERATIEWE PASIENT

C. F. B. Hofmeyr*

„There is hardly any limit to the extent of a successful surgical venture as long as one can maintain the circulation" — Dr. John Elliott.

Skok is 'n verskynsel wat altyd in die gedagte van die chirurg is, aangesien dit so dikwels mag optree as 'n komplikasie van ernstige operasies en so prominent teenwoordig is in 'n groot deel van chirurgiese gevalle met swak prognose. Hoewel die laaste woord oor skok nog lank nie gesê is nie, is daar reeds feite bekend wat ons in staat stel om skok die hoof te bied.

Oorsig van Literatuur

Koger[17] beweer dat sistemiese „stress" by besmetting of trouma optree a.g.v. relatiewe tekort aan kortikoïede. Levine, Huddlestun, Persky en Soskin[18] sê dat daar progressiewe weefselanoksie in skok bestaan en dat die oksiderende meganisme van senuweeweefsels so beskadig word. Asidose kan beskryf word as die vergete faktor in skok. Hulle kwoteer ander outeurs oor die verhouding tussen skok en asidose en deel mee dat hulle bevindings ooreenstem. Daar is grafies 'n reglynige verhouding tussen val in bloeddruk en vermindering van die alkalieréserwe. In neurogeniese skok is daar egter geen of geringe asidose.

Melcher en Walcott[19] het die werking van skok op die hartspier nagegaan en gevind dat selfs in dié gevalle wat klinies herstel het na langdurende skok, vettige infiltrasie en nekrose van die miokardium plaasgevind het. Anoksemie word voorgehou as die oorsaak.

In dieselfde rigting het Brown en Miller[9] die effek van koolstofdioksied ondersoek. Asemhaling het opgehou wanneer CO_2-konsentrasie in ingeasemde lug 60–65% bereik het, en die bloeddruk het begin val met 'n konsentrasie van 50–70%. Kunsmatige asemhaling moes toegepas word. Hartarres het plaasgevind met konsentrasies van 90% en meer. Op dié stadium was die bloed pH 6.41.

Weil[23] het die lopende gedagtes oor die oorsake van skok in die volgende groepe saamgevat:—

(1) Vermindering van die volume bloed.
(2) Hartversaking.
(3) Bakteremie.
(4) Oorgevoeligheid.
(5) Neurogeniese oorsake.
(6) Obstruksie van sirkulasie.

Hall en Gold[13] het hulle toegespits op bakteremie as 'n oorsaak van skok. Hulle vind dat dit in die mens baie spoedig die dood

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teweeg kan bring. Uit 100 gevalle het 35 hipotensie gehad. Tweederdes van hierdie gevalle het Gram-negatiewe organismes in die bloedstroom getoon. In 50–75% van gevalle was die urogenitale stelsel die bron van die besmetting. Van die res was die maagdermstelsel die volgende mees algemene bron. Kolon en parakolon basille, *Bacillus proteus*, *Pseudomonas aeruginosa* en *Aerobacter aerogenes* was die gewone organismes. Vireers mag die tekens nie tipies vir skok wees nie: die geval is wakker met bonsende pols en koors. Lae bloeddruk mag die enigste aanduiding wees van bakteremiese skok.

Schweinburg, Frank en Fine[21] het die bakteriese faktor in eksperimentele bloedingskok in honde nagevors. Die gevalle het gewoonlik herstel wanneer die bloed weer ingetap is na 2 uur van bloedingskok. Wanneer hulle ingespuit word met gemaalde lewer van normale honde tree herstel ook op, maar hulle gaan dood indien gemaalde lewer van honde in terminale skok ingespuit word, tensy hulle met penisillien behandel word. Daar is 'n verhouding tussen bakteriële aktiwiteit en die omkeerbaarheid van bloedingskok.

Frank, Jacob, Schweinburg, Goddard en Fine[11] het gevind dat indien 40% van maksimum-bloedingsvolume van 'n hond uitgebloei word, reïnfusie alleen 'n tydelike pressorreaksie veroorsaak, daarna volg terugkeer van skok en dood in 90% van gevalle. Clostridia is gevind in die lewers van 91% van 66 honde na bloedingskok en in 35% van 96 normale honde. Aerobiese organismes is minder dikwels gevind in lewers van honde in skok maar meer dikwels as in die geval van normale honde. Al hierdie organismes is normalerwys teenwoordig in die dermkanaal. Met bloedingskok sonder antibiotiese behandeling het 12% gevalle herstel. In die geval van behandeling met een dosis aureomisien het 45% herstel en waar langdurige antibiotiese behandeling toegepas is, het 88% herstel.

In toestande van anoksie mag ingewandsclostridia 'n bloedvattoksine of weefseltoksine afskei wat sellulêre outoliese tot gevolg het. Met oorvermoeienis of in uremie vind ingewandskieme maklik toegang tot die sirkulasie. Die waarskynlikheid dat weefselanoksie die weg mag baan vir instroming in die lewer e.a. weefsels van ingewandsorganismes of dat dié wat passief in die lewer is tot aktiwiteit geprikkel mag word, word genoem.

Ingraham, Goldberg, Roemhild en Wiggers[15] het vasgestel dat pentobarbital geen invloed het op die verloop van bloedingskok nie.

Bailey[2] het die baanbrekende werk van Selye ten opsigte van die verklaring van die skoksindroom kort saamgevat. 'n Franse chirurg het in 1934 'n toestand beskrywe wat tipiese sirkulasieafwykinge getoon het en dikwels gevolg het op groot chirurgiese ingrepe. Hy het dit genoem "maladie postoperative".

In 1936 het Selye waargeneem dat 'n verskeidenheid van beskadigende faktore 'n uitskeiding van bynierskorshormone veroorsaak, wat verhoogde weerstand teen hierdie faktore bewerkstellig. Verder, in die afwesigheid van uitskeiding van dié hipofise is verskynsels van beskadiging baie meer opsigtelik (bv. hipotermie, hipoglisemie) terwyl die verdedigingsmeganisme (hiperglisemie en dissolusie van limfositêre selle) uitgeskakel word. Daarom is dit logies om die liggaam te help

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met die toediening van bynierskors-steroïede. Selye het toe sy teorie van die Algemene Aanpassingsindroom (AAS) (General Adaptation Syndrome) ontwikkel.

Die AAS het drie stadia:—

(1) *Die Alarmreaksie.*

Dit het 'n (*a*) skokfase en (*b*) teenskokfase.

Die skokfase toon algemene depressie, val in temperatuur, spier- en bloedvattonus, bloedkonsentrasie, verhoogde kapillêre en seldeurlaatbaarheid, hipokloremie, asidose, hypoglisemie, leukopenie gevolg deur leukositose, eosinopenie en akute maagdermerosies.

Die *teenskokfase* toon vergroting van die bynierskors en verhoogde uitskeiding en in die algemeen 'n omkering van die skokfase.

(2) *Die Stadium van Weerstand.*

Die liggaam ontwikkel 'n verhoogde weerstand teen die spesifieke toestand waarteen hy reageer, maar word tegelykertyd meer vatbaar vir ander prikkels.

(3) *Die Uitputtingstadium.*

Deur aanhoudende blootstelling aan skadelike prikkels waarteen aanpassing ontwikkel het, word die liggaam uitgeput en mag die toestande van die alarmreaksie terugkeer.

Keating[16] het hom grootliks op dieselfde veld toegespits en ook 'n opsomming gegee van Selye se teorie.

Weil[23] verklaar dat bloedvatsametrekmiddels aangewys is in hartversaking en oorgevoeligheid, maar teenaangewys in bloedvatobstruksie en bloedvolumetekort totdat volume herstel is. In teenwoordigheid van asidose is reaksie teenoor vasopressors baie verminder.

Levine *et al* [18] het bloedingskok verwek op 64 honde ten einde sg. „onomkeerbare" skok te bestudeer. Die bloedvolume is bereken as 1/13 van liggaamsgewig. Bloeding is uitgevoer volgens 'n vaste patroon totdat die sistoliese druk na 50 mm Hg en laer gedaal het—so laag moontlik sonder om onmiddellike dood te veroorsaak. Terapie is weerhou totdat die CO_2 kapasiteit van die bloedplasma na 15 vol. % of minder gedaal het. Die stadium bereik was dié van sg. onomkeerbare skok.

Die proefdiere is verdeel in sewe groepe. Dié wat onbehandel is, is almal dood; 25% van die groep het bly leef nadat uitgetapte bloed weer ingetap is; 50% het geleef met bloed en natriumsuksinaat (0.42 gm. anhidr. sout/Kg); 75% het geleef met bloed en natriumlaktaat (0.58 gm/Kg); 57.5% het geleef met bloed en glukose (0.38 gm/Kg); 50% het geleef met bloed en natriumbikarbonaat (0.43 gm/Kg); en 62.5% het geleef met bloed plus glukose plus natriumbikarbonaat.

Koger[17] noem dat prednisone waardevolle ondersteuningterapie is in die behandeling van skok en stress na uitgebreide beserings of groot chirurgiese ingrepe.

Bailey[2] beweer dat prednisone die hersteltyd van groot operasies met een-derde kan inkort. 100 mgm. prednisone binnespiers word beskou as voldoende vir operasies vir troumatiese gastritis in die bees.

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50 mgm. binneaars voor operasie en 50 mgm. binnespiers na operasie is miskien beter. Skok in groot diere word aangetoon deur 'n stadige, swak pols, dispnee, subnormale temperatuur, glasige oë en swakheid.

Olga Uvarov[22] het mededelings gedoen oor die kortikosteroïede in diergeneeskunde. Die metabolisme van o.a. koolhidraat word beïnvloed: glikogenese van eiwit of vet, Na- en Cl-terughouding mag voor kom, gepaard met verhoogde K-uitskeiding, wat die gevolg mag wees van eiwitopbreking en direkte werking op die nierbuisies.

Die volgende effekte op mesenkiemweefsels kan waargeneem word: verhoogde kapillêre weerstand en verminderde deurlaatbaarheid van weivliese.

Sekere gevalle van skok reageer nie op bloedplasma, volume-aanvullers en vasopressormiddels nie. Aanduidings is dat hierdie gevalle te wyte is aan akute tekort van die adrenaalskors. Dit word gepostuleer dat kortikosteroïde die werking van bloedvatvernouers versterk. Die hoofteenaanwysings in die gebruik van die kortikosteroïde is soos volg:

(1) Hart- en nierswakheid.
(2) Degeneratiewe oogsiektes.
(3) Besmettings (tensy volle antimikrobiese behandeling hiermee gepaard gaan).

Plotselinge onttrekking, veral na langdurige toediening van hierdie groep middels is gevaarlik.

In hierdie verband kan bygevoeg word die bevindings van Gy. Molnar en Szilvasy[20] dat die steroïde van die bynierskors verklewings van die buikvlies kan verhoed.

Na verwysings in die literatuur oor skok en die gebruik van die steroïde is dit wenslik om na te gaan wat aanbeveel word t.o.v. bloed en ander intraveneuse vloeistofbehandelings. In hierdie opsig is dit ook van belang om te sien wat verskillende werkers meedeel omtrent bloedvolume, bloedoortappings e.d.m.

Bild[3] kwoteer Selye as sou die bloedbesinkingspoed in stress-toestande verhoog wees. Bild noem ook dat as die hematokritlesing 30% of minder is, hy 'n bloedoortapping gee en miskien ook binneaarse vloeistof.

Bild[4] bespreek vloeistofterapie in die hond. Hy waarsku dat hartblokkering, longkollaps en uitputting soms die gevolg kan wees van verkeerde gebruik van elektroliete.

Dit is nie baie jare gelede nie dat vasgestel is dat Na- en Cl-ione in die liggaam dissosieer en dat die verhouding in die bloed nie 1:1 bly soos in NaCl nie. Die pasiënt mag ly aan 'n tekort aan H_2O of NaCl of ander soute. In die mens na chirurgiese stress en narkose is die niere nie daartoe in staat om soute te elimineer nie—d.i. stress mag lei tot soutterughouding. In die mens is dit dus dikwels die gebruik om na operasie water en dekstrose in te tap (plus, dikwels, vitamine en antibiotika).

In die hond is dit geregverdig om veelvuldige elektroliete, dekstrose en water intraveneus te gebruik omdat mens en hond biologiese verskil toon. 'n Hond in skok vorm geen urine nie totdat die toestand beredder

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is. Skok kan dikwels verhoed word deur vroeë vloeistofgebruik. As 'n opwekker gedurende of na narkose gebruik Bild nou laktaat-Ringeroplossing: 22–33 ml/Kg. binneaars. As roetine voeg hy by elke liter 2 ml. 10% kalsiumglukonaat, 1,000 mgm. askorbiensuur, 10 ml. van vitamien-B-kompleksoplossing en 500,000 eenhede kristalvormige penisillien. Daar is waarskynlik 25 maal meer gevalle wat elektroliete nodig het as bloed. Bloed word toegedien teen 18 ml/Kg. Opgewondenheid in 'n geval lei tot oorventilasie en alkalose. Onderhuidse of intraperitoneale inspuiting van dekstrose in 'n baie siek geval kan lei tot die dood aangesien hierdie oplossing verdere onttrekking van vloeistof uit die sirkulasie sal aanmoedig.

In laktaat-Ringeroplossing (Hartman se oplossing) is die soute in min of meer dieselfde verhouding as in die hond se plasma en kan wel by geleentheid onderhuids ingespuit word.

Bild[5] beveel aan dat bloed in die aar gegee moet word en die volume gelyk moet staan aan dié verloor, plus 22 ml/Kg., as daar dreigende skok is. Hy meen dat daar altyd bakteremie is wanneer skok teenwoordig is in die hond. Hoe minder barbiturate gebruik word in skoktoestande, hoe minder die skok.

Bild[6] herhaal sommige van sy vorige standpunte en voeg by dat in skok daar dikwels metaboliese asidose is. In gevalle van aanhoudende vermeer of diarree of van geen inname van vloeistof kan 33–44 ml/Kg. laktaat-Ringer binneaars toegedien word.

Bonnycastle en Cleghorn[8] het 106 ongeoefende honde gebruik om die plasmavolume (31.8–64.6 ml/Kg.), selvolume (21–49.2 ml/Kg.) en totale bloedvolume (60–107.5 ml/Kg.) vas te stel.

Gibson, Keeley en Pijoan[12] het gevind dat in honde wat 5–30 Kg. weeg, plasmavolume 41.2–51.7 ml/Kg., selvolume 36.4–54.6 ml/Kg. en totale bloedvolume 84–97.3 ml/Kg. is.

Wat die bloeddruk betref het Hamilton, Pund, Slaughter, Simpson, Colson, Coleman en Bateman[14] in 215 honde gevind dat dit baie varieer. In baie jong honde was die druk minder as in ouer honde. In die gevalle met gehialiniseerde arteriole en glomerulonefritis was die druk hoër as die gemiddelde. Sistoliese druk was 100–275 (gem. 180) mm. Hg. en diastoliese druk 30–140 (gem. 89) mm. Hg.

Aforsky[1] gee syfers vir verskillende bloedbestanddele, maar beweer dat die skommeling so groot is dat normale waardes nog nie aangegee kan word nie.

Die bloedgroepe in honde word bespreek deur Young, O'Brien, Swisher, Miller en Yiule[24]. Hulle het vyf verskillende isoteenliggame gevind maar dit is nie juis van groot kliniese belang nie. Die sg. „Canine A Factor" is wel belangrik aangesien 37% van honde hierdie faktor nie het nie en 63% wel. Dit word bereken dat 23% van die onuitgesoekte oortappings die moontlikheid het om anti-A liggame in die ontvangers te verwek. Die A faktor is hoogs antigenies en anti-A kan so vroeg as 9 dae na oortapping voorkom. Hemolitiese anemie is al gevind in A-positiewe klein hondjies van tewe wat A-negatief is en A-positiewe bloed ontvang het.

Ferguson[10] het op dieselfde gebied gewerk. Hy noem dat hemolitiese siekte (soos hierbo genoem) kan voorkom in perde, varke en

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honde. Hy verklaar dat die hond baie min of geen natuurlike teenliggame het nie, maar dat daar deur iso-immunisasie sekere antigene gedemonstreer kan word in die rooiselle van die hond.

Bland[7] in 'n omvattende studie van vloeistofmetabolisme het o.a. die volgende waarnemings gedoen:—Die presiese oorsaak van *metaboliese asidose* soos gesien in skok moet nog verklaar word. Klinies toon hierdie asidose 'n hyperpnee wat besonder diep is en al die hulpasemhalingspiere in gebruik neem.

Metaboliese alkalose toon homself deur vlak en onegalige asemhaling-hypopnee. Hierdie alkalose word nie reggestel alvorens die liggaamskalium aangevul word nie.

Asemhalings-asidose toon hyperpnee gedurende rus en dyspnee met ligte oefening—hoofsaaklike oorsaak is longemfiseem. *Asemhalings-alkalose* tree op a.g.v. hiperventilasie—soos met groot opgewondenheid.

Gedurende aktiewe skok ly alle weefsels aan die uitwerkings van verminderde bloedvloei. Funksionele versaking van sulke kritiesbelangrike organe as die lewer en niere dra by tot die metaboliese en biochemiese chaos. Die uiteindelike oorsaak van dood by skok van watter etiologie ook al is die ernstige versteuring van die liggaamswatervolume en elektrolietkonsentrasie sowel as die verandering van die vloeistofverhoudings tussen die intrasellulêre en ekstrasellulêre kompartemente.

Die metaboliese veranderinge in skok is soos volg: hiperglisemie volg gereëld op bloeding en trouma, asook as reaksie op werking van ander stressors. Indien skok voortsnel tot onomkeerbaarheid is daar 'n geleidelike val in bloedsuiker.

In alle gevalle van oligemiese skok is verlies van plasmaeiwitte algemeen van albei albumen en globulien. Daar volg dus nou 'n vermindering van kolloïdale osmotiese druk van die bloed, en so word die herstel van bloedvolume bemoeilik deurdat intappings van elektroliet- en glukoseoplossings nie in die sirkulasie bly nie.

Die belangrike fisiologiese afleiding is: hoewel elektroliet- en glukoseoplossings mag help om bloedvolume te herstel, mag hulle vir 'n gevorderde skok waardeloos wees of selfs kwaad doen.

Gedurende die alarmreaksie van Selye reageer die sindroom nie op die toediening van sout en chloried nie, die geval mag selfs erger word. Reaksie is vinnig sodra kaliumsoute toegedien word. Die rede is dat die hiperfunksionerende bynierskors terughouding van natrium en chloried veroorsaak en uitskeiding van kalium. Binne-aarse vloeistowwe voordat die geval aan bloedvolumetekort, skok of bloeding ly, baat nie, aangesien die liggaam gou van oormaat volume ontslae raak.

Die doeltreffendheid van bloed in skokbehandeling is aan die rooisel te danke. Dit kan nie van die sirkulasie ontsnap nie, is uitstekend as buffers en die hemoglobien tree op as 'n waterstofioonakseptor.

Van die pressormiddels is norepinefrien (noradrenalien) die beste. Dit veroorsaak geen verhoogde hartwerking of prikkeling nie, maar wel

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verhoogde perifere weerstand a.g.v. bloedvatvernouing. Die werking is ook van betreklik korte duur. Uiteindelik kan skok gedefinieer word as „'n toestand waarin die volume van die sirkulerende bloed te klein is vir die kapasiteit van die vaatbed".

EIE OORWEGINGS

Die beeld van troumatiese skok soos so dikwels waargeneem na straatongelukke is goed bekend aan die klinikus: die koue ore en ledemate, die bleek, dikwels sianotiese slymvliese, die hartslag wat vinnig en swak mag wees, of stadig en onreëlmatig en die vinnige diep asemhaling met al die bykomende asemhalingspiere ingespan. Hierdie beeld lyk besonder veel na terminale buikbloeding en is soms verbonde aan dergelike bloeding. Die enigste manier om seker te maak is parasentese.

Verder is alle klinici bekend met die voorkoms van die geval van distokia in die teef wat miskien baie ure lê met een of meer emfisemateuse fetusse in die uterus: dikwels koue uiteindes, ingesonke oë, gebrek aan velelastisiteit, blou-bruinerige getinte slymvliese, subnormale temperatuur en 'n swak, gewoonlik vinnige pols. Verder kan die verwante beeld genoem word van die hond met gevorderde dundermobstruksie: aanhoudende vermeer, ontwatering en die res van die skokbeeld. Indien ernstige bloeding in die eerste geval teenwoordig is en in altwee die laasgenoemde gevalle, is operasie dringend nodig, maar elke chirurg weet dat sy pasiënt waarskynlik op die operasietafel sal omkom indien sonder meer geopereer word.

Welke chirurg het nie die geval gehad wat in redelike toestand op die operasietafel kom en óf deur bloeding óf deur niébloedingskok deur sy vingers gly nie?

Dit is dus vir die chirurg om, sovêr hy kan, 'n oordeel te vel oor die toestand van die geval voordat geopereer word. Afgesien van die kliniese tekens aangestip, is daar ander aanduidings wat van hulp is. Bloedarmoede, al dan nie, kan min of meer klinies vasgestel word, of, indien fasiliteite beskikbaar is, deur middel van die hematokrit. Ontwatering word aangedui deur gebrek aan vel-elastisiteit, droë slymvliese en ingesonke oë. Indien daar 'n gebrek aan balans van elektroliete is, dan is die toestand wat dit veroorsaak het, dikwels 'n gids oor watter elektroliete te kort mag wees.

Indien daar 'n vreemde voorwerp in die maag is, sal die vermeer 'n verlies van chloor-ione teweegbring en dus eventueel alkalose. Indien die obstruksie verder na agter is, bv. na die uitmonding van die gal- en pankreatiese buise, sal daar tegelykertyd 'n verlies van natrium-ione wees: dus 'n meer gebalanseerde elektrolietverlies. In geval van ernstige diarree is daar belangrike verlies van kalium-ione. As algemene aanduiding kan aangeneem word dat met alkalose die asemhaling vlak en stadig is en met asidose is dit diep en vinnig.

Indien die vreemde voorwerp in die maag is, sal vermeer spoedige ontwatering meebring aangesien daar min geleentheid sal wees vir opname van vloeistowwe uit die maag. Indien die obstruksie verder aan in die dunderm is, sal ontwatering stadiger optree hoe verder die

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voorwerp na agter geleë is, omdat meer derm beskikbaar sal wees vir vloeistofopname.

Die ideale toestand is eintlik dat 'n kliniese-patologiese laboratorium die elektrolietbalans, hematokrit ens., moet vasstel. Aangesien hierdie fasiliteite vir min diereartse toeganklik is sal ons ons hier bepaal by kliniese aanduidings.

Soos aangedui draai die skoksindroom eintlik om die toestand van die arteriole en kapillêre. Die haarvaatjies word beskadig deur bv. die volgende faktore:

(*a*) Bakteriese toksines.

(*b*) Anoksie wat weer bevorder word deur

(i) Bloeding.

(ii) Ander oorsake van lae bloeddruk soos swak hartwerking.

(iii) Enige faktor wat genoeg suurstofopname verhoed.

(*c*) Histamien of histamienagtige stof van weefselkwetsure.

Wanneer daar nou 'n bloedvatverwyding plaasvind na beskadiging en die haarvatdeurlaatbaarheid verhoog word, stroom elektroliete en die vloeibare deel van die plasma in die weefsels in. Daar is 'n val in bloeddruk, 'n verdere anoksie, hemokonsentrasie en verder haarvatbeskadiging. Grootskaalse verlies van kolloïde vind ook plaas wanneer daar uitgebreide trouma is.

Die terapie word nou daarop toegespits om die kringloop te onderbreek, of, indien die sindroom nog nie ontwikkel het nie, op maatreëls om dit te verhoed.

(1) In alle gevalle word verseker dat daar 'n vrye toevoer van lug of suurstof sal wees. In veral die brachycephaliese rasse word na narkose die tragea intubeer met Magill se endotrageale kateter omdat in hierdie gevalle die lang uvula dikwels die asemhaling bemoeilik. Indien enige aanduiding daarvoor is, kan die suurstofsilinder maklik ingeskakel word.

(2) Indien daar 'n skoktoestand vir enige geruime tyd bestaan het, of indien die geval skok na 'n straatongeluk opgedoen het, word antibiotika (gewoonlik penisillien) gevoeg by die binne-aarse vloeistof wat toegedien gaan word. Indien daar 'n uitgebreide bakteriese toestand en die gevaar van skok bestaan, word die penisillien ook toegedien. *In gevalle van bestaande skok word inspuitings alleen intraveneus toegedien, aangesien opname onderhuids, intraperitoneaal, rektaal of per os onseker is.*

(3) Waar skok teenwoordig is, of verwag word, as gevolg van bestaande getroumatiseerde weefsel of moontlik as gevolg van verwagte chirurgiese trouma, is dit goeie beleid om 'n antihistaminiese middel in die infusievloeistof in te sluit.

(4) Voorbehoedend kan hidrokortisoon of een van sy derivate binnespiers ingespuit word, of, waar skok bestaan, binne-aars. Die bynierskorssteroïde bevorder die integriteit van die haarvate.

(5) Met bloedverlies is die ideaal om die verlore bloed met bloed te vervang. Daar dien egter onthou te word dat, indien 'n A-negatiewe-

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teef met A-positiewe bloed ingespuit word, haar kleintjies, as die vader A-positief is, heel moontlik aan hemolitiese anemie kan doodgaan sodra hulle kolostrum na geboorte drink.

(6) Indien daar geen bloedverlies is nie, en daar is geen besondere bloedarmoede nie, is bloed tog waardevol om die kolloïde wat dit bevat. In hierdie opsig vul die rooisel ook 'n waardevolle funksie. Andersins kan plasma gebruik word as 'n ideale vloeistof om die volume van die sirkulasie en die osmotiese druk van die bloed op peil te bring. By afwesigheid van plasma is die bekende 6% gelatien ('n proteïn wat feitlik geen antigenisiteit het nie) van groot waarde vir binne-aarse toediening. In skok waar daar geen elektrolietverlies was nie, is glukose- en fisiologiese soutoplossing soms selfs skadelik, aangesien dit die reeds oormatige weefselfvloeistof aanvul en nie in die bloedsomloop bly nie.

(7) Met ontwatering en elektrolietverlies is aanvulling van die bloedvolume noodsaaklik. Veral in die afwesigheid van laboratorium toetse, is dit moeilik om die presiese relatiewe hoeveelhede van die elektroliete vas te stel. In die geval dus, is 'n vloeistof soos laktaat-Ringer, wat veelvoudige elektroliete bevat, wenslik. Hierby kan 5% dekstrose gevoeg word aangesien dit maklik bekombare energie voorsien. Waar skok teenwoordig is, moet aandag gegee word aan die metaboliese asidose wat dit kompliseer. Hier is die byvoeging van natriumbikarbonaat by die intappingsvloeistof (as 5% oplossing) aangedui. 'n Verdere byvoeging is dié van die beste vasopressormiddel onder hierdie omstandighede: noradrenalien of norepinefrien. Enige vasopressormiddel se werking is beperk gedurende asidose, maar wanneer die asidose onder beheer is, tree die volle werking op. Die noradrelien gee tonus aan die arteriole, sonder om die hart aan te tas, en veroorsaak 'n styging in die bloeddruk.

Die toediening is nou soos volg: Voor operasie word die toestand van die pasiënt in oënskou geneem. Is skok teenwoordig dan word die behandeling soos hierbo uiteengesit, toegepas. In die afwesigheid van skok word bloed en elektroliete aangevul soos benodig. Waar die skok gepaard gaan met 'n chirurgiese toestand wat dadelik operasie vereis, dan gaan die behandeling aan gedurende operasie. Indien operatiewe skok verwag word in 'n geval wat in 'n niegeskokte toestand onder operasie gaan, word die nodige voorsiening gemaak volgens die aard van die skok verwag. Voor die ingryp word hidrokortisoon binnespiers ingespuit (of gedeeltelik binnespiers en gedeeltelik binne-aars). 'n Bottel met bloed, indien moontlik (of plasma, of 6% gelatien word opgestel en na narkose in die sirkulasie ingeskakel, met net 'n druppel nou en dan om te verhoed dat die pasiënt se bloed in die naald stol. Dieselfde word gedoen met laktaat-Ringeroplossing, en 'n byvoeging van 0.5–1.0 ml. noradrenalien (soos Levophed) per 100 ml. vloeistof en vitamien-C en ander inspuitbare vitamine kan ook bygevoeg word.

Gedurende operasie kan die inloop van die vloeistowwe gereël word na gelang van die geskatte bloed- en ander vloeistofverlies. Dit

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is belangrik dat die twee met mekaar tred moet hou. Dit is egter wenslik om eerder effens teveel as te min vloeistof toe te dien.

Ten spyte van wat ander outeurs mag dink, kan barbiturate tot skok predisponeer aangesien hulle soms die asemhaling onderdruk en dus 'n hipoksie mag veroorsaak. Die suurstof moet dus byderhand wees.

Daar dien gelet te word op 'n paar feite. Die bloed-volume van die hond is 6–12% van die liggaamsgewig met die meerderheid gevalle so by 8–9%. Die bloedvolume kan dus in 'n pasiënt geskat word. Dertig persent bloedverlies gedurende operasie kan selfs in 'n gesonde hond die dood veroorsaak. Die bloed of ander vloeistof word teen 60–100 druppels per minuut ingetap. In geval van bloeding kan dit net so vinnig geskied as wat bloedverlies plaasvind. As algemene leidraad kan geneem word dat die bloed toegedien word teen 20 ml/Kg. liggaamsgewig en ander vloeistowwe teen 20–30 ml/Kg. Hierdie syfers is egter net 'n algemene gids.

Hierdie bespreking is toegespits op die hond, omdat daar veel meer werk op hierdie gebied oor dié diersoort as oor die ander huisdiere gedoen is. In breë beginsels geld dieselfde oorwegings vir ander diere. As voorbeeld van die laaste gedagte kan hier beskryf word die geval van 'n Jerseyvers met hydrops amnii. As gevolg van die geweldige hoeveelheid water in die uterus kon sy nie loop nie. Daar is besluit op 'n keiserhisterotomie. Die bloedverlies sou gering wees maar daar is verwag dat skok sou optree nadat die groot volume vloeistof verwyder is. Een liter fisiologiese soutoplossing bevattende 5 ml. Levophed is toe opgestel en in die V. jugularis ingeskakel. Toe die vloeistof met operasie uitgedreineer is, het met die vinnige val van druk in die buik die bloeddruk ook gedaal, die pols het verhoog in spoed en die snelheid van soutoplossing en Levophed toediening is ook verhoog. Die gevolg was dat, ten spyte van die feit dat daar net soveel vloeistof verwyder is as wat haar liggaamsgewig was (160 Kg. amniotiese vloeistof en ook liggaamsgewig), sy in so 'n goeie toestand na operasie was dat sy kon opstaan en onmiddellik begin eet het. In honde is dit ook nie vreemd om na operasie 'n geval te hê wat beter is as voor operasie nie.

Dit is dikwels vir die private praktisyn baie moeilik (en ook onekonomies) om 'n bloedbank te hou. Van die benodigdhede wat genoem is, is hierdie item die enigste wat 'n mate van moeilikheid oplewer. Met die verkryging van hondeplasma is daar minder moeilikhede. Die apparaat, wat maklik aangeskaf kan word, maak versameling van bloed so maklik (bv. in geval van genadedood) dat met min moeite die plasma 'n dag tot 2–3 weke na die tyd verwyder kan word en dan vir 'n onbepaalde tyd beskikbaar kan wees.

Hier is tot dusvêr geen melding gemaak van plasmasubstitute nie (afgesien van 6% gelatien) omdat daar 'n mate van bedenking bestaan oor hulle waarde, veral aangesien sommige die R–E stelsel blokkeer.

Bloed en elektrolietoplossing het grootliks tot die verbreding van die chirurgiese horison bygedra. In gevorderde chirurgie is die gebruik hiervan onontbeerlik.

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THE VALUE OF THE DETERMINATION OF TRANSAMINASE ACTIVITY IN PLASMA AS A SCREENING TEST FOR LIVER DISEASE IN ANIMALS

W. D. Malherbe*

INTRODUCTION

The foundations for an understanding of transaminating enzymatic mechanisms in the animal body were laid as long ago as 1937.[1] These consist of chemical reactions in which an amino group is transferred from one molecule to another without intermediate participation of ammonia.[6] Such enzyme systems were known to be widely distributed in the tissues of man and animals, and also in plants and micro-organisms. Wroblewski, La Due and co-workers[2], [3], [4], [7] were pioneers in 1954 and 1955 in assessing the clinical value of transaminase activity in human serum and commenced studies of the variations of this activity in health and disease. This led to the establishment of a valuable addition to the battery of tests available to the clinical pathologist.

The enzymes are found in the greatest concentration in certain body cells[3], [5], [7]: in the case of man in heart muscle, skeletal muscle, brain, liver and kidney in decreasing order. The clinical importance was found to arise from the fact that as a result of necrosis or degenerative processes in these cells the concentration of enzyme in the serum rose appreciably. From the point of view of diagnostic importance the glutamic oxaloacetic and glutamic pyruvic transaminases were found to be clinically useful. These involved chiefly the amino-acids alanine, aspartic acid and glutamic acid.

La Due and co-workers[7] in 1954 showed the marked increase of serum glutamic oxaloacetic transaminase (SGO–T), from 2 to 20 times normal, during the first 24 hours after myocardial infarction in man, the level returning to the normal range within three to six days. This transaminase was unaffected by angina pectoris, coronary insufficiency, heart failure or cardiac arrhythmias. It was therefore of great value where electrocardiographic results were equivocal. Manso *et al.* in 1956[8] recorded high values in various other conditions characterized by more or less extensive cellular damage, e.g. acute myocarditis, acute pancreatitis, haemolytic crises, extensive crush injuries, after surgery and large doses of aspirin. Normal levels however, were found in pericarditis, pulmonary infarction, rheumatic fever, rheumatoid arthritis and acute cholecystitis.

The Wroblewski–La Due group, after their finding in myocardial infarction, investigated[3] the concentration of SGO–T in liver disease

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such as carbon tetrachloride poisoning, viral hepatitis, cirrhosis, obstructive jaundice and carcinomatosis. This investigation was coupled with determinations of serum bilirubin, cephalin flocculation, thymol turbidity, albumin–globulin ratio, alkaline phosphatase, cholesterol and esters, bromsulphalein retention and prothrombin time. From these studies they concluded that SGO–T was impressively elevated as a result of acute liver cell injury due to tetrachloride poisoning and to viral hepatitis. Moreover this increased activity was found to be an index of liver cell *injury*, not at all necessarily correlated with the usual tests of liver dysfunction. On account of the very considerable functional reserve of the liver considerable destruction is required to disturb these "functions". This evidence of liver cell *injury* or *necrosis* was certainly significant and useful and has led to considerable interest in various parts of the world. In Denmark in 1958 Madsen *et al*[15]. reported similar results on a large series of patients suffering from various liver diseases.

In a further series of publications[11], [12], [13] Wroblewski and co-workers in 1956 and 1957 showed the value of serum glutamic *pyruvic* transaminase (SGP–T) as an even more sensitive index of liver cell damage than SGO–T. In myocardial infarction SGP–T levels were found to be effected only at exceedingly high levels of SGO–T. In man then the picture emerged that if SGO–T only was elevated involvement of the heart muscle was likely, whereas if both SGO–T and SGP–T were present in large amounts, particularly the latter, liver necrosis could be expected to be present. Confirmation of this work was provided by Pryse-Davies and Wilkinson[16] particularly in viral hepatitis.

In the veterinary field fundamental work was carried out by Cornelius and co-workers[21]. They studied tissue transaminases by a modification of the procedure of Tonhazy *et al.*[22] and for the estimation of transaminase activity in serum they used the methods of the Wroblewski group [23],[13] and of Reitman and Frankel[20]. Liver necrosis was induced in the various species by means of dosage of carbon tetrachloride in mineral oil.

SGO–T activity was increased following liver damage in horses, cattle, pigs and dogs, but in contrast SGP–T figures were affected *only in dogs*. This was a reflection of the fact that of these animals the liver of the dog only contained large amounts of GP–T.

MATERIALS AND METHODS.

For the determination of transaminase activity one is confronted with a variety of methods with different unit systems[9]. Fundamentally these procedures consist of incubating serum with substrates consisting of α-ketoglutaric acid and aspartic acid or alanine to determine SGO–T or SGP–T respectively. Techniques of different authors, however, differ chiefly in respect of incubation temperature and duration, and the method by which the rate of reaction is determined. This rate is a measure of enzyme activity. For this, paper chromatography, spectrophotometric determination of fall of extinction at fixed wavelengths,

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and chemical reactions of various kinds are used but there remains considerable doubt and confusion as to the comparative value of the units. Daly and Jordan[9] make a strong plea for the use of the King[10] unit which is defined as that activity in 100 ml. of serum which will convert 1 mol of amino-acid to keto-acid in one hour at 37°C. This activity is expressed in terms of the reactant formed and is measured colorimetrically. The units are of a convenient order as they avoid decimals or very large figures in the normal range.

In this laboratory the King method has been followed and found to be eminently suitable and feasible with not a great deal of equipment required. The units are roughly three times as high as the Sigma-Frankel[20] units (which are extensively used in the United States) but cost very much less to carry out. Mohun and Cook[14] devised a simple method, similar to King's, and gave comparative costs while also examining the resultant figures most critically. The difference in cost was found to be considerable and the results very satisfactory. In both these latter methods protein precipitation, and finally toluene extraction, are eliminated, thus reducing the number of steps leading to the desired end.

The material for the present study was derived from determinations of SGO–T on plasma from a number of horses, cattle, sheep and dogs considered to be clinically normal and from a number of these animals suffering from various disease conditions.

RESULTS.

The results of these determinations are presented graphically (fig. 1.)

(1) *Normal values.*—Although a different method was employed, the findings of Cornelius[21] in the cases of horses, cattle, sheep and dogs were qualitatively confirmed. From the graph the distribution of normal figures for the types of transminase can be seen. Although there is a measure of scatter, the majority of determinations fall within certain limits. A large number would be required for more exact limits but the trend is clearly indicated. This can be tabulated as follows:—

	SGO–T	*SGP–T*
Horses	250–320	15–50
Cattle	80–180	30–110
Sheep	100–140	10–50
Dogs	30–90	30–100

(2) *Liver Damage.*—This was assessed for the purpose of this paper by means of the sum of information derived from clinical examination, other laboratory tests and in some cases autopsies and histopathological examination.

In the case of horses liver cell necrosis was evidenced by considerably enhanced SGO–T activity whereas the SGP–T remained unaffected. This applied also to cattle and sheep, although in the latter animal in

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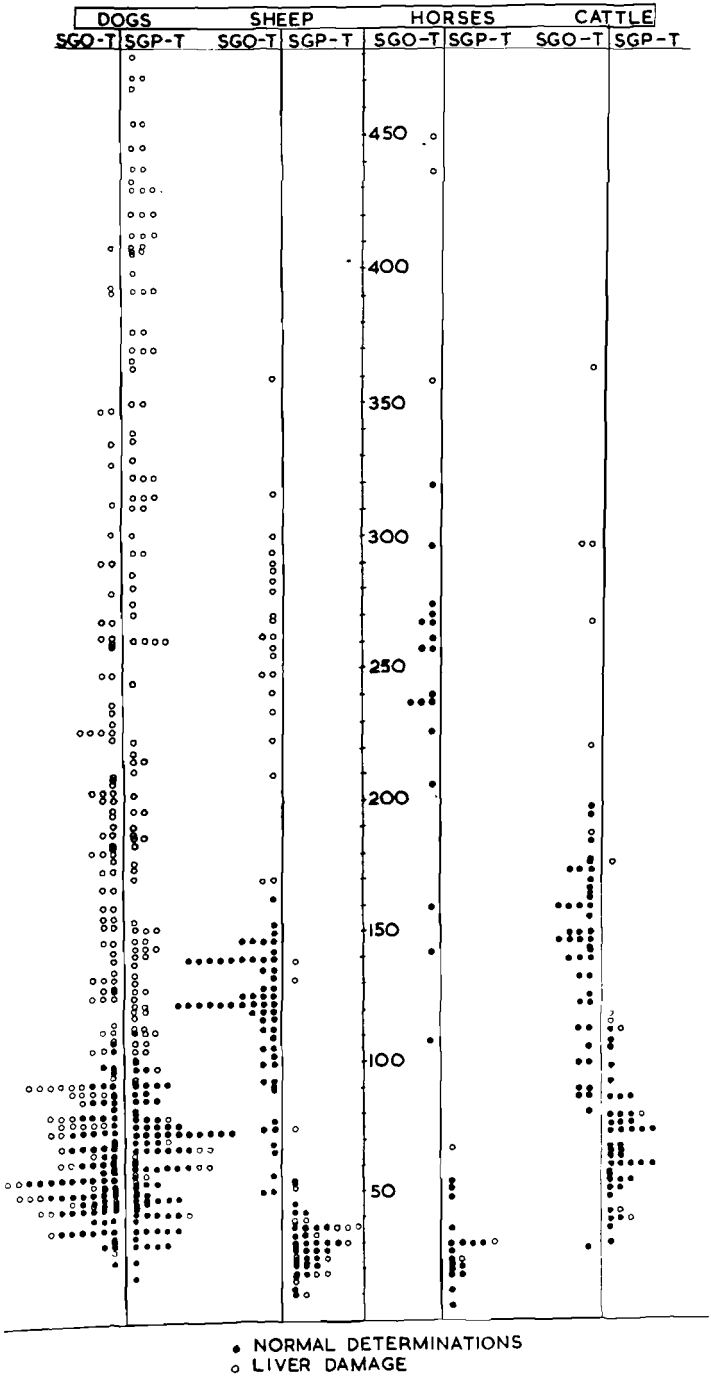


Fig. 1.

Transaminase activity values found in domestic animals between March 1959 and May 1960

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the few cases of liver damage encountered there seemed to be a mild degree of lability in the SGP–T figures.

Dogs in contrast showed much more sensitivity in the pyruvic transaminase fraction, although to a lesser extent the oxaloacetic was also affected. Of these animals studied the only one resembling the picture in human liver damage was the dog.

(3) *Differential diagnosis of virus disease in dogs*

About a dozen cases of distemper at various stages were studied and in no case was any elevation of transaminase activity determined. In three cases of infectious canine hepatitis (Rubarth's disease) markedly elevated values were found at a stage when differential diagnosis had been found impossible. How regularly this occurs in Rubarth's could not be assessed from the small number of natural cases available during the period of this study. A likely conclusion seemed to be that in cases of doubt between these two diseases a raised SGP–T would be strong evidence in support of infectious hepatitis.

In order to follow the findings in the course of disease, albeit on a small scale, four dogs received injections of hepatitis virus, two (12249 and 12252) intraocularly and two (12253 and 12254) subcutaneously. Of the four only one (12254) did not show any reaction clinically or with laboratory tests. The other one injected by the subcutaneous route reacted abortively. Both the intraocular cases reacted severely, one (12252) dying in under a week and the other (12249) recovering after an extended and severe illness.

A battery of liver function tests and haematological examinations were carried out at intervals of a few days. The results are presented graphically in figs. 2 to 4 for 12253, 12252, and 12249 respectively for the reacting dogs. Haematological figures were not affected.

The determination of transaminase activity in this small series showed itself to be a better index of liver cell damage than the older socalled liver function tests. This was particularly noticeable in the abortive case where both alkaline phosphatase and bromsulphalein retention were completely unaffected. Bilirubin was present only on one day. According to Weiss[26] this dog was subsequently proven to be immune.

(4) *Protozoal diseases*

The figures in cases of *Babesia equi* (horses), *B. bigemina* and *bovis* (cattle), *Anaplasma marginale* (cattle) and *B. canis* (dogs) conformed to a pattern of more or less normal figures in early or mild cases with marked rises in severe, well developed, or neglected cases. The majority of high values in the graph (fig 1) was derived from these diseases.

(5) *Other liver diseases*

Two cases of intrahepatic cholestasis in dogs one of which has been described in some detail[25] gave values of around 400 for pyruvic transaminase. In a single case of hepatic carcinoma it was as high as

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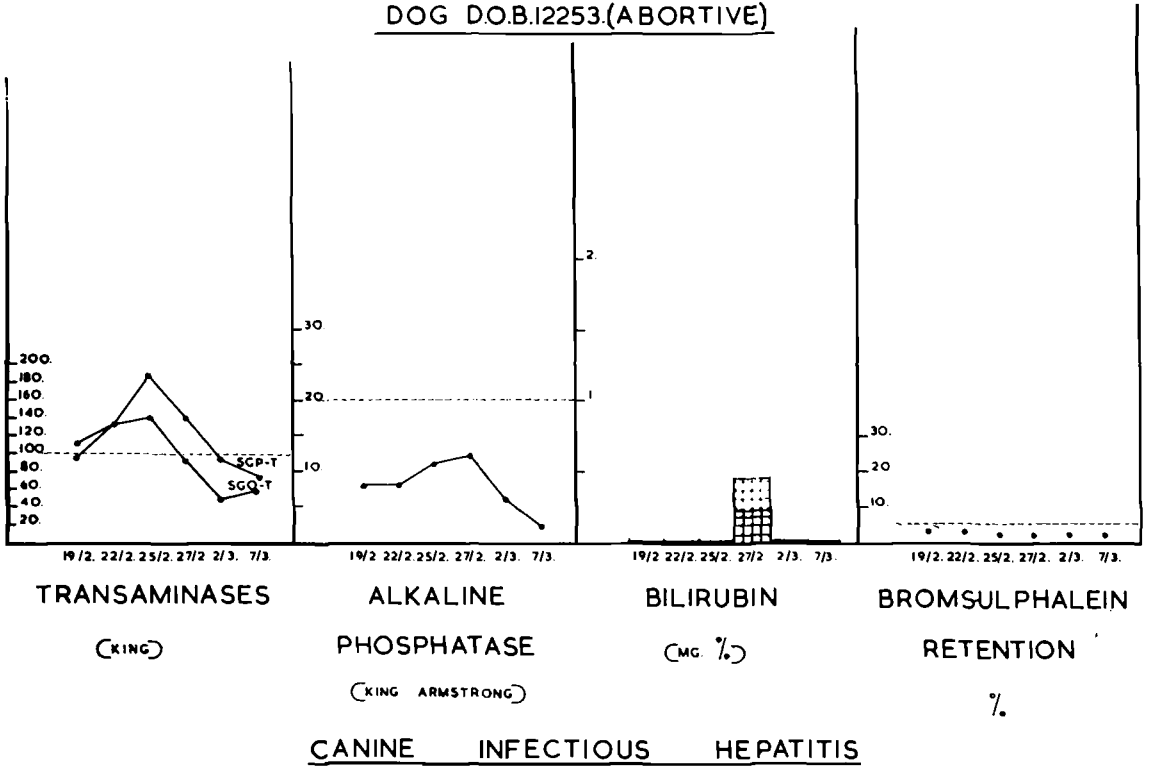


Fig. 2
Comparative liver "function" findings in an abortive infection with canine infectious hepatitis.

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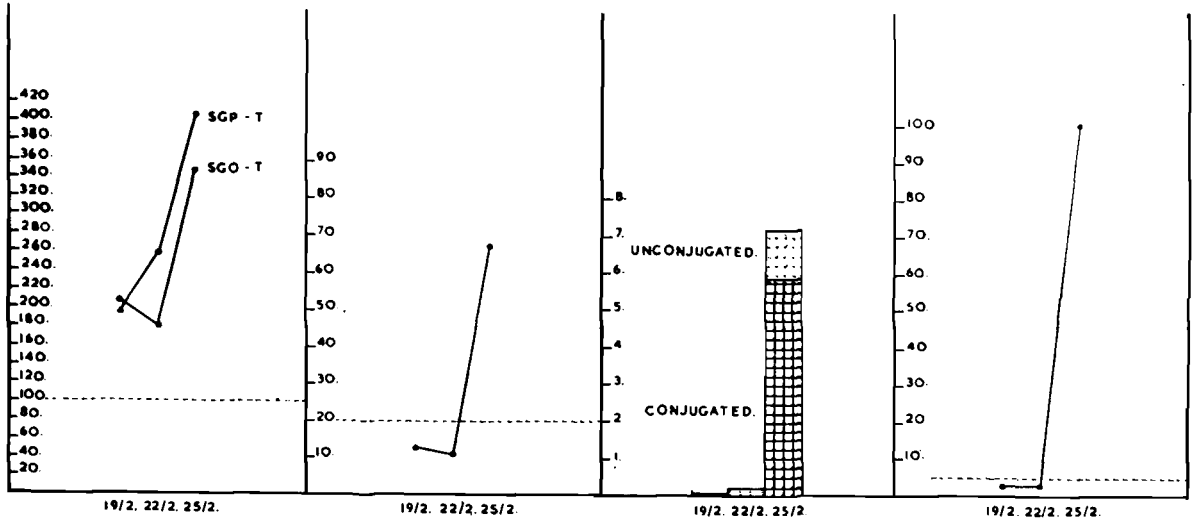


Fig. 3
Comparative liver "function" findings in a fatal infection with canine infectious hepatitis.

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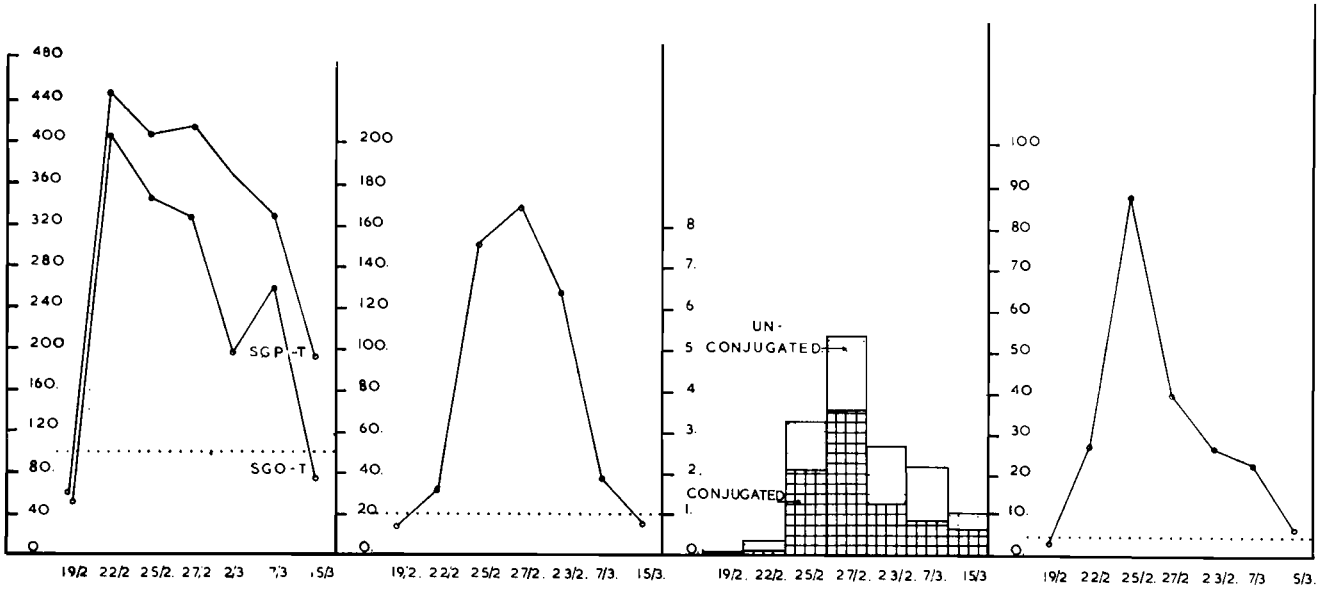


Fig. 4

Comparative liver "function" findings in a severe infection of canine hepatitis followed by recovery.

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445. Enhanced values were also found in a case of purulent hepatitis, one of cirrhosis and three of suspected (but unconfirmed) cases of leptospirosis (*L. icterohaemorrhagiae*). The high values of SGO–T in sheep were obtained from cases of bacterial icterus encountered in the course of field investigation by Adelaar and Tustin of this institute and from cases produced by them. Their work is published elsewhere in this Journal[28].

Liver function was studied in all these cases. Transaminase activity (in the case of sheep the oxaloacetic transaminase only) proved to be a sensitive indicator of the incidence of liver damage. By way of illustration two sets of findings are reproduced, fig. 5 illustrating the course of the disease from date of injection of the causal agent to the date of death, and fig. 6 showing findings during the disease. Incidentally, blood urea nitrogen was affected much later in the course of the disease than was the liver, indicating late kidney involvement. Serum alkaline phosphatase remained within normal limits in all affected cases and the flocculation and turbidity tests did not show any significant rise during the short course of the disease.

DISCUSSION.

The place of the determination of transaminase activity in the whole scheme of "liver function" has been commented upon editorially in *The Lancet*[17], [18]. It cannot be regarded as a function test but "estimations are likely to prove important additions in most biochemical laboratories" in that actual cellular damage can thereby be detected regardless of the degree of decompensation as a result of damage. Moreover the results are, unlike those of the bromsulphalein test, valid in the presence of jaundice. The levels tend to be higher in jaundice resulting from hepatocellular disease than in those resulting from extrahepatic obstruction[18]. As alkaline phosphatase behaves in the opposite manner, Latner and Smith[19] have suggested a transaminase/phosphatase ratio as being useful in the differential diagnosis of jaundice.

For the detection of early liver damage and in the differential diagnosis of diseases in which the presence or absence of liver cell necrosis constitutes important evidence one way or the other—regardless of the degree of *functional impairment*—the determination of transaminase activity becomes a most useful procedure. In the present study, with a limited number of cases of infectious hepatitis of dogs available, there was reason to believe that in circumstances of equivocal symptomatology they could be differentiated readily from cases of distemper. In a number of undoubted distemper and "paradistemper" cases, transaminase activity was of a normal order whereas high values for both oxaloacetic and pyruvic transaminases were obtained in cases that subsequently gave clinical or other confirmation of a diagnosis of infectious hepatitis, and in cases produced experimentally.

When this study was well advanced the author's attention was drawn to an exhaustive study[24] by a group of Italian workers on viral hepatitis in a closed community in Rome, one consisting of 104

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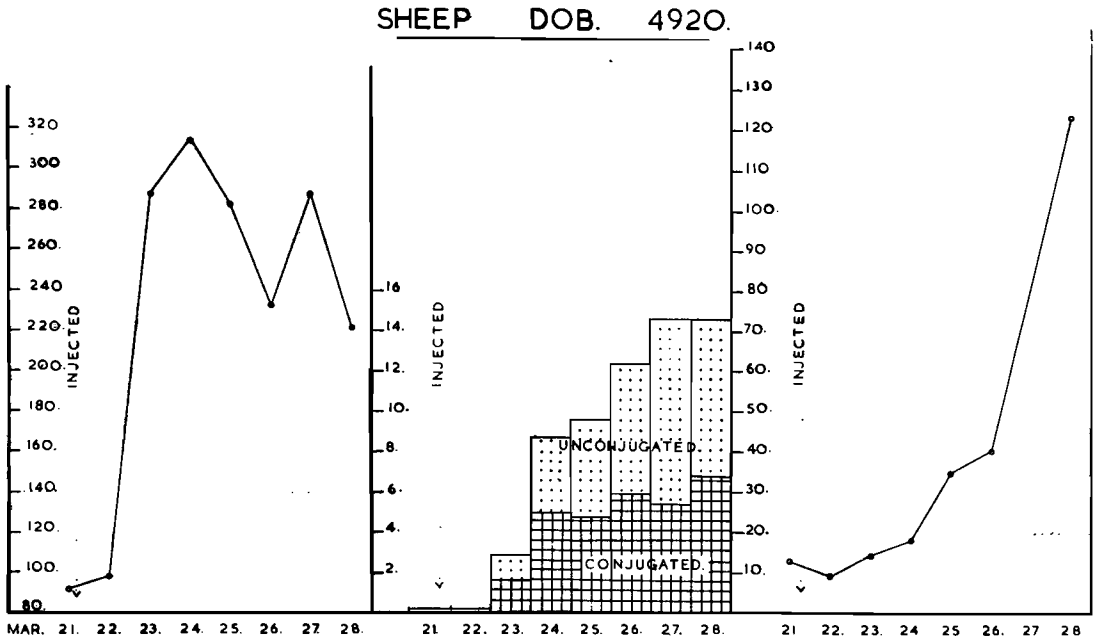


Fig. 5

Transaminase, bile pigment and urea nitrogen levels in bacterial icterus from infection to death.

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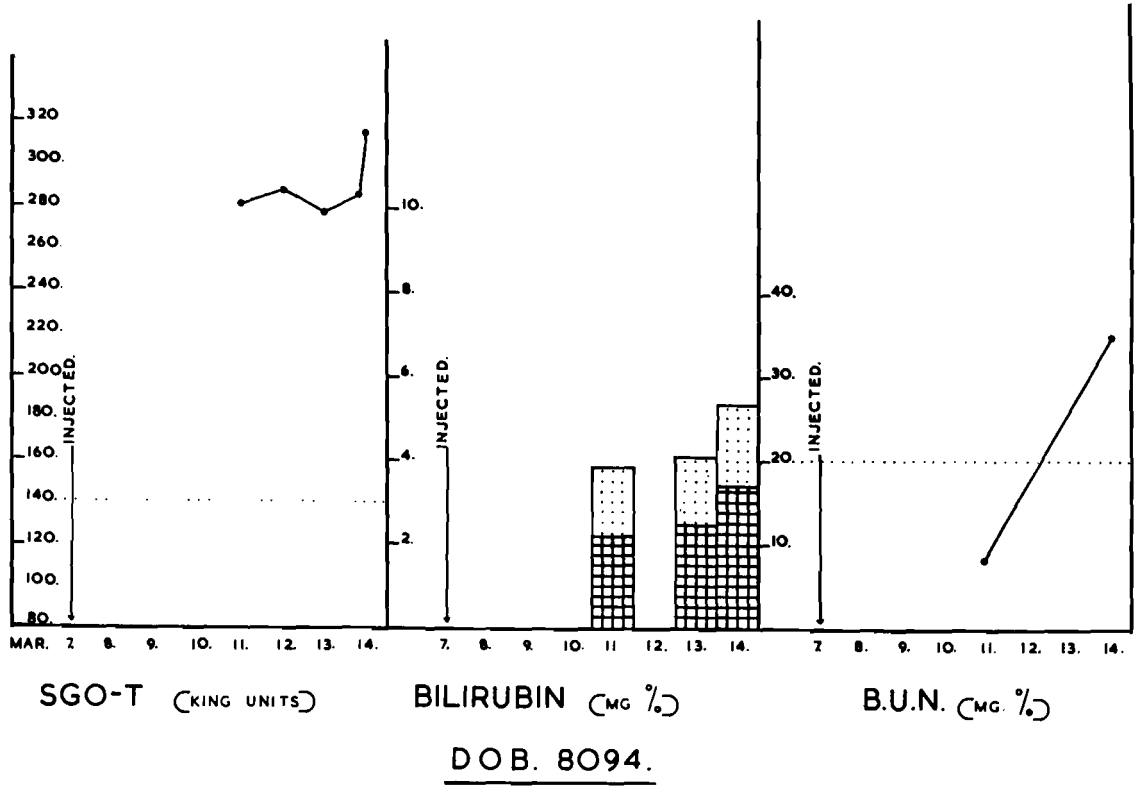


Fig. 6
Transaminase, bile pigment and urea nitrogen levels in the later stages of infection.

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children and 11 adults in an institution for children. They found that only with the determination of transaminase activity did "it become possible to assess with any degree of accuracy the real frequency of the anicteric forms" as against more easily diagnosed cases manifesting overt jaundice. Their criteria for confirmatory diagnosis of anicteric cases were entirely satisfactory. The incidence of non-jaundiced hepatitis in children predominated in a ratio of about 30:1 while in adults the jaundiced cases are known to occur relatively more frequently.

In an even more recent paper Bang and co-workers[27] in the U.S.A. demonstrated the value of determination of SGO–T in screening for latent hepatitis among prospective blood donors, i.e. for both carriers and subclinical cases of viral hepatitis. They concluded that this procedure would be more useful than most or all of the tests currently used for donor screening and that homologous serum hepatitis after transfusion could thus be significantly reduced.

From the data presented in the present paper it is clear that also in veterinary medicine the value of this test has been demonstrated. It is reasonable to anticipate that it will take an important place in the future assessment of liver damage (even of a subclinical nature) in studies of liver toxicity as a result of drug administration or of the effects of various diseases on animals. In diagnosis, too, it is likely to prove of considerable value as has been shown in the differentiation between distemper and Rubarth's infectious canine hepatitis.

SUMMARY.

A resumé is given of the development in recent years of the clinicopathological use of determination of transaminase activity for the assessment of early liver cell damage. The application of this procedure in the large and small animal clinics at Onderstepoort is illustrated, and its value in the differential diagnosis between distemper and Rubarth's infectious canine hepatitis shown. In the investigation of a newly-found disease entity, bacterial icterus of sheep, it proved its value for diagnosis and for following the progress of liver damage during the course of the disease.

As an index of liver cell damage or necrosis it provides a valuable addition to the hitherto used liver "function" tests in that it gives reliable information when functional impairment of the liver is not yet or not at all demonstrable.

It has been proved to be as useful in veterinary as it is in human medicine.

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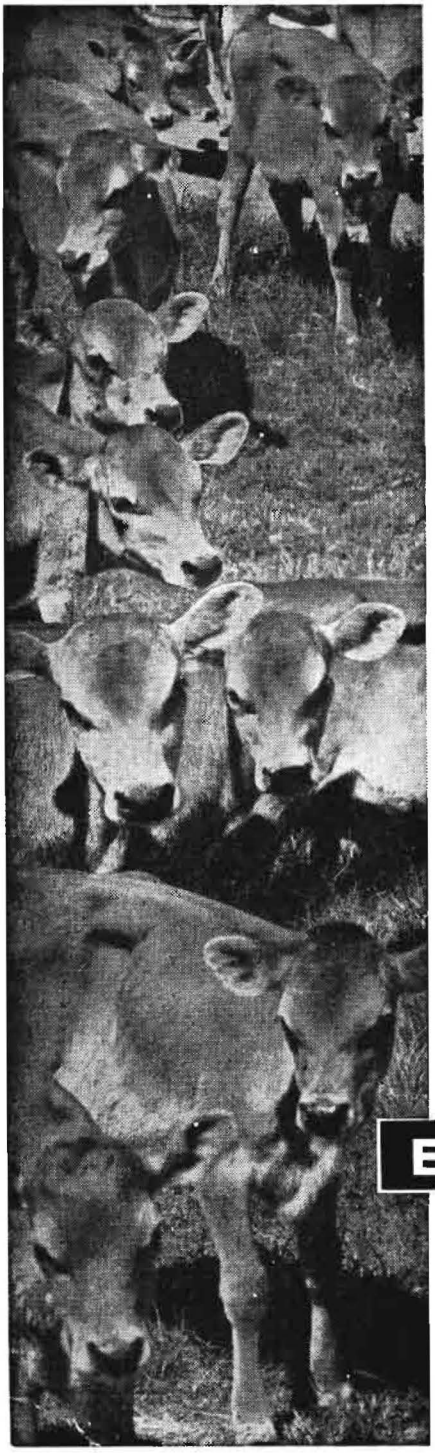
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Unieke nuwe Pfizer-inspuiting bestry onvrugbaarheid by beeste ... verhoog mate van bevrugting

EMBRYOSTAT *

Die mate van bevrugting van moeilike telers en repeteertelers word grootliks verbeter as hulle in die skede of in die baarmoeder ingespuit word met Embryostat—'n unieke formule wat deur Pfizer vir die eerste maal beskikbaar gemaak is en deeglik getoets is oor die afgelope 3 jaar.
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Embryostat inspuitings het 80% bevrugting gelewer by diere wat voorheen nie wou bevrug raak met minder as drie dekkings per dier nie. 83% bevrugting by 280 repeteertelers.
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BOOK REVIEWS

BRITISH VETERINARY CODEX 1953, *Supplement* 1959

The Pharmaceutical Press, London, pp. 134. Publ. price in U.K. 35/-

With the publication in 1953 of the British Veterinary Codex the long existing need for a really authoritative work on all important aspects of pharmaceutical and biological products was met for the first time. Its reception by veterinary, pharmaceutical, and allied professions throughout the English speaking (and probably English reading) world was most gratifying.

From the very nature of such a Codex, its essential dynamic, and also as a result of the publication of new editions of the British Pharmaceutical Codex in 1958 and 1959, a new edition of the B. Vet. C. became necessary. In order however to make the new information available as soon as possible, the needs of users of the Codex, it was decided, would best be served by first publishing a supplement to the first edition, while the work of revision continued.

This compilation resulted from the fact that improvements in methods of standardization and formulation had indicated that certain standards, assay procedures and formulae required amendment, and that the number of new medicaments which had become of importance in veterinary medicine since the publication of the Codex had made it desirable that standards and authoritative information on their actions and uses should be published as soon as possible.

Apart from a considerable number of amendments to information on substances previously treated in the B. Vet. C. this volume contains monographs on a surprising number of drugs that have come into use in veterinary medicine since 1953. These include, to mention but a few taken at random, the newer antibiotics and corticoids, antiseptics, insecticides, vermicides, and drugs acting on the nervous and blood vascular systems. A shorter section gives information on new vaccines and antisera while the monographs on new formulations of drugs comprise a quite considerable portion of the new material in the book.

As in the B. Vet. C. one of the appendices at the end of the book gives a useful list of synonyms for drugs better known to many users under their trade names than under their "generic" or Codex titles.

The acquisition of this Supplement is a "must" for all users of the B. Vet. C. who wish to remain up to date in their knowledge and use of drugs and biological substances available today.

W. D. M.

CLINICAL VETERINARY SURGERY—Vol. II. Operative Procedure by A. C. Shuttleworth M. V.Sc. F.R.C.V.S., Senior Lecturer in Veterinary Surgery at Liverpool University and Examiner in Surgery to the Royal College of Veterinary Surgeons, and R. H. Smythe, M.R.C.V.S. Examiner in Surgery to the Royal College of Veterinary Surgeons. Fig. 214. Pp. viii—398. 1st Ed. 1960. Crosby Lockwood & Son, Ltd. London. Price 42/-.

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This book is the companion volume to one reviewed fairly recently. The contents are divided into chapters on restraint, surgical instruments, materials and appliances, surgical procedure, anaesthesia, and from p. 118 onwards on operative technique of operations on the various systems and parts of the body.

The first impression is that the scope of this book is too wide—a failing of many veterinary text books. The result is that there is a tendency to deal too superficially with various important chapters, e.g. the very important subject of anaesthesia covers a scant 17 pages. As authoritative works on veterinary anaesthesiology are readily obtainable this chapter could have been omitted.

The chapter on surgical procedure is silent on my important points concerning autoclaving, draping, arranging of sterile bundles, and operating room routine.

As operative apparel, inter alia, a rubber apron is advised after soaking for a few hours prior to use in an antiseptic fluid. How much simpler is the proper thing, an autoclaved surgical gown, worn over drill shirt and trousers! It is further stated that sterile masks are rarely worn—but no unnecessary talking, sneezing or coughing is to be done above the patient. Similarly a sterile cap is unnecessary—if the hair is kept short! This proves that both are necessary!!

Surgical gloves are apparently not particularly highly regarded for routine use—yet what practitioner has not had the experience of having infected surgical wounds, despite stringent aseptic precautions, if he operates gloveless within a few days of handling septic material?

All of the descriptions of operations are not of the same standard—some are good and informative, others refer to techniques long outdated. As an illustration of the latter oöphorectomy of the bitch may be mentioned.

Certain very interesting surgical developments are not referred to. This may be explained by the fact that the references indicate that the literature consulted was from one journal mainly (admittedly a very good one) a few others in the U.K., a few in North America with no reference to work done in Europe and elsewhere.

The surgeon with some experience and discrimination will find this book useful as he will recognise and avoid certain weaknesses, while benefiting from those parts authoritatively presented.

C. F. B. H.

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CORRIGENDUM

In the article by Hofmeyr: "Investigations into the Toxicity of Carbocaine (Bofors)—a New Local Anaesthetic", this Journal, Vol. 30 No. 4, p. 459, the heading to Table I shoud read: "2% Carbocaine" not "20% Carbocaine".

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NAME OF ANIMAL F. VETALENTA 2ND

DATE	REMARKS
7·10·52	Calved (Heifer calf)
1·1·53	Served (B. Flash Prince)
9·10·53	Calved (Bull calf)
27·12·53	Served (B. Flash Prince)
17·1·54	" " " "
12·2·54	" " " "
	Injected 'Lutormone' — 1500 I.U. I/V.
28·2 54	Injected 'Protormone' — 10 c.c. I/m.
19·11·54	Calved (Bull calf)



Maintenance of pregnancy

Mild luteal deficiency in the cow results in abortion or resorption of the fœtus. Until recently the common method of correcting this condition was to inject 'Lutormone' brand Luteinising Hormone at the time of service and then to give further doses until the cow was at least 12 weeks in calf.

Workers at The Wellcome Veterinary Research Station, Frant, have now demonstrated a more reliable method. This is the administration of 'Lutormone' at the time of service followed 14-16 days later by one injection of progesterone ('Protormone').

'Protormone' is also of value in marked luteal deficiency, for example persistent ovarian cysts. In cases where two doses of 'Lutormone' have failed to correct the condition a response may often be elicited with 'Protormone'.

'Protormone' is issued in 20 c.c. rubber-capped multi-dose containers, containing 10 mg progesterone per c.c.

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ADVANCES IN "GEELDIKKOP" (*TRIBULOSIS OVIS*) RESEARCH.

4. The Pathology of Geeldikkop—Part I.

J. M. M. Brown, J. M. W. le Roux and R. C. Tustin.*

There is probably no other disease or group of diseases occurring amongst the domestic animals in South Africa, which has given rise to as much confusion regarding its pathology, as has the ovine icterus complex of diseases. Since Theiler's pioneer work on Geeldikkop published in 1918[1], in which the pathology of numerous cases encountered by him was discussed, very little indeed has appeared in print regarding this important aspect of the disease. What other descriptions there are, are meagre and limited to small paragraphs or isolated sentences, to be found in a few of the large number of papers on the disease published since Theiler's initial investigations.

This, on first thoughts, is rather surprising, since the views expressed regarding the pathology of Geeldikkop in nearly all of these odd references are at variance with some of the points made in Theiler's paper. However, this apparent reluctance on the part of later workers to publish more details on the pathology of the disease, and to clarify some of the statements made, becomes quite understandable when one considers the confusion which must have arisen during the twelve years or so following Theiler's investigations. During this period papers appeared describing, apparently for the first time, conditions such as Enzootic Icterus, "Dikoor" in lambs, Bacterial Icterus and "Swelled Head" in rams. Typical of these diseases was an icterus of varying severity or swelling of the head and ears, suggestive of a photosensitization disease. Very little was then known of their aetiology or the basic processes involved in the genesis of the icterus or photosensitization. It was mainly due to the later work of Quin, Rimington and Steyn (reviewed in the first paper of this series[2], that much of this confusion was eliminated, and certain of these diseases recognised as clinical entities distinct from true Geeldikkop (Tribulosis). However, Enzootic Icterus still remained an enigma and must, as it does today, have considerably complicated the studies made on the pathology of true Geeldikkop.

During the past three years the use of the mobile laboratory, described in the second paper of this series[3], has enabled us to undertake two very extensive field investigations into Geeldikkop in the affected areas. During these investigations a thorough study was made of the epizootology, symptomatology, pathology and chemical pathology of the disease as well as chemical studies on *Tribulus terrestris*.

* The field observations, post-mortem examinations, haematology and chemical Pathology were done by J.M.M.B. (Department of Physiology, Onderstepoort), the histopathology of cases of the disease from the Vosburg area was done by J.M.W. le R. (Department of Anatomy, Onderstepoort) and the histopathology of cases from the Victoria West area by R.C.T. (Department of Pathology, Onderstepoort).

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The epizootology has been discussed in the preceding paper of this series[4]. This present paper will deal with the gross and microscopic pathology of natural cases of the disease, and the other studies will be presented in appropriate succeeding papers.

The first of these investigations was conducted in the late summer of 1958 in the Vosburg district, during an outbreak of Geeldikkop, which, although severe, was restricted mainly to this area. The cases seen were considered typical of true Geeldikkop and represented all stages of the disease. They will be referred to in the following discussion as *the Vosburg cases.*

The succeeding investigation was conducted in the districts of Victoria West, Loxton, Vosburg, Hofmeyr and Teviot in the late summer of 1959, during one of the worst and most widespread outbreaks of the disease for many years. Once more, cases in all stages of the disease were studied, and although most of them were clinically Geeldikkop cases and their chemical pathology characteristic of this disease, it was found, in some instances, that both the gross and microscopic pathology showed certain features hitherto regarded as germane to Enzootic Icterus only. On one of the farms visited an outbreak of typical Enzootic Icterus changed in a dramatic manner to an outbreak of typical Geeldikkop within the space of about one week. This has been discussed in the preceding paper[4]. On other farms cases of both conditions were encountered simultaneously in the same flocks. As will be seen from later papers in this series, it is not always easy to distinguish clinically between the two conditions, but a critical study of the chemical pathology of such cases makes the distinction much easier.

For the purposes of this discussion Geeldikkop will be considered as a separate entity, notwithstanding our present belief that the two conditions are very closely related as regards their aetiology. The pathology of the typical Enzootic Icterus syndrome will be presented in one of the succeeding papers. In the discussion which follows, cases described as typical of Geeldikkop are those which show the characteristic symptomatology of the disease, and in which the chemical and pathological findings conform to those which we have established as typifying the condition.

The cases examined during the second investigation in the Victoria West and other districts mentioned, will all be referred to as *the Victoria West* cases in the text which follows.

Materials and Methods

All the observations in this particular aspect of our studies on the disease were made on freshly slaughtered animals sacrificed by the owners for this work, and all material used for the preparation of sections for the histopathological studies was collected immediately thereafter and placed at once in the appropriate fixative fluids. Sections from such material were then made as soon as possible on the return of the team to Onderstepoort, and stained by the usual routine methods. Erythrocyte, leukocyte, eosinophile and differential white cell counts were performed using standard methods in routine use.

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Observations

The Vosburg Cases

During the course of our studies on the chemical pathology of the disease, it was considered necessary to compile a set of figures of normal values for all the estimations and tests performed, obtained from clinically normal sheep in each affected area. Accordingly blood samples were collected from sheep which on clinical examination appeared to be perfectly healthy, as well as from obvious cases of the disease. This proved to be most fortunate, since, on completion of all the biochemical work, it was found that many of these apparently normal sheep showed certain very marked disturbances, and could, in fact, be classified as preclinical cases of the disease. This was particularly true of the sheep from one farm; specimens from these were collected at a time when fresh cases of the disease were appearing in large numbers each day amongst the flock. In the discussion which follows these animals will be referred to as *preclinical cases*. Since these animals were selected as being apparently normal in the first instance, all work done on them was limited to the haematology and various chemical estimations performed on the blood specimens. Similarly, the only available information on the cases classified later in the text as *early cases*, is again confined to the symptomatology, haematology and chemical pathology, since there was an understandable reluctance on the part of most farmers to sacrifice any animals which may possibly have recovered.

A. *Preclinical Cases*:—

Regarding the haematology of these cases, the most striking disturbances observed were: (*a*) an extremely severe leukopaenia; so severe in some of the animals, that repeated careful examinations of stained blood films from them failed to reveal the presence of any leukocytes at all; (*b*) in all cases there was a very marked "shift to the left" in the neutrophiles. Owing to the very marked immaturity of these cells, it was impossible to perform a reliable differential white cell count in every case; (*c*) there appeared to be in all cases a marked lymphocytopaenia; (*d*) eosinophile counts in general were low, figures ranging from 50–150 per cu. mm. being obtained (figures obtained from normal healthy sheep at Onderstepoort range from 200 to 500 per cu. mm.[5]; (*e*) there appeared to be an extremely severe thrombocytopaenia in most cases. This was suspected when careful examination of stained blood films from these animals showed an almost total absence of thrombocytes, which are usually very numerous in such preparations from healthy sheep. Repeated attempts were made to perform thrombocyte counts by established methods, but these proved unsatisfactory under field conditions, and no reliable figures are at present available to substantiate this important finding; (*f*) All cases showed some degree of anaemia, being reflected cheifly by erythrocyte anisocytosis, varying from mild to very pronounced, the presence of numerous erythrocytes containing Jolly bodies, and in some cases numerous normoblasts as well; (*g*) in two of the

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most severe leukopaenic animals numerous bacilloid and coccoid organisms were encountered in the stained blood films.

Four apparently normal sheep from another flock, in which no fresh cases of the disease had appeared for two weeks prior to our visit, showed the following interesting disturbances:—(*a*) a definite but by no means severe leukopaenia; (*b*) a very definite lymphocytopaenia; (*c*) a very marked neutrophile "shift to the left"; (*d*) no evidence of thrombocytopaenia; (*e*) slightly low or normal eosinophile counts, figures of 150–290 per cu. mm. being obtained; (*f*) the presence of numerous normoblasts in stained blood films. It is most difficult to decide what state of the disease is represented by the changes seen in these four animals, especially in view of the fact that no fresh cases of the disease were apparent in the flock. They could have been preclinical, abortive or recovering cases of the disease.

B. *Early Cases*:—

These were cases which were examined within four days after the onset of clinical symptoms. All of them showed an icterus varying from moderate to severe, as well as very severe lesions of photosensitization on the exposed parts of the body. The salient haematological findings in these animals were the following:—(*a*) leukopaenia varying from mild to severe: (*b*) a distinct lymphocytopaenia; (*c*) a marked neutrophile "shift to the left", it being once more virtually impossible to perform reliable differential white cell counts: at least 60—80% of the leukocytes present were apparently immature neutrophile forms; (*d*) in a small percentage of these cases there appeared to be a thrombocytopaenia but the majority of cases showed no apparent decrease of these on examination of stained blood films; (*e*) eosinophile counts were extremely low throughout, figures of 0–130 per cu. mm. being obtained, with an overall average of 30 per cu. mm.; (*f*) the majority of cases showed no anaemic changes. In a few animals mild to moderate anisocytosis with a fair number of erythrocytes containing Jolly bodies was observed.

C. *Advanced Cases*:—

These were cases of at least one week's standing. Most of them were *in extremis* when encountered, showing a slight to severe icterus and photosensitization lesions, varying from healing lesions to severe sloughing necrotic areas on the exposed parts. *Haematology.*—The following were the most significant findings:—(*a*) no anaemic changes; (*b*) no leukopaenia; (*c*) a marked neutrophile "shift to the left", at least 60—80% of the leukocytes present being immature neutrophiles; (*d*) a definite lymphocytopaenia; (*e*) no apparent decrease in thrombocytes; (*f*) very low eosinophile counts, figures of 0—10 per cu. mm. being the usual figures encountered.

Gross Pathology.—In general the following may be seen on autopsy of cases of at least one week's standing:—(*a*) photosensitization

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lesions varying from oedema and erythema of exposed portions of the skin, e.g. the face, eyelids, lips, nostrils, ears, axillae, groins, coronets and genitalia, to acute dermatitis and severe necrosis with extensive sloughing of the affected skin of these structures. The eyes are invariably involved secondary to the lesions of the eyelids and conjunctivitis, keratitis, and even a purulent panophthalmia with rupture of the cornea may be encountered; (*b*) depending on the stage of the disease and the severity of the various lesions, the condition of the animal may be fair or more generally markedly emaciated, and often severely dehydrated; (c) icterus varies from moderate to intense; (*d*) there may or may not be a mild hydropericardium, hydrothorax or ascites; (*e*) the liver is usually enlarged and noticeably stained with bile pigment, the colour varying from dark brown to orange brown in severe cases. Its consistence is often reduced, The gallbladder will contain, depending on the stage and severity of the disease, from 10 to 50 ml. of bile, which varies in nature from very pale greenish or almost colourless, to a very dark green thick tarry fluid. In early cases, where there is a marked paralysis of the gastro-intestinal tract, the gallbladder may be distended with very light green bile; (*f*) the kidneys are usually pigmented, the cortex appearing dark brown in colour. In other respects the kidneys usually appear normal. This is extremely interesting since both the biochemical and histopathological examinations of these animals reveal renal lesions of marked severity. Furthermore it is important to note that, while in these typical cases of Geeldikkop the kidneys appear macroscopically to show only some degree of pigmentation, the kidneys of a typical case of Enzootic Icterus are grossly abnormal. In the latter condition they are extremely enlarged, the cortex has a deep "gunmetal" bluish appearance which, in severe cases, appears almost black or brownish black, with numerous pinpoint haemorrhagic areas often scattered throughout the cortical tissue; (*g*) there is usually a fair to marked atrophy of the spleen with a marked atrophy of the splenic lymphoid tissue; (*h*) there is a very marked atrophy of all the lymphoid tissue in the body, the lymph nodes being severely involved and often difficult to find; even the large mesenteric lymph glands are so affected. The parotid, submaxillary and prescapular lymph nodes, which drain the areas involved in the photosensitization reactions, are often enlarged and very oedematous; (*i*) the adrenals appear to be severely affected. They may show marked atrophy or marked enlargement, their colour varying from normal to light yellowish brown; and their structure on section is often very indistinct; (*j*) there is usually a severe atony of the entire gastro-intestinal tract, the contents of which is often extremely desiccated throughout, especially that of the rumen, omasum and caecum.

Histopathology.—The most constant change noticed was an extensive bile pigmentation of the liver cells. The pigment was scattered diffusely through the cells and it did not seem to have accumulated in any particular region of the lobules. The Kupffer

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cells also contained bile pigment. The liver cells appeared swollen and hyaline degeneration could be demonstrated in some of them. The cytoplasm showed a moderate degree of fatty infiltration. The bile ducts were empty. The bile canaliculi were prominent and, with the exception of one case, no bile could be demonstrated in them either. There was no evidence of any large scale necrosis of liver cells as described by Theiler[1]. A few isolated liver cells showed signs of necrobiosis, such cells appearing shrunken; their cytoplasm tended to be more eosinophilic with a tendency to pyknosis of their nuclei. The presence of interstitial connective tissue was noticed in a few cases but to such a moderate degree that it did not seem to bear any relation to the disease as such. The sinusoids contained some red blood cells and neutrophiles.

The kidneys showed signs of a marked nephrosis. The tubules were dilated and the tubular cells showed fatty infiltration hyaline degeneration and bile pigmentation. Bile pigment could be demonstrated in both the convoluted as well as the straight parts of the tubules. The pigmentation tended to be confined to focal groups of tubules. The tubular cells appeared swollen and were either packed with pigment granules or showed varying degrees of fatty infiltration of the cytoplasm together with hyaline droplets and pigment. In some cases desquamation of the affected epithelial cells took place into the lumina which also contained bile pigment.

No marked changes could be demonstrated in the other organs. There was some evidence of atrophy of the lymphoid tissue. The spleen showed a diminution of the cellular content of the Malphigian bodies and the cortical nodules of the lymph nodes appeared to be similarly affected. In these organs the reticulo-endothelial cells were prominent and showed phagocytosis of bile pigment. The lungs, myocardium and adrenals did not show any particular changes.

In general the changes noticed were to a great extent similar to those described by Theiler[1], especially with regard to the fatty changes and distribution of bile pigment. However, the constant necrosis of liver cells and the well marked subacute interstitial hepatitis with proliferation of the bile ducts described by him, were not constant findings in the present series, neither was there any evidence of bile stasis with atrophy of liver cells. In view of this one feels inclined to entertain the idea that in Theiler's cases there might have been other factors which complicated the picture.

In one case evidence of haemosiderosis was seen. This finding was much more prominent in cases examined during the 1959 outbreak of the disease in the Victoria West area.

D. *Long Standing Cases*:—

It is a well-known fact and one which we have observed frequently, that many cases of Geeldikkop make an apparent

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recovery, and then about three to four weeks after the initial onset of symptoms suddenly become critically ill again and usually die within a day or two. From the chemical pathology of such cases we have concluded that the relapse and death are directly due to failure of the adrenals. This intriguing aspect of the disease will be fully dealt with in a later paper in this series. One such case was studied in detail during this particular investigation. it was a case of at least three weeks standing, which showed some temporary improvement but then rapidly became emaciated, and was finally slaughtered *in extremis* for collection of specimens.

The following were the most significant findings from this animal:—

Haematology.—No anaemic changes were seen. There appeared to be a marked neutrophilia and the circulating neutrophiles were very immature and an approximate differential count only could be obtained, viz:. neutrophiles 80%, lymphocytes 15%, monocytes 5%. The eosinophile level in the blood was extremely low, a count of 0 per cu. mm. being obtained. The animal showed in addition a fair degree of thrombocytopaenia.

Pathology.—There was marked emaciation and extreme dehydration. The blood was extremely concentrated and very dark red in colour. The flesh appeared dry and had a peculiar pungent odour. The skin was dry, parchment-like and tore apart easily. There was virtually no subcutaneous tissue and the animal was in fact skinned by merely pulling the skin off the carcass. A very mild icterus was present. The liver was enlarged and severely pigmented, while the gallbladder was filled with about 400 ml. of dark, greenish-black, very tarry bile.

On microscopic examination of sections of the liver extensive and diffuse bile pigmentation of the parenchymal and Kupffer cells was seen. There was extensive centrolobular fatty infiltration and necrobiosis of a few isolated cells. The bile ducts and canaliculi were empty.

Atrophy of the spleen and all lymphatic tissue, confirmed microscopically, was present.

The kidneys were interesting. They were severley pigmented but the colour of the cortex was the "gunmetal" greyish-blue, seen so frequently in Enzootic Icterus. Histological examination revealed only bile pigmentation and fatty infiltration, with desquamation into the tubular lumina of pigment-containing epithelial cells. The chemical pathology showed only a very moderate urea nitrogen retention (34.96 mgm.%).

Macroscopically the adrenals appeared very small although their structure was quite distinct. On microscopical examination with the usual routine stains, nothing unusual could be demonstrated. However, the chemical pathology revealed marked electrolyte disturbances which, after a study of the complete chemical pathological picture, could be attributed only to adrenal hypofunction. Illustrative of such findings were the plasma electrolyte

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figures: sodium 122 m.eq. per litre, potassium 3.4 m.eq. per litre and chlorides 72.88 m.eq. per litre (normal values for sheep: sodium 144± 6 m.eq. per litre,[6] potassium 4.8 ± 0.7 m.eq. per litre[6] and chloride 100 ± 5 m.eq. per litre[7].

There was severe atony and atrophy of the entire gastro-intestinal tract. The rumen contents were absolutely dry and powdery. The omasum was about the size of a golf ball and its contents were quite desiccated. The small intestine was no greater in length or cross-section than the intestinal tract of an average sized fowl. The colon and caecum were also markedly reduced in size. As this sheep was full-grown these latter statements give a good indication of the degree of atrophy which had taken place in the gastro-intestinal tract.

THE VICTORIA WEST CASES

These cases illustrate very nicely the complex nature of the disease and the variable clinical, post mortem and histopathological pictures which it presents. In general, most of these cases do not conform to the typical clinical appearance of the disease since, although photosensitization was a prominent and severe symptom, icterus was very mild or negligible. When cases showing both severe icterus and photosensitization were encountered in this series, it was found that they showed in addition many features of Enzootic Icterus. The only cases encountered during the course of this investigation, which may be regarded as typical of Geeldikkop in nearly all respects, were the cases examined in the Hofmeyr and Teviot areas. Since these particular cases were in most respects similar to the typical Geeldikkop cases just described under the Vosburg cases, they need not be considered further. The following then were the most significant features seen in the cases from the Victoria West and surrounding areas:

A. *Early cases*:

These were again cases examined within four days of the onset of clinical symptoms. Ten animals were studied in this group, all of which showed photosentsitization lesions in the form of mild to severe oedema of the ears, lips, eyelids and dewlap, with sometimes superficial sloughing of necrotic skin on the affected parts. Coronitis was seen in one of the cases. Icterus varied from very mild in a few cases, to negligible in the majority of cases.

Haematology.—In general signs of anaemia were absent; haematocrits varied from 41—49%, red cell counts from 10.03—12.50 $\times 10^6$ per cu.mm., and haemoglobin values ranged from 9.65—11.82 gm.%. In two cases a very mild anisocytosis was present. Total leukocyte counts were within normal limits, varying from 3.9—10.65 $\times 10^3$ per cu.mm., but in all cases there was a marked neutrophilia, the differential count for neutrophiles ranging from 70—91% with, in most cases, a very marked immaturity of the neutrophiles. In all cases a marked lymphocyto-

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paenia was present. Eosinophile counts were low in nine cases (0—60 per cu.mm.) and normal in one case (360 per cu.mm.). In only three cases was an apparent moderate thrombocytopaenia encountered.

Gross Pathology.—Two of these cases were sacrificed for the collection of material for the histopathology. Both animals were in good condition. Icterus was mild in one and absent in the other. The livers of both were enlarged and dark brownish-orange in colour. The gall-bladders contained about 100 ml. of dark green, tarry bile. The kidneys of both were somewhat enlarged, but, apart from some bile pigmentation, showed nothing unusual. The adrenals showed obvious degenerative changes. There appeared to be a marked atrophy of all lymphoid tissue; the lymph nodes were very small and difficult to find with a concomitant marked decrease in splenic lymphoid tisse. In both cases there was a very severe gastro-intestinal stasis.

Histopathology.—The livers showed mild fatty changes, the bile ducts were empty and the canaliculi not prominent. Some of the parenchymal cells and Kupffer cells were loaded with bile pigment. In the kidneys mild to moderate nephrosis was observed, involving especially the proximal convoluted tubules, which also contained small granules of bile pigment and albuminous material in their lumina. The lymph nodes and spleen exhibited atrophy of the lymphoid nodules. In the lymph nodes the reticulo-endothelial cells were loaded with a finely granular iron-free pigment. In one of the cases the lymph nodes contained numerous macrophages loaded with an iron-free pigment, which resembled the "giant pigment cells" seen in cases of Enzootic Icterus. A fair amount of iron-containing pigment was present in the spleens of both cases. Nothing specific was to be seen in the adrenal glands.

B. *Advanced Cases:*

These were again cases of one to two weeks standing. Nine cases of this nature were studied. In most of then the severe oedematous swellings of the photosensitization reaction had subsided and sloughing of much of the affected skin had commenced. Oedematous swellings of the nose and lips could still be seen in a few cases. In three cases there was a mild icterus while in the others icterus was negligible. One case was suffering from a severe purulent panophthalmia and one showed signs of an acute coronitis.

Haematology.—In some of the cases there was a fair anaemic tendency. Haematocrits ranged from 22.5%—47.5%, red cell counts from 7.18—13.55 $\times 10^6$ per cu.mm., haemoglobin from 6.27—10.62 gm.% and some degree of anisocytosis was observed in stained blood films from four animals. Total leukocyte counts were in most cases normal or in a few cases in the low range of normal, varying from 3.55—7.8 $\times 10^3$ per cu.mm. In all cases there was a marked neutrophilia with a very pronounced "shift

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to the left" and a moderate lymphocytopaenia. In two of the animals eosinophile counts were normal, but in the remainder they were very low, varying from 0—140 per cu.mm.

Gross Pathology.—Two animals in this group were slaughtered for specimens. The condition of both was fair. Icterus was mild in one and absent in the other. A slight hydropericardium was present in both. The livers in both cases were enlarged and dark brownish in colour and the gall bladders contained from 100—200 ml. of dark green, tarry bile. The kidneys of both were slightly enlarged and in one case the cortex was dark brownish-grey in colour. The adrenal glands of both were light brownish-yellow in colour. In both cases the lymph nodes and spleen presented no unusual features, nor did the gastro-intestinal tracts of either.

Histopathology.—In both cases there was a fair amount of bile pigmentation of the liver parenchymal and Kupffer cells. Numerous large macrophages, loaded with an iron-free pigment, were present in and around Glisson's capsule. In addition, a mild interlobular cirrhosis with necrobiotic changes in a few scattered cells was present in the liver of one case. Mild to moderate nephrotic changes were present in the kidneys of both animals and the tubular cells contained both bile pigment and some iron-containing pigment. A fair amount of iron-containing pigment was present in the spleens of both cases. Slight atrophic changes were apparent in the lymph nodes of each animal, in addition to the presence of a fair number of large macrophages laden with non-iron containing pigment.

C. *Cases showing features of both Geeldikkop and Enzootic Icterus:*

Three cases of this nature were studied in detail. They were all cases which had been ill for at least a week and were *in extremis* when examined. All were severly icteric, the photosensitization oedema had subsided and there was a severe necrotic cheilitis and rhinitis. One case showed a severe purulent panophthalmia and another an acute coronitis.

Haematology.—Mild anaemic changes were observed in two of the cases,(haematocrits 30% and 38%, red cell counts 6.83 and 6.92 $\times 10^6$ per cu.mm., haemoglobin 8.93 and 7.24 gm.% and anisocytosis), but not in the third. Total leukocyte counts were within normal limits but in all cases there was a pronounced neutrophilia (80%, 83%, 90%), most of these cells being highly immature. A severe lymphocytopaenia was again present and eosinophile counts were very low in all three cases. (20, 40 and 40 per cu.mm.).

Gross Pathology.—All three animals were markedly emaciated and severely icteric. The livers of all three were enlarged, dark yellowish brown in colour and showed an increased consistence. The gallbladders all contained about 200 ml. of dark green, tarry bile. In all three cases the kidneys were extremely enlarged, their cortices being dark "gun metal" grey in colour with numerous

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reddish pinpoint areas scattered throughout the cortical tissue. In all cases the adrenal glands were very enlarged, light brownish-yellow in colour and their structure was very indistinct. A marked atrophy of all the lymph nodes and splenic lymphoid tissue could be seen in each animal. All three animals exhibited a complete and severe gastro-intestinal stasis as well as a very severe hypoplasia of the red bone marrow in all the long bones. The yellow marrow in these structures was intensely stained with bile pigment.

Histopathology.—Moderate fatty changes could be observed in all three livers with necrobiotic changes visible in a few scattered cells. There was a fair degree of bile pigmentation of the parenchymal and Kupffer cells, and a fair number of large pigment-containing macrophages were present in Glisson's capsule. No bile was present in the bile ducts or bile canuliculi. In all three cases moderate to severe nephrotic changes were present in the kidneys, some of their tubules being distorted in appearance, the epithelial cells of which showed hyalin degeneration or fatty changes with much granular debris present in the lumina. Granular debris present in the lumina. Granular iron-free pigment was present both in and outside the tubular cells. Some of the Bowman's capsules contained pigment casts and the glomeruli of these appeared atrophic. In some tubular cells a slight amount of iron-containing pigment was present.

A large amount of iron-containing pigment and bile pigment was present in all three spleens and all showed atrophy of the Malphigian corpuscles. The lymph nodes in all cases showed marked atrophic changes involving especially the cortex and lymph nodules. Both bile pigment and iron-containing pigment was found to a fair degree in these structures, and the lymph nodes of one animal contained in addition some very large macrophages loaded with iron-free pigment, resembling the "giant pigment cells" of Enzootic Icterus.

Discussion

Probably the first descriptions of the gross pathology of Geeldikkop to be published were those of Hutcheon[8],[9] and Dixon[10],[11]. Their work is reviewed by Theiler in his paper[1], which up to the present time was the most complete description of the pathology of the disease extant. Later brief accounts appear in the works of Henning[12] and Watt and Breyer-Brandwijk[13] based mainly on the details given in Theiler's paper. A later brief note is to be found in a book by Steyn[14]. Quin, in two of his papers on the disease,[15],[16], makes brief mention of the fact that the livers of natural cases of the disease show very little morphological change microscopically, and notes only bile staining, fine fat droplets and necrosis in some parenchymal cells.

The present studies confirm Quin's observations and many points made by Theiler. However, many new and important findings have appeared, which serve not only to explain various aspects of the disease and, together with the chemical pathology, the cause of the high

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mortality observed in many outbreaks of Geeldikkop, but also indicate the close relationship of Geeldikkop and Enzootic Icterus.

The cases examined in the Vosburg area may be considered as typical of the pathology of Geeldikkop. The anaemia seen in those animals was mentioned by Theiler[1] and again by Henning[12] in the earlier literature of the disease but no details of its nature were given. It is of considerable interest to note that these anaemic changes occur in the preclinical cases and some of the early cases, but were apparently not present in the advanced cases seen in this outbreak. Similarly thrombocytopaenia and leukopaenia are extremely severe in the preclinical cases, less so in the early cases, and absent in the more advanced cases of the disease. From this it could be deduced that, in the initial stages of the disease, there is a temporary disturbance or arrest of haemopoietic function involving the red bone marrow. On consideration of all the data available pertaining to these cases, it is felt that the anaemic changes are of a hypoplastic type, rather than any of the other forms of anaemia. It is an established fact that transient leukopaenias —mostly a reflection of neutropaenia—may be caused by temporary toxic inhibition of the bone marrow, due to bacterial or virus toxins, various drugs or other toxic compounds. The neutrophilia seen in these cases of Geeldikkop is probably of a relative nature, since the pronounced immaturity of the neutrophiles is accompanied by a marked lymphocytopaenia and very low eosinophile counts. Thrombocytopaenias are frequently the result of toxic action of chemical, vegetable or physical agents and are also seen during the course of other conditions like haemolytic icterus, certain other anaemic conditions or infections. That the thrombocytopaenia observed is of a transient nature is borne out by the fact that bleeding tendencies do not develop during the course of the disease, nor have any such disturbances been observed in normal healthy Karroo sheep.

Marked hypoplasia of the red bone marrow has been observed in many cases of typical Enzootic Icterus, and in the carcases of apparently healthy sheep slaughtered in areas where this disease is prevalent[17]. It will be recalled that it was also a feature of the pathology of the mixed Geeldikkop-Enzootic Icterus syndrome cases described in the foregoing text

However, whether the mild anaemic changes are of toxic origin or not, there are certain other factors to which certain of the blood cell dyscrasias could be attributed, notable amongst which is a disturbance of the adrenal cortex. When all the findings relevant to these cases are considered, especially the chemical pathology, there is little doubt that the adrenals are severely affected throughout the course of the disease. Whether the adrenal disturbances are due directly to the agents causing the disease, or whether these changes represent a very severe stress reaction, is not yet clear. Leukopaenia is encountered in adrenal cortical deficiency states[18]. The neutrophile disturbances, severe lymphocytopaenia and atrophy of the lymphoid tissue throughout the body, are in accordance with a prolonged stress condition in sheep, and are similar to those described by Clark[19] as occurring in ovine pregnancy toxaemia.

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Various tests, based on the response of circulating eosinophiles to the administration of A.C.T.H., have been introduced in human medicine as measures of adrenal function, It has been found that the eosinophile count, in consideration with the plasma electrolyte values, is very useful for this purpose in sheep. It has been found, that although a rise in the eosinophile count is sometimes encountered in sheep showing an apparent adrenal hypofunction, there is more generally a marked fall in the level of these cellular elements, very low counts being the rule until the animal recovers or death supervenes[20]. More will be said of this aspect in the paper dealing with the chemical pathology of the disease.

The thrombocytopaenia is not easy to explain in terms of an adrenal disturbance, yet besides being seen in the earlier cases, it was seen in the long standing case described, which was clearly one of adrenal exhaustion. It may however have been due to some or other secondary toxic influence in this case.

One other rather interesting finding is the mild bacteraemia found in two of the most severely leukopaenic clinical cases. As these sheep showed no febrile reaction these bacteria were apparently non-pathogenic, and probably after gaining access to the blood through the mucous membranes, were able to proliferate in the body owing to the severe leukopaenia. This is a well-known finding in agranulocytosis due to any particular cause. It is scarcely a wonder that many natural cases of the disease succumb to secondary infection of the lesions of photosensitization.

The cases from Victoria West are most interesting since they show certain features which have always been regarded as characteristic of Enzootic Icterus, notably the presence of numerous large macrophages containing granules of iron-free pigment in organs like the liver, lymph nodes, the haemosiderosis and the appearance of the kidneys at autopsy.

Geeldikkop and Enzootic Icterus, in their typical forms, could be considered as the two extremes of a single basic condition. The former is characterized by a severe photosensitization, a severe icterus in the nature of an intrahepatic cholestasis and severe nephrosis, while in the latter a severe acute haemolytic icterus and extremely severe nephrosis is considered the typical disease pattern. Each syndrome seems to be precipitated by a different set of circumstances or factors, e.g. in the case of Geeldikkop, *Tribulus* is in some way concerned, while in the case of Enzootic Icterus some severe stress condition plays the major rôle, for instance climatic conditions, transport of the animals any long distance by rail or a virus infection like Wesselsbron virus disease (vide le Roux 1959[21]). In between the two extremes are encountered the various symptom complexes which may be considered as atypical of either. The following are representative of these:—

(*a*) severe photosensitization with mild or transient icterus as seen in the early and advanced Victoria West cases;

(*b*) severe photosensitization and severe icterus of the typical Geeldikkop type with many of the pathological features of En ootic Icterus superimposed;

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(c) cases of mild photosensitization and severe icterus with a high percentage of unconjugated bilirubin and little conjugated pigment in the blood, the pathological picture being predominantly that of Enzootic Icterus;

(*d*) lingering cases showing either a very mild retention icterus or no icterus at all but a very severe nephritis, and pathological features of neither major syndrome.

In general the liver lesions of typical Geeldikkop seem to be essentially similar to those described by Ender[22] as encountered in "Alveld", the Norwegian photosensitization disease of sheep, and have some features in common with the liver pathology of chlorpromazine icterus in humans. However no evidence of obstruction of minute bile ducts near the portal triads (caused presumably by pressure on them from the more or less heavy infiltration of round cells, polymorphonuclear cells, histiocytes or eosiniphiles in the surrounding tissue) as seen in the latter condition has been encountered yet, nor is there any evidence of inspissated bile or bile plugs in the small bile capillaries. Thus although we regard the icterus of Geeldikkop as an intrahepatic cholestasis, albeit for want of a better name at present, it is apparently not typical of the human primary intrahepatic cholestasis on purely pathological grounds.

The pathology of Facial Eczema in New Zealand has recently been described by various authors[23],[24],[25]. The liver lesions found in this disease differ markedly from those of typical Geeldikkop and include marked fibrosis of the intra- and extra-hepatic bile ducts, necrosis of the lining of these, obstruction of many of the smaller ducts by cellular debris, mucin or bile plugs, numerous areas of focal necrosis within the liver lobule and at the angles of the portal triads and certain interesting vascular changes.

There is little doubt that the majority of Geeldikkop cases which succumb to the disease do so as a direct result of the kidney lesions with the attendant uraemia and electrolyte imbalances. Adrenal exhaustion, with its associated electrolyte disturbances and water depletion, is probably the second important cause of mortality, especially in long standing cases, while death following septicaemia from secondarily infected lesions of photosensitization is by no means uncommon.

Summary.

The haematology, gross pathology and histopathology of cases representing all stages of the typical Geeldikkop syndrome are presented. Cases representing various deviations from the typical pattern are discussed and some thoughts regarding the close relationship of Geeldikkop and Enzootic Icterus advanced. Certain blood cell dyscrasias such as thrombocytopaenia, leukopaenia, lymphocytopaenia and a marked "shift to the left" in the neutrophiles are reported for the first time as part of the pathology of the disease. Other new findings recorded are atrophy of the lymphoid tissue throughout the body, severe renal lesions, hypoplasia of the red bone marrow and severe

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involvement of the adrenals. The liver pathology is discussed fully and compared with that of "Alveld", "Facial Eczema", and human chlorpromazine icterus. Some thoughts are also advanced regarding the cause of death of natural cases of the disease.

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ISOLASIE VAN DIE PROTOKSIEN VAN *CLOSTRIDIUM WELCHII* TIPE D.

D. W. Verwoerd*

INLEIDING

In 1933 het Glenny[1] vir die eerste keer *Clostridium welchii* tipe D van die ander lede van die groep onderskei, op grond van die serologiese eienskappe van die toksiene wat dit afskei. Hy het bewys dat tipe D hoofsaaklik epsilon-toksien vorm, hoewel daar ook klein hoeveelhede alfa- en theta-toksiene in 'n jong kultuur teenwoordig is. Nicholson[3] en later ook Guillaume[2] het bewys dat tipe D se toksien minder hittelabiel as die ander is. In 1934 het Bosworth en Glover[4] gevind dat die toksisiteit van 'n tipe D-filtraat verhoog kan word deur dit met tripsien in 'n broeikas te laat staan. Hulle het ook bewys dat slegs epsilon-aktiwiteit in 'n kultuur wat 'n paar dae oud is voorkom, aangesien die ander twee binne 'n paar uur vernietig word. Verder het hulle sonder twyfel vasgestel dat die epsilon-toksien verantwoordelik is vir die patogenese van enterotoksemie by skape. Glenny[1] het weer opgemerk dat kulture wat in fosfaat-buffer staan, ook in giftigheid toeneem.

Hierdie resultate is in 1943 deur Turner en Rodwell[5] bevestig, en die toename in aktiwiteit is verklaar deur aan te toon dat die epsilon-toksien deur die organisme in 'n onaktiewe vorm, protoksien genoem, afgeskei word, wat dan deur proteolitiese ensiemwerking in aktiewe toksien omgesit word. 'n Mate van self-aktivering deur middel van die organisme se eie proteolitiese ensieme vind plaas, veral in gebufferde milieu, maar hierdie proses kan versnel en tot 'n maksimum gevoer word deur inkubasie na byvoeging van tripsien.

Wat die chemiese aard van die toksien en sy fisiologiese werking betref, is daar maar min gegewens beskikbaar. Intraveneus is dit dodelik vir laboratorium-diere, terwyl selfs groot dosisse *per os* geen effek het nie. Onlangs het Bullen en Batty[6] egter bewys dat die gifstof die permeabiliteit van die dermwand beïnvloed en dat die toediening van vyf dosisse gekonsentreerde filtraat, met een uur tussenpose, lei tot die absorbsie van 'n letale hoeveelheid. Kellaway[7] het die invloed van 'n gedeeltelik-gesuiwerde ammoniumsulfaat-presipitaat op longweefsel ondersoek, en vrystelling van histamien gevind. Later verklaar hy egter self dat hierdie effek aan onsuiwerhede in sy preparaat toegeskryf moet word, en dat die neurotoksiese effek van die gifstof te wyte is aan 'n direkte aantasting van die basaalganglia. Die enigste spesifieke patologiese letsel wat deur die toksien veroorsaak word, is 'n tipiese dermonekrose na intra-dermale inspuiting wat deur Batty en Glenny[8] gebruik is om die toksien te tipeer.

Van die chemiese aard van die toksien is daar niks bekend nie, behalwe dat dit 'n proteïen is. Onlangs is 'n stappie verder gevorder toe Meisel-Mikolajozyk[9] polisakkariedes uit 'n welchii-D-filtraat

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geïsoleer het wat 'n positiewe komplementbindingsreaksie gegee het. Op grond van chromatografiese analises van die polisakkariede kon hy ook verskille tussen stamme aantoon.

Uit hierdie oorsig blyk dit duidelik dat daar nog maar min bekend is van hierdie ekonomies belangrike gifstof. Die grootste struikelblok in die weg van 'n deeglike ondersoek na die aard daarvan, is die feit dat dit nog nooit in suiwer vorm geïsoleer is nie. Met hierdie doel voor oë is onderstaande studie aangepak.

Materiaal en Metodes.

Kweking van die Organisme.

Kweekbodem.—'n Chemies-gedefinieerde medium, opgebou uit aminosure, vitamiene, puriene en minerale, is gebruik vir die kweek van die organisme, om sodoende die isolasie van die proteïen-gifstof nie onnodig te kompliseer deur die teenwoordigheid van vleisproteïene nie. As basis is die medium van Boyd, Logan en Tytell[10] gebruik met die volgende byvoegings (per 100 ml. kweekbodem).

Nikotiensuur	0.02 mg.
Tiamien-HCl	0.02 mg.
Cholien-HCl	0.005 mg.
Inositol	0.005 mg.
p-Aminobensoësuur	0.001 mg.
Foliensuur	0.0001 mg.
$CoCl_2$	0.05 mg.
NH_4Cl	0.1 mg.
$CaCl_2$	1.0 mg.
Na-asetaat	1.0 mg.

Verder word die glukose vervang deur die onoplosbare fraksie (by 4°C) van kommersiële dekstrien. Op hierdie kweekbodem is dieselfde peil van toksienproduksie as op vleissop gehandhaaf en soms selfs 'n hoër opbrengs verkry, ten spyte van swakker groei (gemeet aan seldigtheid)

Stamkultuur.—'n Stam wat vir hoë toksienproduksie uitgesoek is, ET. 95, is van dr. B. Jansen, Onderstepoort, verkry, en vir alle eksperimente gebruik.

Metode.—40 L. kultuur is vir elke bereiding van ru-toksien gebruik. Vitamiene en die dekstrien is afsonderlik gesteriliseer om die vorming van toksiese karamel-produkte te voorkom. Kulture is vir 48 uur geïnkubeer en dan so gou as moontlik tot 0°C afgekoel, waar dit vir 'n paar weke sonder afname in aktiwiteit houbaar is.

Tripsien-aktivering.

Alle monsters van kulture en presipitate is met tripsien geaktiveer volgens die voorskrif van Novotny[11], d.w.s. deur 0.25% tripsien by te voeg en vir 45 min. by 37°C in 'n waterbad te hou. Onmiddellik daarna is die toetstitrasies gedoen.

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MUIS-TRITRASIES.

Biologiese aktiwiteit van die toksien is deur middel van MLD.-titrasies in muise bepaal. Witmuise van ongeveer 20g. gewig is gebruik en 'n dosis van 0.2 ml. binne-aars ingespuit. Drie tot vier muise is by akkurate bepalings vir elke verdunning gebruik, anders meestal net twee. Die positiewe eindpunt van die titrasie is beskou as die verdunning waar drie uit die vier (of albei waar net twee gespuit is) binne 24 uur dood is. Toksien-inhoud is uitgedruk as die aantal MLD. per ml., en na stikstofbepaling as die aantal MLD. mgm. N. Laasgenoemde getal dien ook as suiwerheidsindeks (S.I.) omdat dit 'n aanduiding is van die hoeveelheid aktiewe eiwitte wat 'n fraksie bevat.

MIKRO-STIKSTOFBEPALINGS.

Die metode van Johnson[12] is gebruik. Dit is gebaseer op 'n gewone Kjehldal-vertering van die monsters, gevolg deur kolorimetriese meting van die kleur wat met Nessler se reagens ontwikkel word.

ANTIGEEN-ANALISE DEUR GEL-DIFFUSIE.

In breë trekke is die metode van Oakley en Fulthorpe[13] gevolg in die bepaling van die aantal antigene teenwoordig in die verskillende fraksies. Die doel hiervan was om 'n direkte aanduiding te kry van die vordering van die suiweringsproses. Die metode berus op die feit dat antigene en hul homoloë teenliggaampies, wat by geskikte konsentrasies van weerskante deur 'n gel diffundeer, sigbare presipitien-bandjies vorm.

Hoewel die aantal bandjies geen absolute weergawe van die aantal antigene is nie, aangesien 'n oorvleueling van twee of meer bande moontlik is, gee dit tog 'n baie sterk aanduiding van die antigeniese suiwerheid al dan nie, van 'n toets-fraksie.

Die beste resultate is verkry met langsame diffusie deur 1.5% gelatien by 20°C. Onder hierdie omstandighede kan die resultaat egter eers na 12–14 dae gelees word. Diffusie deur 1% agar by kamertemperatuur is veel vinniger (48 uur), maar resultate was nie altyd so goed nie. Die presipitasie-patroon van die ongesuiwerde protoksien, wat bestaan het uit al die neergeslaande eiwitte van 'n 48-uur oue kultuur, het 18 bandjies aangetoon, en is gebruik as standaard waarmee elke verdere presipitasie-stap vergelyk is. 'n Voorbeeld van die resultate wat verkry is, verskyn in fig. 1. As antiserum is 'n homoloë perdeserum gebruik wat gekonsentreer en ensimaties geraffineer is. Dieselfde antiserum is vir alle eksperimente gebruik.

METANOL-PRESIPITASIE BY LAE TEMPERATURE.

Met fraksionele presipitasie deur byvoeging van metanol is die beste resultate behaal in die suiwering van die ru-toksien. Verskeie eiwitte, o.a. ook bakteriese toksiene, is deur veral Pillemer en sy medewerkers[14-22] geïsoleer volgens 'n verskeidenheid van metodes wat almal op metanol-presipitasie gebaseer is. Hy was bv. die eerste werker om 'n kristallyne tetanus-toksien te berei. Deur noukeurige beheer van die pH, temperatuur, ioonkonsentrasie, alkoholkonsentrasie en konsentrasie van die eiwit, word omstandighede geskep wat differensiële

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presipitasie van die verskillende eiwitte bewerkstellig. Eksperimentele prosedure het dus bestaan uit 'n sistematiese studie van die geskikste toestande daarvoor. Veral belangrik, wat die tegniek betref, is die feit dat alle stappe in die fraksioneringsproses by –5 °C uitgevoer moet word, en dat die temperatuur nooit hoër as –1 °C mag styg nie, anders vind denaturering van die eiwit, gepaard met verlies van aktiwiteit, plaas.

Resultate.

Bereiding van 'n Gekonsentreerde Ru-toksien.

Nadat die 40 L. kultuur tot 0 °C afgekoel en die pH op 4.6 ingestel is met 1N asynsuur, word alle eiwitte daaruit met 35 % metanol, wat tot —20 °C afgekoel is, neergeslaan. Die neerslag word afgeswaai, tweemaal met 1 L. yskoue gedistilleerde water gewas, en daarna met 1 L. yskoue 0.1 M fosfaatbuffer by pH 6.3 (sien volgende paragraaf) ekstraheer. Resultate van die bereidingsproses word in tabel I aangegee. Die gifstof blyk relatief min oplosbaar in suiwer water te wees, dus gaan daar nie veel met die wasproses verlore nie. Die bufferekstrak word gevriesdroog, in welke vorm dit vir maande lank sonder verlies in aktiwiteit houbaar is.

Tabel I. Bereiding van Rutoksien.

Kultuur (40 L.) Toksieninhoud 10,000 MLD/ml.

(35 % MeOH by –5°C)

Filtraat 1 — Presipitaat 1

(Was met 1 L. water, sentrifugeer)

1,200 MLD/ml. F2 — P2.

(Was met 1 L. water, sentrifugeer)

1,000 MLD/ml. F3 — P3.

(Los op in 1 *L.* fosfaatbuffer, 0.1M pH 6.3, sentrifugeer)

20,000 MLD/ml. F4 — P4.

(Vriesdroging)

Groep	*Toksieninhoud MLD/ml.*	*Toksieninhoud na 4 maande MLD/ml.*
1	10,000	10,000
2	5,000	5,000
3	5,000	5,000

Suiwering van die Ru-Protoksien.

Oplosbaarheidseienskappe:

Deur die gevriesdroogde ru-produk in 'n aantal buffers by verskillende konsentrasies en pH-waardes op te los, skoon te swaai en te analiseer, is gevind (tabel II) dat die protoksien maksimaal oplosbaar is in 0.1 M fosfaatbuffer (0.1 M Na_2HPO_4 + 0.1 M NaH_2PO_4) by pH-

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waardes bo 5.9. Kontaminerende eiwitte wat teenwoordig is, se oplosbaarheid neem egter ook toe met stygende pH, dus sal die suiwerste oplossing verkry word by ongeveer 5.9—6.0. Uit tabel II kan verder gesien word dat die fraksie wat by hierdie pH oplos, 'n suiwerheidsindeks van 6.0×10^5 MLD./mgm. N toon, teenoor 2.0×10^5 by die ru-produk.

TABEL II: OPLOSBAARHEID BY VERSKILLENDE pH-WAARDES.

pH	*MLD/ml.	mgm. N/ml.	S.I.=MLD mgm. N.
Rutoksien	200,000	0.95	200,000
4.5	10,000	—	—
5.0	10,000	—	—
5.5	20,000	—	—
5.75	100,000	—	—
6.0	200,000	0.325	600,000
6.25	200,000	—	—
6.5	200,000	0.46	410,000
7.0	200,000	0.56	375,000
7.5	200,000	0.57	370,000

*Gevriesdroogde materiaal in $^1/_{20}$ van die oorspronklike volume opgelos, d.w.s. 20x gekonsentreer.

Metanol-Presipitasie:

Om die aantal faktore wat die neerslaan-proses beïnvloed tot 'n minimum te beperk, is die volgende konstant gehou in alle eksperimente

1. Die temperatuur op —5°C.
2. Die eiwitkonsentrasie op 0.5 mgm. N/ml.
3. Die ioonsterkte van die oplossing op 0.125.

Die invloed van die twee oorblywende faktore, nl. pH en metanolkonsentrasie op die presipitasie-eienskappe van die protoksien is daarna ondersoek. Resultate word saamgevat in tabel III.

TABEL III: PRESIPITASIE-EIENSKAPPE.

pH	Metanol konsentrasie	Ontleding van neerslag		
		mgm.N/ml.	MLD/ml.	MLD/mgm.N.
5.5	10%	0.635	400,000	600,000
	20%	0.025	8,000	320,000
	30%	0.175	20,000	100,000
6.0	10%	0.29	200,000	700,000
	20%	0.12	200,000	1,600,000
	30%	0.0875	20,000	200,000
6.5	10%	0.225	100,000	400,000
	20%	0.22	50,000	230,000
	30%	0.05	20,000	400,000
7.5	10%	0.09	—	—
	20%	0.4	40,000	100,000
	30%	0.3	20,000	60,000

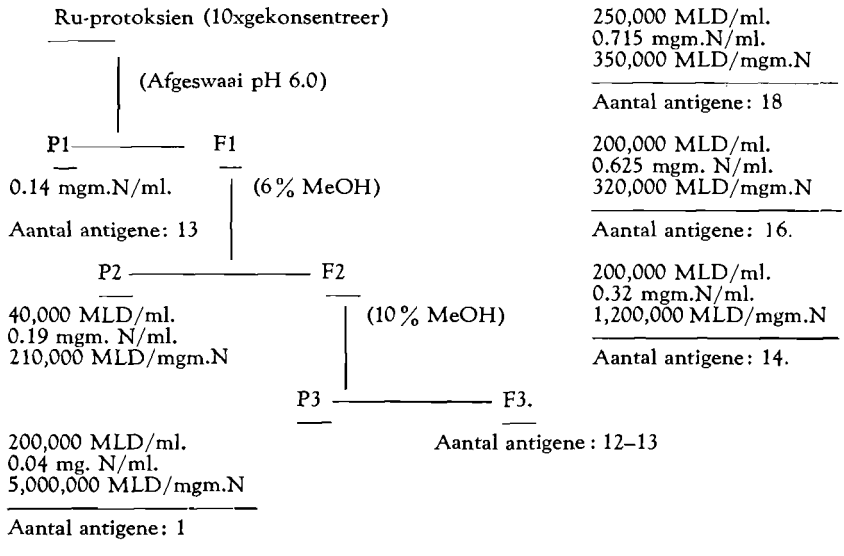
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Hieruit blyk duidelik dat die toksiese faktor moeiliker neerslaan hoe hoër die pH word. Verder is dit duidelik dat die grootste deel van die aktiwiteit by die laere pH-waardes reeds deur 10% metanol neergeslaan is.

Ondersoek van die gebied onder 10% het gevolg, en gelei tot die bevinding dat 'n klein fraksie met 'n baie hoë aktiwiteit berei kan word deur eers met 6% metanol te presipiteer, die konsentrasie dan tot 10% te verhoog en af te swaai. Hierdie fraksie het 'n suiwerheidsindeks van 3×10^6 MLD/mgm. N gehad, en gel-diffusie-analise het slegs een antigeen aangetoon.

Pogings om hierdie resultaat te reproduseer het aanvanklik misluk, maar deur uiters noukeurige beheer van eksperimentele toestande, veral wat temperatuur betref, toe te pas is later nog tweemaal daarin geslaag om op klein skaal preparate te berei met vergelykbare S.I. nl. 2.2×10^6 en 5.0×10^6 MLD/mgm. N. en slegs een antigeen. Mislukkings is blykbaar aan denaturering, wat baie maklik plaasvind, te wyte. Dit gebeur veral maklik as die metanol te vinnig bygevoeg word. Die volledige metode, asook die resultate daarmee behaal, word in tabel 4 skematies opgesom. Die verloop van die proses volgens die aantal antigene in elke fraksie teenwoordig, word in fig. 1 weergegee.

TABEL IV: BEREIDING VAN SUIWER EPSILON-PROTOKSIEN.



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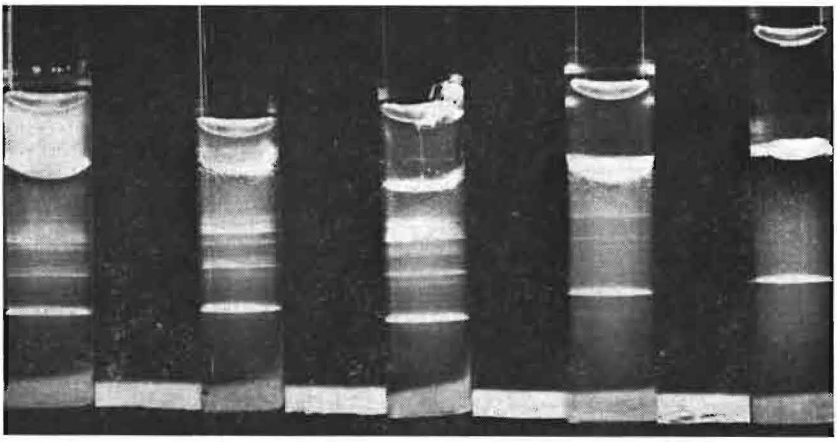


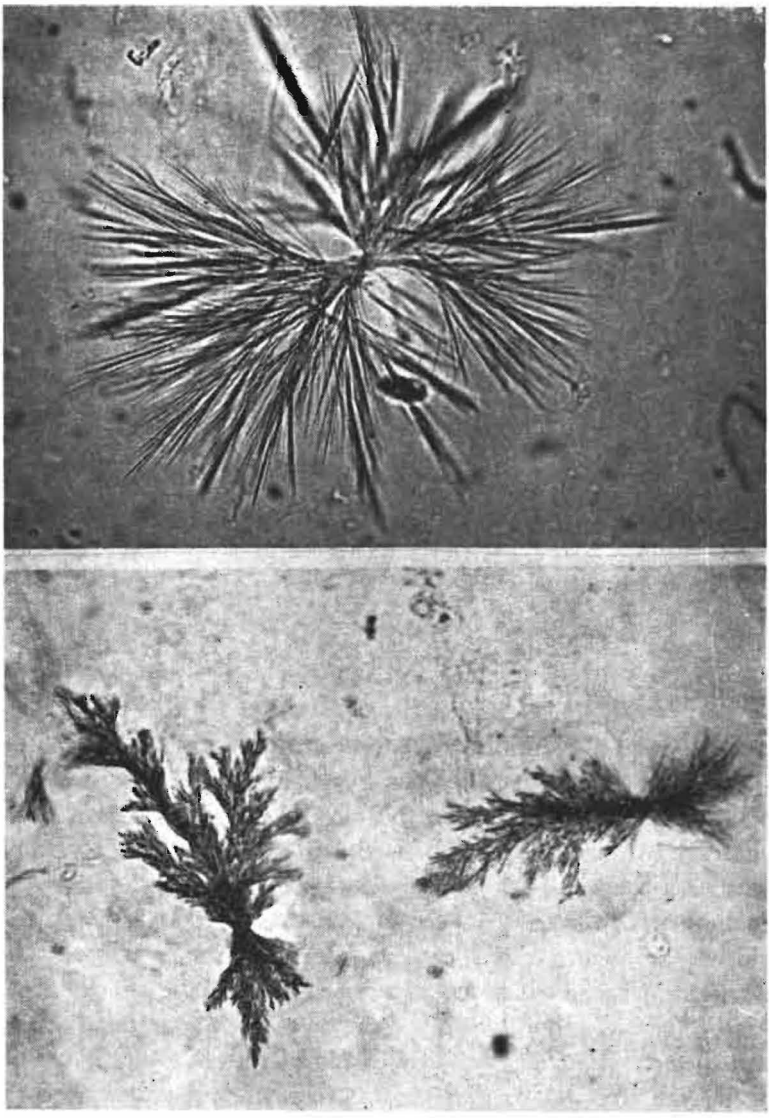
Fig. 1: Aantal Antigene in die fraksies van Tabel 4: V.l.n.R. R, F1, F2, F3, P3.

Kristallisering:

Ongeveer 1–2 mgm. van die gesuiwerde materiaal is in 1 ml. 0.1 M fosfaat-buffer pH6.0 opgelos. Na byvoeging van 35% metanol by —5°C is dit by —20°C gebêre. Na agt dae is 'n aantal klein kristalle gevind wat byna nie met die blote oog sigbaar was nie, en by temperature hoër as ongeveer—2°C weer opgelos het. Mikroskopies was die kristalle gerfvormig met 'n sterk ingesnoerde middel (fig. 2A), en die beeld was homogeen, d.w.s. geen ander kristalle is gevind nie. Nadat die kristalle weer in oplossing gegaan het, is dit vir 'n tweede keer vir 7 dae by —20°C gehou, waarop daar weer kristalle gevorm het, maar hierdie keer hoofsaaklik met 'n effens ander bou-vorm (fig. 2B). Enkele kristalle van tipe A was ook teenwoordig. Toksien-titrasies met en sonder tripsien-aktivering het 'n groter persentasie toksien teenoor protoksien in die tweede preparaat aangetoon, wat moontlik die twee kristalvorms kan verklaar. Ongelukkig was daar nie genoeg materiaal beskikbaar vir verdere werk in hierdie verband nie, en die identiteit van die kristalle kon nie bo alle twyfel vasgestel word nie.

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FIG. 2(A) EN (B): KRISTALVORMS.



Summary

A method is described by which isolation of purified epsilon-protoxin of Clostridium welchii type D was achieved. Precipitation with methanol at —5 °C was used to prepare a crude extract from a culture grown on a synthetic medium. After freeze-drying, this preparation was further purified by the same method. Antigenic analysis by gel diffusion as well as titrations in mice were used as criteria of purity. Comparable results were obtained in three independent experiments, preparations showing only one antigen-precipita-

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tion line, and containing 2.2.–5.0 $\times$ 10^6 MLD/mg.N, being obtained. Crystallization of one preparation occurred on storage for ten days at —20°C after addition of 35% methanol, but lack of material prevented definite identification.

Erkenning

Hierdie artikel word met die vriendelike vergunning van die Direkteur van Veeartsenydiens, Onderstepoort, gepubliseer. Die materiaal daarin bevat, het deel uitgemaak van 'n verhandeling by die Universiteit van Pretoria ingedien.

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THE EXPERIMENTAL REPRODUCTION OF PULPY KIDNEY DISEASE

B. C. JANSEN*

The earlier attempts at the experimental reproduction of enterotoxaemia are admirably reviewed by Bullen and Batty[1]. These efforts, involving the oral dosing of large amounts of actively growing culture of *Clostridium welchii* type D, were not regularly attended by success. In the same publication Bullen and Batty describe the stepwise development of a procedure by which the disease can be produced with certainty. In this method, *Cl. welchii* type D is introduced into the duodenum by a continuous drip of culture, while the experimental sheep are subjected to a digestive upset by being allowed to over-eat on wheat. The over-eating is associated with the appearance of undigested starch granules in the intestine, which stimulates the continued multiplication and increased toxin production of the bacteria. This rapidly leads to death in spite of the development of a diarrhoea which normally acts as a protective clearing mechanism.

In the course of the production of a vaccine against pulpy kidney disease at the Onderstepoort Research Laboratories it was found that dextrin, of all the carbohydrates tested, stimulated toxin production to the greatest extent. This confirmed the finding of de Mello, Clemente, Kiser and Williams[2]. In comparing the ability of dextrin to enhance toxin production with that of glucose, the following experiment was conducted.

Horse-meat broth was prepared by boiling minced horsemeat in tap water (1 lb. of meat to 1 litre of water) for thirty minutes. For each litre of water was added:

Peptone.....	10 gm.
$Na_2HPO_4.2H_2O$	2 gm.

The liquid part of the broth was siphoned off and the meat particles were transferred to twelve-litre flasks to fill about one-fifth of the volume of the flasks. Subsequently the flasks were filled to about ten-litre capacity with the liquid broth and sterilized at 120°C for 1½ hours. After sterilization the medium was allowed to cool and then placed in an incubator at 37°C to attain the desired temperature.

Before inoculation of the medium its pH was adjusted to 8.0 with sterile N.NaOH and the following additions were made to different, numbered flasks:

(1) A concentrated sterile solution of commercial dextrin to give a final concentration of one per cent. dextrin.

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(2) Dextrin washed repeatedly in cold water, dried and resuspended in water in a concentrated form. The concentrate was sterilized and added to the medium to give a final concentration of one per cent. dextrin.

(3) A concentrated sterile solution of glucose to yield a final concentration of one per cent. glucose.

The carbohydrate solutions were sterilized by tyndallization.

Each flask was inoculated with 30 ml. of actively growing culture of *Cl. welchii* type D and from two and one half hours after inoculation (when active gas production became evident) the pH of the medium was readjusted to 8.0 every hour. Incubation took place at 37°C. After seven hours' growth samples were withdrawn from each flask and the Lf value determined. The flocculation units correspond to the Wellcome Research Institute units for epsilon antitoxin. The results are recorded in Table I.

TABLE I

Flask No.	Toxin (Lf/ml.)
1 (Dextrin)	250
2 (Washed dextrin)	100
3 (Glucose)	25

From these results it is obvious that dextrin stimulates the production of higher yields of toxin than the same concentration of glucose. The soluble portion of the dextrin plays an important rôle in this respect.

On the strength of these findings an experiment was planned in which quantities of both actively growing culture of *Cl. welchii* type D and a solution of dextrin were injected into the duodenum of sheep.

Sheep showing no detectable epsilon antitoxin in their sera were selected.

The experimental sheep were fed on lucerne hay and green lucerne *ad lib.* in addition to half a pound of concentrate containing 16 per cent. protein per sheep for at least fourteen days prior to being infected. It was found that enterotoxaemia could not be produced in sheep that were fed on a poor diet, e.g. teff hay only.

A six-hour culture of *Cl. welchii* type D in tubes of Robertson's meat broth was used for injection. The dextrin solution contained 40 gm. of commercial dextrin to every 100 ml. water.

To expose the duodenum the abdomen was opened on the right side by making a one-inch incision below the chondral arch about four inches from the ventral midline. Local anaesthetic was used.

From Table II can be seen the quantities of culture and dextrin solution injected, as well as the results.

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TABLE II

THE QUANTITIES OF *Cl. Welchii* TYPE D CULTURE AND DEXTAIN SOLUTION INJECTED INTRADUODENALLY INTO SHEEP

Sheep No.	Volume culture (ml.)	Volume dextrin solution (ml.)	Result
8095	20	—	Lived
8124	20	—	Lived
7448	1	20	Lived
8114	5	20	Died overnight
8098	10	20	Died overnight
8097	15	20	Died overnight
8126	20	20	Died overnight
8709	5	5	Lived
8702	5	10	Lived
8690	5	15	Lived
8688	5	20	Died overnight

All the deaths were confirmed as being due to enterotoxaemia by:

(*a*) detection of epsilon toxin in high concentrations in the gut contents,

(*b*) isolation of *Cl. welchii* type D from the gut contents,

(*c*) finding typical organisms in large numbers in intestinal smears, and

(*d*) isolation of *Cl. welchii* type D from liver and kidney specimens collected with aseptic precautions.[3]

From the results in Table II it can be concluded that:

(1) The injection of as little as 5 ml. of a six-hour culture of *Cl. welchii* type D together with 20 ml. 40 per cent. dextrin into the duodenum of a susceptible sheep leads to the development of fatal enterotoxaemia.

(2) When less than 20 ml. 40 per cent. dextrin solution is injected intraduodenally together with 5 ml. culture the disease does not develop.

(3) The intraduodenal injection of 20 ml. of culture alone does not affect a sheep.

THE LEVEL OF EPSILON ANTITOXIN TO PROTECT SHEEP AGAINST ARTIFICIAL CHALLENGE

A number of sheep which had been vaccinated with alum-precipitated pulpy kidney vaccine were selected and bled immediately before being infected with enterotoxaemia. The sera were recovered from the blood and their antitoxin value determined in terms of Wellcome units by toxin-antitoxin titration in white mice. An injection of 20 ml. of a six-hour culture of *Cl. welchii* type D in Robertson's meat broth and 20 ml. 40 per cent commercial dextrin solution were then administered into the duodenum of each. The results are recorded in Table III.

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TABLE III

THE RESULTS OF CHALLENGING SHEEP WITH DIFFERENT LEVELS OF CIRCULATING ANTITOXIN

Number challenged	Antitoxin level (units/ml.)	Result
2	0	2 died
7	0.1	3 lived, 4 died
4	0.15	4 lived
2	0.3	2 lived

The deaths occurred overnight and the diagnosis of enterotoxaemia was confirmed at post-mortem by applying the same criteria as in the previous experiment.

From these results it can be reasonably concluded that an antitoxin level of at least 0.15 units per ml. of circulating antitoxin renders a sheep immune to pulpy kidney. This finding is in agreement with the estimate of Thomson and Batty[4] that an antitoxin level in the order of 0.1 unit per ml. is the lowest level which will afford protection against enterotoxaemia in nature.

SUMMARY

(1) The ability of the soluble fraction of commercial dextrin to stimulate epsilon toxin production by Cl. *welchii* type D to a greater extent than the same concentration of glucose has been demonstrated.

(2) An experimental method has been developed by which a susceptible sheep can be infected with fatal enterotoxaemia. This consists of the injection of 5 ml. of an actively growing culture of Cl. *welchii* type D into the duodenum together with 20 ml. of a 40 per cent. solution of dextrin under local anaesthesia.

(3) It was proved that a level of at least 0.15 Wellcome units of circulating epsilon antitoxin is sufficient to protect all sheep against the experimental reproduction of the disease.

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THE OCCURRENCE OF PULPY KIDNEY IN SHEEP DOSED WITH PHENOTHIAZINE

B. C. Jansen*

During a discussion at the Seventy-fifth Annual Congress of the British Veterinary Association, Harbour[1] stated that "it is a common experience to see outbreaks of pulpy kidney disease immediately following dosing with phenothiazine". Many farmers in South Africa have reported the same phenomenon to the Onderstepoort Veterinary Research Institute but, although the evidence seemed sufficiently convincing, there was no experimental confirmation.

Subsequent to the development of a method for the experimental reproduction of pulpy kidney disease (enterotoxaemia),[2] a study was made of the possibility of facilitating the onset of the experimental disease by dosing phenothiazine.

Experimental Procedures and Results

Sheep which showed no detectable epsilon antitoxin in their sera were selected. They were fed on lucerne hay, green lucerne plus a supplement of crushed yellow maize. Immediately prior to the operation six sheep were dosed with a therapeutic quantity of phenothiazine (0.2 gm. per kilo body weight) each. The operation consisted of the introduction of 20 ml. of a six-hour culture of *Cl. welchii* type D into the duodenum under local anaesthesia.[2]

As control the same quantity of culture was injected into the duodenum of two susceptible sheep which had not received a dose of phenothiazine.

Four of the six sheep administered phenothiazine as well as 20 ml. of *Cl. welchii* culture died during the night following this experimental procedure. Two of the sheep survived, as did the two control sheep which had received culture only. From this it was concluded that in a considerable proportion of sheep containing *C. welchii* type D in their intestines the dosing of phenothiazine will provoke the development of fatal enterotoxaemia.

To investigate the circulating epsilon antitoxin level which protects a sheep against the development of enterotoxaemia following on the dosing of phenothiazine, sheep that had been vaccinated with an alum-precipitated epsilon toxoid were used. They were bled and immediately afterwards dosed and infected by the method described. Subsequently the antitoxin values of their sera were determined in terms of Wellcome units by toxin-neutralization in mice. It was found repeatedly that sheep with a serum value higher than 0.1 unit of antitoxin per ml. did not die, while some of the sheep with 0.1 unit and the majority of the sheep with values lower than 0.1 unit per ml., died of enterotoxaemia.

*Section of Bacteriology, Onderstepoort.

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Discussion

As Bullen[3] found that a large percentage of normal sheep harbour *Cl. welchii* type D in their intestines, it will be readily appreciated, on the evidence produced above, that enterotoxaemia can result from farmers dosing their sheep with phenothiazine. It seems a fair assumption that the digestive changes effected by phenothiazine stimulate growth of and toxin production by the organisms artificially introduced into the intestines, as well as in the case of the organisms naturally present. The effects of phenothiazine on the digestive system of the ruminant are being studied at present and may provide information leading to an explanation of the sequence of events culminating in the disease.

The fact that sheep can be protected against the onset of enterotoxaemia after dosing with phenothiazine is of practical importance, and farmers should be advised to vaccinate their sheep before starting a programme of worm control involving the dosing of phenothiazine.

Summary

1. Experimental evidence has been produced to prove that the dosing of phenothiazine to sheep in therapeutic doses can induce the development of enterotoxaemia if the animals harbour *Cl. welchii* type D in their intestines.

2. A circulating antitoxin level higher than 0.1 Wellcome unit per ml. is sufficient to protect sheep against the development of enterotoxaemia subsequent to the dosing of phenothiazine.

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2. JANSEN, B. C., 1960: *This issue.*
3. BULLEN, J. J., 1952: *J. Path. Bact.* 64, 201.

CORRESPONDENCE

The following copy of a letter has been published at the request of Dr. Walters.

6, Napier Street,
Worcester, C.P.
25*th April*, 1960.

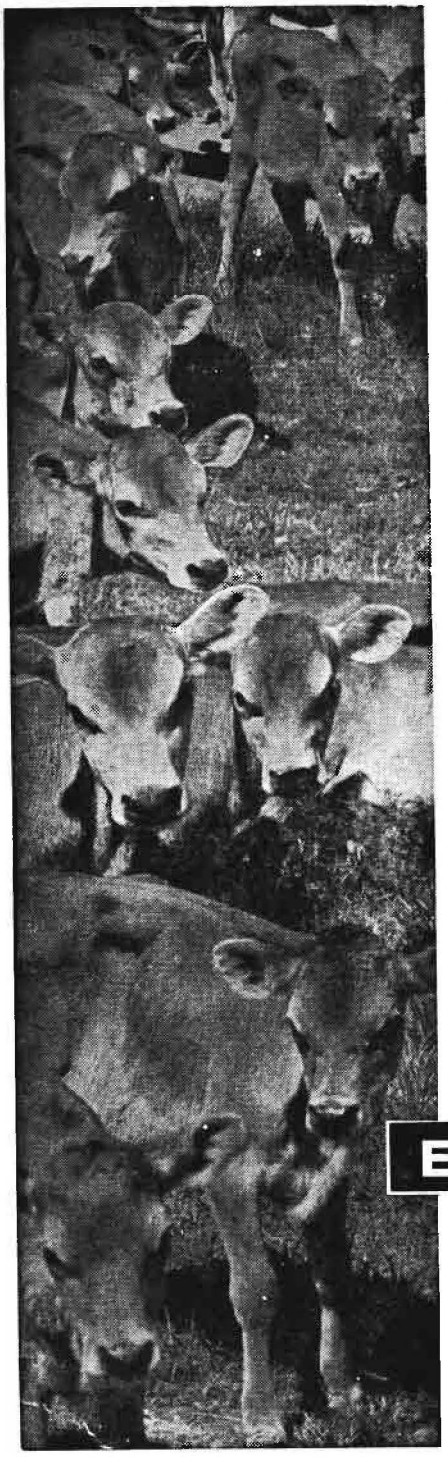
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Professional Provident Society of Southern Africa,
P.O. Box 6268, Johannesburg.

Sir,

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With many thanks,
Yours sincerely,
Signed: DENNIS M. WALTERS, B.V.Sc.

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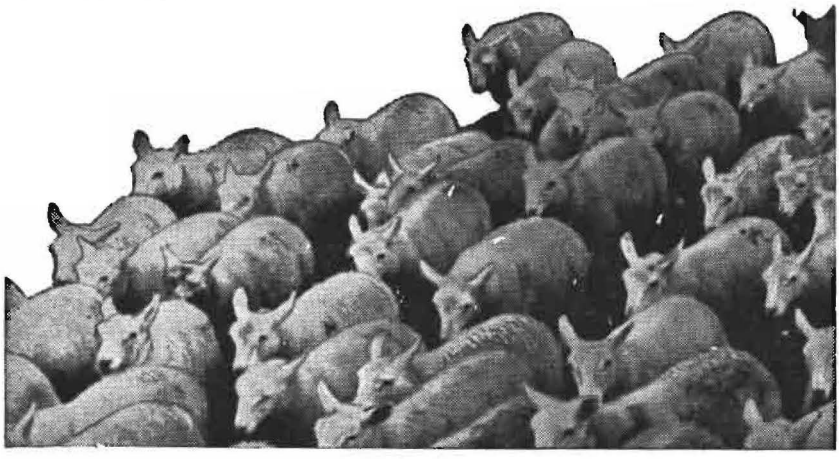
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1. Aust. Vet. Jl. 1954, 30:209.
2. Vet. Rec. 1955, 67:74.

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THE TUBERCULIN TEST IN CATTLE

H. H. Kleeberg*

The single intradermal tuberculin test in which mammalian tuberculin is used, is the official test for the detection of bovine tuberculosis in the Union of South Africa. This test was applied for the complete eradication of bovine tuberculosis from Holland, Denmark, Finland, Sweden and Switzerland. When the control of a disease depends on the application of a single test, it is imperative that such a test should be reliable. The reliability of a method of tuberculin testing depends on standardisation of the tuberculin, precision of the instruments used, uniformity of the technique employed and the knowledge and experience of the testing personnel. The results must furthermore be interpreted according to a standard schedule. An account of the different factors concerned in conducting a tuberculin test and the interpretation of the results is given in this article, in an attempt to standardise the factors involved and indicate certain pitfalls in the interpretation of reactions.

Four different factors are concerned in the test, viz.:—

(*a*) the tester;
(*b*) the tuberculin;
(*c*) the instruments and technique used;
(*d*) the animal.

Of these only (*b*) and (*c*) can be standardized. Human error can be excluded to a considerable extent by using automatic instruments, applying uniform technique, and interpreting the reaction according to set formulae.

Post mortem examination of reactors is important in ascertaining the reliability of the test.

Tuberculin

From the 1st January, 1960, the standard tuberculin issued from Onderstepoort has been bovine PPD prepared from strain AN5. Fresh batches of tuberculin are prepared every three months, and the expiry date is three months from the day of dilution. At present the concentration used is 2 mgm tuberculo-protein per ml., which is equivalent to 100,000 I.T.U. (International Tuberculin Units) per ml. The standard dose is 0.1 ml. (10,000 I.T.U.) which is very large in comparison with the 1 to 100 I.T.U. used for the intradermal test in humans.

The lowest dose which causes a reaction in tuberculous cattle lies between 10 and 500 I.T.U. As it appears that the strength of the tuberculin in current use can be reduced, a field experiment is being conducted to evaluate the effect of a more dilute tuberculin containing 7,000 I.T.U. per dose.

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Acid-fast organisms do not contain absolutely specific antigens. By physico-chemical and immunobiological methods, qualitative differences in the antigenic structure of the mammalian varieties of *Mycobacterium tuberculosis* have been demonstrated. The antigenic fractions isolated from the different varieties have shown low reactivity when used in skin tests, and quantities sufficient for significant tests have not yet been isolated.

PPD tuberculin must be stored in a refrigerator at 4°C. It is sterile and there is no hazard attached to storing it in a household refrigerator. Tuberculin should not be carried around in a car except on the days of testing, as shaking, high temperatures and light are detrimental. Tests performed at Onderstepoort showed that the potency of bovine PPD, kept at room temperature for six months, decreased down to 50 per cent. of its original value.

When tuberculin has been withdrawn from a container, the contents of that particular container should not be used after the following day, due to the possibility of the introduction of contaminants under field conditions.

Veterinarians must ensure that "expired" tuberculin is destroyed, since it is still sufficiently potent to be used for fraudulent purposes many months after the date of expiry.

The potency of all batches is checked by biological and chemical assays at monthly intervals up to the expiry date. There is a tendency among veterinarians to blame the tuberculin when difficulties are experienced with the interpretation of test results, but, compared with other biological products, it is a very reliable diagnostic preparation.

Human and avian PPD tuberculin are still available on request.

Instruments and Technique

Successful testing is largely dependent upon the choice of suitable instruments. The McLintock pre-set syringe and the Hauptner broadlip calliper with dial have been designed specifically for tuberculin testing, and are preferred for work under South African conditions. It is an advantage to use a belt and holder for the McLintock syringe so that both hands can be free for measuring. The Schimmel, the Record and the $\frac{3}{8}$ inch dental are all suitable needles.

The syringe should be cleaned with alcohol after use. For lubrication of the plunger a little thin oil, vaseline or glycerine should be used, avoiding contamination as far as possible. Immediately before use the syringe should be drawn full of sterile distilled water and emptied completely. Curved scissors, a razor or hair clippers may be used to remove the hair from a $1\frac{1}{2}$ inch square patch of skin at the site of injection. When using a razor, care should be taken not to injure the epidermis. The "Oster" hair clipper (blade size 000) has proved to be satisfactory and durable.

It is essential that the thickness of the skin at the site of injection is measured before injecting the tuberculin. By palpating the skinfold before measuring one can recognise possible nodules and avoid injecting at an unsuitable site. The lips of the calliper should clasp a

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skinfold lifted about one inch. The measuring of a fold at an adjacent site, after the reaction has developed, often leads to faulty interpretation.

There are two types of Hauptner callipers available. One is a simple sliding calliper with broad lips and a dial calibrated in units of 1/10th of a millimetre. The jaws are closed by thumb pressure which may be responsible for variations in measurement. The second (improved) model is held comfortably by a pistol grip. The jaws, which are opened by finger traction, close automatically due to the action of a coil spring. This arrangement ensures uniform pressure at each measurement. Furthermore it has a larger dial than the former instrument. When using this calliper, the reading must be taken before a soft oedematous swelling can be compressed.

Every animal to be tested must be clearly identified and properly marked, in such a manner that the marks cannot be erased. A paint mark or a chain round the neck is insufficient.

The injection should be given intracutaneously on the side of the neck midway between the head and shoulder and equidistant from the top and bottom necklines. This site shows the least variation in sensitivity. Bulls, oxen, and some Afrikander cattle, have very thick skins on the side of the neck and should therefore be injected immediately in front of the shoulder or even in the caudal fold. When a comparative test is applied the two tuberculins should be injected at identical sites on different sides of the neck. When this is impossible, both injections should be given on one side, at sites 6–8 inches apart. Oblique insertion of the needle from above is the best method of injecting into thin skin. It is important that a wheal is visible or palpable after injection; if not, the injection must be repeated at a site nearby. Injections should not be given too superficially.

The Reaction

It is generally accepted that in a positive reaction, antigen-antibody fixation takes place, which results in the liberation of histamine-like substances by the sensitized cell. The histamine-like substances in turn act as cell poisons and the ensuing inflammation constitutes the reaction. The swelling of the injection site shortly after injection is an anaphylactic reaction and of no significance. This, as well as the swelling caused by the insertion of the needle, will have disappeared after 24 to 48 hours. The tuberculin test is read after 72 hours.

The following clinical signs have to be looked for:—

(*a*) oedema—this is a very reliable symptom, it may be diffuse or circumscribed;
(*b*) pain—painfulness of the injection site is not detectable in all positive reactions. The intensity of pain evidenced depends to some extent on the temperament of the animal;
(*c*) local erythema, exudation and necrosis,—when present, usually point to a specific reaction;
(*d*) local heat—this does occur in positive reactions but is difficult to detect especially during summer;

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(*e*) swelling of the prescapular lymphnode—this can be determined by comparison with the same node on the other side;

(*f*) engorgement of the surrounding lymphatics—this can be seen in severe reactions;

(*g*) classically positive reactions are usually adherent to the subcutaneous tissues;

(*h*) two-thirds of all cattle reacting positively develop a rise in body temperature;

(*i*) tuberculin syndrome—a transitory disturbance of the general condition of the animal, diminished rumination and muscular tremor—this occurs rarely and, when present, can be noticed during the first 24 hours.

The extent and thickness of the swelling are the only factors which can be measured objectively. Measurements must be taken carefully since a difference of even 1 mm. may decide whether an animal is positive or negative. The measurement must be taken across the breadth of the swelling, i.e. the swelling must be at the apex of the skin-fold when picked up for measurement. A mistake can be made by having the swelling at the side of the picked-up skin-fold, with the result that the callipers register the depth of the swelling as well as the thickness of a piece of normal skin.

Interpretation of the Results, with the Aid of Formulae.

Most countries engaged in the eradication of bovine tuberculosis have introduced standard formulae as a guide to the interpretation of reactions. The increase in the thickness of the skin-fold is the cardinal criterion and can be recorded easily. Not only the measurable but also the visible and palpable changes must be taken into account.

To illustrate the application of a formula the following example is given:—

On seven different dairy farms a total of 941 cattle was tested with the present standard tuberculin, either after the milk from the farm had been found to contain tubercle bacilli, or cases of tuberculosis had been detected at meat inspection. Of this number, 427 cattle were found infected with tuberculosis. According to formula I (see below) their reactions were divided into the following groups:

	Increase in skin-fold measurement	Number of cattle	Percentage of total
Negative range	0–1.9 mm.	12	2.8%
Suspicious range	2–3.4 mm	15	3.5%
Positive range	3.5–4.9 mm	40	9.4%
	5–7.9 mm	83	19.4%
	8–12 mm	116	27.2%
	more than 12 mm	161	37.7%
Total		427	100%

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The twelve non-reacting cattle in the first group were detected as tuberculous by clinical examination. This should always be undertaken as an important part of the investigation into the status of tuberculosis in a herd. Most of these cases had chronic tuberculous mastitis; some, advanced tuberculous pneumonia, and one case showed a suppurating lymphadenitis of a retropharyngeal lymph node.

The fifteen cattle in the suspicious group showed positive reactions at a retest eight weeks later and were then classified as positive.

Out of 427 infected cattle 110 were subjected to careful post-mortem examination at Onderstepoort, and tuberculous lesions found in all but three cases. The ages of these animals ranged from three to eighteen years.

Of the 107 tuberculous cattle 14 per cent had exhibited reactions of less than 5 mm. skin-fold increase at their first tuberculin test. The reactions were grouped as follows:—

4 reacted with 0 –1.9 mm. increase
3 ,, ,, 2 –3.4 mm. ,,
8 ,, ,, 3.5–4.9 mm. ,,

Eight of these fifteen cattle showed generalized tuberculosis, while among the remaining 92 there were only 18 generalized cases. The whole group of 427 tuberculous cattle contained 15.7 per cent of cases showing a low reactivity.

The following formula should be used for the interpretation of reactions in herds where tuberculosis has been established previously.

FORMULA I

Skin-fold increase	Pain. Consistence of swelling. Other changes	Interpretation of reaction
Below 2 mm.	Not tender. Hard, circumscribed. No other changes.	Negative
2–4 mm.	Almost painless. Circumscribed or slightly diffused. No other changes.	Suspicious
3–4 mm.	Almost painless to painfull. Diffuse, oedematous. With or without other changes.	Positive
4 mm. or more	Painless or tender. Circumscribed or diffuse. With or without other changes.	Positive

Where the presence of tuberculosis in a herd has not been established previously, difficulties may be experienced in deciding on the aetiology of the reactions. Until the specific cause of sensitization in a herd has been established, the following formula should be applied:

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FORMULA II

Skin-fold increase	Consistence of Swelling, Pain. Other changes	Interpretation of reaction
0–3 mm.	Hard, circumscribed. Painless. No other changes.	Negative
3–6 mm.	Hard or slightly oedamatous; circumscribed or slightly diffuse. Painless or almost painless. No other changes.	Suspicious
6 mm. or more	Hard or oedematous; circumscribed or diffuse. Tender or painless. Without or with other changes.	Positive

This formula should only be used for the initial test of a herd or an animal, and any retests interpreted, either according to the first formula, or by using a formula for nonspecific sensitivity, given below.

The aim should always be to get to know the true state of affairs as soon as possible. One should endeavour to test and slaughter at least some of the reacting animals. The herd history is of primary importance. In the case of a self-contained herd with rare introductions and no outside contacts, indefinite reactions showing no more then 6 mm. skin-fold increase, should receive careful consideration.

In conducting a test, the reaction of each individual member of the herd should be read and recorded, before attempting to interpret the status of the herd as a whole.

THE ACCURACY OF THE TUBERCULIN TEST

The results of tuberculin testing can be assessed with a fair degree of accuracy. In the above example 97% of negative diagnoses and the same percentage of positive diagnoses were correct. About the same measure of accuracy is reported by several overseas investigators. The test is most reliable when applied to animals between the ages of three months and three years. Infected calves under two months old often do not react and should be retested later.

According to the writer's experience, a retest of the negative part of an infected herd often detects additional reactors—a finding which can be most important in the eradication of tuberculosis in that particular herd.

In general the number of false positive reactors will be very low in a herd with a high incidence of tuberculosis, while the number of false negative reactors will be relatively high. In contrast, in a herd free or almost free from tuberculosis, the number of false positive and doubtful reactions increases, and the percentage of false negative cases will be low.

FLUCTUATION OF INTENSITY OF REACTION

The allergic sensitivity may vary during different stages of the disease. It is a well established fact that the degree of reactivity bears

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little or no relation to the extent of dissemination of tuberculous lesions in the body. It is however, related to the age of the infection. During the period of primary infection the body is highly reactive or allergic, consequently the finding of severe reactions in a herd points to a recent infection; under such conditions one expects to find open cases. An animal showing a severe reaction should not be destroyed for diagnostic purposes as the lesion may still be very small and difficult to detect.

Chronic tuberculosis of a single organ is often associated with a low degree of reactivity. Advanced cases of tuberculosis sometimes show no skin reactivity at all, possibly due to all antibodies being fixed in the tissues. Most of these cases, however, show tuberculous pneumonia, pleuritis, or mastitis and a loss of condition or emaciation. These cases can be detected clinically. Complete desensitization often seems to be associated with chronic tuberculous mastitis. Serological tests can assist in the recognition of advanced disseminated cases. Most of them show a high positive titre in the haemagglutination and haemolysis reactions.

Other factors which diminish reactivity are reviewed below.

Retest and Tests for Non-specificity

Although the influence of a recent tuberculin test on a second test is frequently over-estimated, an interval of seven to eight weeks between two tests should be allowed. This applies particularly to retests for the recognition of non-specific factors. In instances where a retest is applied to assess the result of control measures (e.g. disposal of all reactors or isolation of clean stock), an interval of three months is advisable, to avoid testing animals in the pre-allergic phase.

The following procedures are recommended for the detection of non-specific sensitization:

(*a*) retest using mammalian tuberculin two months after the first test;

(*b*) comparative test—simultaneous use of avian and mammalian tuberculin;

(*c*) use of specific tuberculins prepared from mycobacteria other than *M. tuberculosis*;

(*d*) testing with 500–1000 units of tuberculin per dose;

(*e*) double test—a second dose of tuberculin into the same site after twelve hours;

(*f*) repeating the test immediately on the opposite side of the animal;

(*g*) ophthalmic or conjunctival test;

(*h*) subcutaneous or temperature test;

(*i*) Stormont test—double intradermal test at an interval of one week.

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The five last mentioned methods (*e–i*) have proved to be of little value and cannot be recommended for use. Tuberculin-like extracts have been prepared from M. *phlei*, *smegmatis*, *lacticola* and *rabinowitsch*, but their use did not elucidate the confusing issues encountered in South Africa, *e.g.* skin lesion reactors and sensitization by the human type of M. *tuberculosis*. Comparative experimental work is in progress on a tuberculin-like skin lesion antigen, prepared from skin lesion organisms isolated by M. C. Lambrechts (1956).

The use of avian tuberculin is a most valuable aid, in so far as it permits the interpretation of reactions caused by M. *avium*, M. *johnei*, and other non-tuberculous sensitizing agents.

Formula III is recommended for the interpretation of reactions obtained after comparative tests.

FORMULA III

Reaction with Bovine PPD	Reaction with Avian tuberculin	Interpretation of comparative tests
1. Negative 2. Suspicious 3. Negative	Suspicious Positive Positive	Suspicious for avian tuberculosis, Johne's disease or other non-specific sensitization.
4. Suspicious 5. Positive 6. Positive	Negative Negative Suspicious	Suspicious for bovine tuberculosis, skin lesion or sensitivity due to human tuberculosis.
7 Suspicious 8. Positive	Suspicious Positive	(*a*) Suspicious for bovine tuberculosis when the increase to bovine tuberculin is more than 5 mm. greater than the increase to avian tuberculin. (*b*) Group- or non-specific sensitivity if reaction to both tuberculins are similar.

In the following formula the relationship between the findings of the simultaneous test, and an infection with M. *tuberculosis* var. *bovis* is given:

FORMULA IV

Increase to bovine PPD more than 5 mm. greater than to avian tuberculin	Positive for bovine tuberculosis
Increase to bovine PPD 3–5 mm. greater than to avian tuberculin	Suspicious for bovine tuberculosis
Increase to bovine PPD not more than 3 mm. greater than to avian tuberculin	Negative for bovine tuberculosis

The described methods of evaluating the results of a test should be applied on a herd basis.

The overlapping of the sensitivity caused by skin lesions and the human type of tuberculosis may produce reactions with bovine PPD which are indistinguishable from specific reactions.

A detailes anamnesis clinical examination, test slaughter and the history of tuberculin testing of the herd, will assist in the detection of

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the causative agent. If it is established that *M. tuberculosis* var. *bovis* is not the cause of reactions in a herd the following factors should be regarded as determining the existence of group-specific or non-specific sensitivity:

(*a*) clinical evidence of skin lesions;
(*b*) clinical or radiological evidence of tuberculosis in persons in contact with the herd (also human tuberculosis in dogs or cats);
(*c*) clinical or serological evidence of Johne's disease;
(*d*) clinical or pathological evidence of avian tuberculosis on the farm (poultry or pigs);
(*e*) the presence of several cattle showing reactions like the first three combinations in formula III.

If group-specific sensitivity is not established, any increase of more than 6–7 mm. to bovine PPD is suspicious of a specific infection.

TUBERCULOUS NON-REACTORS

The following is a short survey of the conditions which can be responsible for a negative tuberculin reaction, in a bovine infected with *M. tuberculosis* var. *bovis*:—

(*a*) The pre-allergic stage or allergic incubation period (this should not be confused with the actual incubation period).

The period between infection and detectable allergy can vary from eight to sixty-five days, but is usually about twenty-one days.

(*b*) Advanced tuberculosis:

In 1–2 per cent of cattle affected by generalized or advanced tuberculosis of a particular organ, no skin reaction develops probably because of a collapse of the allergic defence mechanism. It is therefore, advisable to regard all older cows in herds with a high infection rate as possibly infected.

(*c*) Inactive tuberculosis:

The frequency of the occurrence of this condition is often under-estimated. In this case the cattle, by virtue of a high resistance to tuberculosis, are affected by small, mostly primary, foci which are encapsulated and rendered inactive. The encapsulated foci do not stimulate antibody production to any extent. The reactions in some of these animals indicate a low, fluctuating sensitivity.

(*d*) Radical cure:

According to recent experimental results obtained at Onderstepoort, it is possible to cure a considerable percentage of reactors completely by prolonged chemotherapy. After the destruction or degeneration of all tubercle bacilli the allergic response to tuberculin disappears.

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(*e*) Artificial desensitization:

After repeated tuberculin tests at short intervals, or after the injection of 2 ml. of tuberculin into a reactor, the reactivity decreases or disappears for some weeks. After the subcutaneous injection of 5 ml. of tuberculin the reaction becomes negative for five to six months.

FACTORS DIMINISHING THE REACTIVITY

These may be due to:—

(*a*) Injection of hormones:

It was proved that the daily injection of cortisone into a reactor for a few days prior to reading the test could depress the reaction very considerably. The same probably applies to A.C.T.H. and the synthetic substitutes of cortisone.

(*b*) Other drugs:

The daily dosing of tuberculous cattle with isoniazid, will render a high percentage of reactors negative to the test after a time, and will depress the reactivity of all treated reactors to different degrees. The injection of atropin, ergotin, and drugs causing vasoconstriction into the reaction site will reduce any oedema that may be present. No other drug, including the antihistamines, could be proved to have a marked desensitizing or depressing effect.

(*c*) Local manipulation for fraudulent purposes:

Many different ways of preventing a positive reaction have been tried by owners, e.g. the application of ice, snow, loam, hair tonic, spirits, vinegar, lysol, petrol, massage, cupping, ironing and clamping. These measures will not reverse a positive result, though some will intensify a positive reaction.

(*d*) Miscellaneous conditions.

Under the following circumstances a decrease in responsiveness to tuberculin is possible.

Parturition and puerperium—for a period of three weeks prior to, and three to six weeks after calving.

Malnutrition.

Exhaustion.

Transport over long distances.

No specific diseases are known which will influence a positive reaction.

REACTIONS IN NON-TUBERCULOUS CATTLE.

Different terms are used to describe this condition, e.g. "non-specific reactor", "non-tuberculous tuberculin sensitivity", "non-visible lesion reactor", "no lesion reactor". In order to simplify reporting on tests care should be exercised in using these terms. The terms "no lesion reactor" should not be used, since it is difficult to be certain that no lesion whatsoever occurs in a bovine carcase. "Non-

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visible lesion reactor" (NVL) could only be stated after a very careful post mortem examination had been performed. An autopsy should include the serial cutting of all lymph nodes of the head, lungs, liver and intestines, and body lymph nodes. Primary foci in organs other than these are exceptional. The lungs should be thoroughly palpated and cut into slices. Any suspicious lesions should be submitted to Onderstepoort. Trials overseas have shown that 50 per cent of the so-called NVL-reactors originating from tuberculous infected herds, had lesions of tuberculosis which were confirmed by histological, cultural or biological investigation.

Reactions can be subdivided according to their origin:

(*a*) TYPE SPECIFIC: reactions caused by the bovine type of M. *tuberculosis*.

(*b*) GROUP SPECIFIC: all reactions caused by mycobacteria other than the bovine type, e.g. human and avian type, M. *johnei*, and skin lesion acid fast organisms. This term is synonymous with "par-allergy".

(*c*) NON-SPECIFIC: the sensitive conditions other than those mentioned in the two previous categories. Possible causes are physiological conditions and bacteria other than mycobacteria. Synonym: hetero-allergy.

GROUP-SPECIFIC REACTIONS

(*a*) M. *tuberculosis* var. *hominis*. Sensitization of cattle by a labourer infected with the human type of organism frequently occurs. Cattle have a strong resistance to infection with this type and the lesions heal naturally. A marked temporary reactivity to mammalian tuberculin develops, however. This sensitivity disappears with removal of the source of infection from the herd.

The greatest increase in skin-fold thickness (6–10 mm.) is noticeable eight to ten weeks after infection. Four months later the reaction falls within the suspicious range (2–4 mm.), and within nine months most of the cattle will be negative again. Sensitization may rarely last for two to four years.

Where herds, suspected of being infected with human tuberculosis, are retested after eight weeks it will be observed that some positive cases have become doubtful reactors; doubtful cases negative; and some previously negative cases, positive.

(*b*) *Mycobacterium avium*. Outbreaks of avian tuberculosis are rare in the Union of South Africa and consequently the sensitization of cattle by M. *avium* seldom occurs. If it is suspected, a comparative test with avian and mammalian tuberculin will clear the situation. Sensitivity disappears six to twelve months after removal of the source.

(*c*) *Mycobacterium johnei*. (*Paratuberculosis*). This disease occurs mainly in herds containing imported stock. Affected animals

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are inclined to show doubtful reactions to mammalian tuberculin. Avian tuberculin should be used comparatively. In differentiating it from avian tuberculosis, the age of the animal should be taken into consideration. Sensitization and clinical symptoms occur mainly in adult animals in the case of paratuberculosis, while sensitisation by the avian bacterium is seen mostly in young stock.

(*d*) *Skin lesion* (*Dermatitis nodosa infectiosa*). The lesions caused by these organisms should not be termed "skin tuberculosis".

In certain areas, such as the Western Cape Province, these infections are the most frequent cause of non-tuberculous sensitivity. The finding of one clinical skin lesion case can explain positive reactions in other animals without lesions in the same herd. In some herds swellings up to 20 mm. increase are not uncommon. The age group between two and three years seems to be favoured.

When skin lesions are present they remain detectable for prolonged periods, although the animal shows tuberculin sensitivity for periods varying from six months to three years. The sensitivity is dependent on the activity of the skin lesions. After surgical removal of all lesions, or if they become calcified or fibrosed, the reactivity disappears. Calves under one and a half years old are usually free of lesions and do not react. Simultaneous infection with tubercle bacilli and skin lesion organisms is possible, and can make the interpretation of a herd test most difficult. The human type of *M. tuberculosis* shows the closest antigenic relationship to the acid-fast skin lesion organisms. Comparative testing with avian tuberculin has proved of little value in South Africa. Isolation, disposal, or surgical treatment of skin lesion reactors in tuberculosis free herds is advisable.

(*e*) *M. phlei*, *M. ulcerans*, *M. balnei* and other saprophytic mycobacteria from grass and pastures have been proved not to be likely sources of sensitization.

NON-SPECIFIC REACTIONS.

Many factors have been named in this group, but the rôle of each one of them as a sensitizing agent has been disputed by certain workers. The following is a list of conditions likely to cause sentitisation:

Sex hormones—cows advanced in pregnancy and during heat may be sensitive. Oxen, mainly between two and four years of age generally react irregularly. Doubtful reactions occur three times more frequently in tuberculosis-free oxen than in cows. Any specific or non-specific sensitivity is intensified.

Actinomycosis.

Actinobacillosis.

Fasciolasis.

Peritonitis due to foreign bodies.

Chronic bacterial infections including brucellosis.

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The following factors are unlikely to have an influence but have been incriminated by certain workers:

Abscesses in general.
Echinococcosis.
Cysticercosis.
Pneumonia.
Mastitis.
Nephritis.
Leucosis.
Calcium deficiency.
Demodicosis.
General hypersensitivity to foreign proteins.
Hyperthyroid type of constitution.
Lumpy skin disease.
Complete change of environment.

The following possible causes of swellings are produced by factors outside the animal:

Bites of flying insects.
Skin parasites.
Skin injuries due to clipping or shaving.
Unclean syringes or needles.

Manipulations in order to produce a reaction for fraudulent purposes. (Done in countries where a high compensation is paid for reactors).

Non-specific reactions have the following characteristics in common:

The swelling is harder, more circumscribed, less oedematous, flatter, painless.

The subcutis is not involved and the prescapular lymph node is not enlarged. The age group 2—4 years is favoured.

Calves are seldom involved in contrast to specific reactions.

Examination after 4 days may also aid in the distinction between specific and non-specific reactions. In most cases the latter show a distinct decrease on the 4th day as compared to the third day, which does not happen in specific reactions.

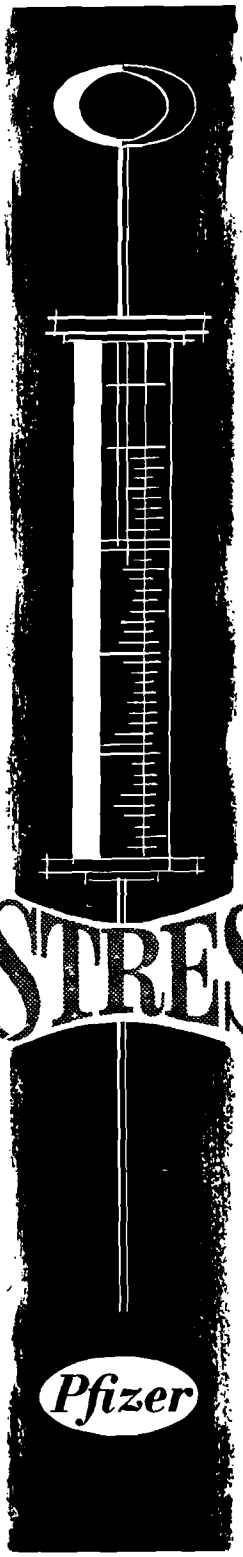
One should not be influenced to interpret possitive reactions too leniently by calling them non-specific.

References to literature cited in this article can be obtained from the author.

ACKNOWLEDGEMENTS.

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THE RESPONSE OF MERINO SHEEP TO VARIOUS DIURETIC DRUGS

R. Clark.*

Introduction.

As little appears to be known of the response of ruminants in general, and sheep in particular, to the diuretic drugs commonly used on man and dogs, it was decided to test a series of such drugs on merino sheep with a view to selecting those which could be recommended for general use on these animals. In this regard sheep could be excpected to show considerable species peculiarities, when compared to monogastric animals, in view of the following factors:—

(i) The possible degradation of orally administered drugs by the runinal flora.

(ii) The effect of such drugs on the flora.

(iii) The fact that these animals normally excrete an alkaline urine.

(iv) The exceptional powers of urine concentration shown by the ovine kidney.

General Method.

A group of merino wethers of 40 to 50 Kg. body weight were reserved for the trials and kept on a diet of lucerne hay *ad lib.* Four sheep were used in each trial during which they were fitted with faeces bags and housed in individual metabolism cages. Food was restricted to 1 Kg. lucerne hay per day. As this was always completely consumed, variations in urine output due to food intake were eliminated. Water was supplied *ad lib.*

In most trials, first two sheep were treated and the other two left as controls. After an interval of a few days the treatment was reversed. The results given are then the averages obtained from all four sheep, each acting both as an experimental animal and as a control. In this way individual variations and climatic factors were counterbalanced.

I. ACETOZOLAMIDE SODIUM

Acetozolamide is 2-acetylamino-1,3,4,-thiadiazole-5-sulphonamide. It is a carbonic anhydrase inhibitor with a diuretic effect due to its action in slowing down the reversible hydration of carbon dioxide and dehydration of carbonic acid in the kidney tubule resulting in the loss of HCO_3 which carries with it base, mainly sodium and potassium, and water. It is therefore a diuretic and urine alkalinizer. It is used in human medicine, e.g. "Diamox", (Lederle.) The material used was kindly supplied, for experimental purposes only, by the American Cyanamid Company under the name "Vetamox". Two packings

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were used, the one for oral use containing 25% of the active ingredient and a pure sample for parenteral use.

Working with normal dogs, Maren, Wadsworth, Yale and Alonso[1] found that acetozolamide caused increased urinary excretion of bicarbonate, sodium, potassium and water, provided the plasma concentration of the drug exceeded 2 micrograms per ml. The magnitude of the response was almost independent of dosage above the rate of 5 mg. per Kg. body weight. When dosed at this level, dogs showed a diuretic phase lasting about six hours accompanied by the development of a acidosis. This was followed by a compensatory phase of sodium retention with restoration of normal balance within 24 hours. Repeated daily dosing elicited the same response on each occasion. The response was lessened or lost in proportion to the acidosis so that larger or more frequent dosing had little effect beyond prolonging the recovery phase.

RESULTS.

TABLE 1.

THE EFFECT OF A SINGLE INTRAVENOUS INJECTION (2 mg./Kg.)

	0–6 Hours		24 Hour Total	
	Control	Treated	Control	Treated
Urine Volume (1) ..	0.34	0.44	1.12	1.14
Urine S.G.	1042	1040	1045*	1046*
Na Output (m.eq.)..	5.6	42.9	15.6	50.7
K Output (m.eq.) ..	151	152	452	493
Water Intake (1)....			3.25	3.58

*=Figure for 6 to 24 hour sample.

Blood samples were taken 6 and 24 hours after injection and the following determinations made:—plasma Na, K, Mg, Ca, Cl, HCO_3, inorganic P and urea. No deviations from the normal ranges were found.

TABLE 2

THE EFFECT OF A SINGLE ORAL DOSE (10 mg./Kg.)

	0 to 6 Hours		24 Hours Total	
	Control	Treated	Control	Treated
Urine Volume (1) ..	0.30	0.38	1.00	1.20
Urine S.G.	1050	1050	1048*	1046*
Na Output (m.eq.)..	9	42	18	70
K Output (m.eq.) ..	154	158	526	553
Water Intake (1) ..			3.26	3.05

*=Figure for 6 to 24 hour sample.

Owing to previous negative findings, blood analyses were omitted.

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The Effects of Repeated Oral Dosing
5 mg. per Kg. Twice daily

This trial was carried out on only one replica, i.e. two treated sheep and two controls. The dose was given at 9 a.m. for four consequetive days. Urine electrolyte determinations and blood analyses were not performed. The figures obtained are shown in Table 3.

Table 3

	Controls	Treated
Average 24 Hour Urine Volume (1)	0.97	1.02
Average 24 Hour Water Intake (1)	2.02	2.81
Average Urine S.G.....	1044	1041

The Effects of Repeated Oral Dosing
25 mg. per Kg. Twice Daily

This trial was initiated in view of the almost negligible increase in urine volume shown in Table 3 but had to be abandoned as three sheep all went off their feed in from 24 to 48 hours after the commencement of dosing. The volume of urine fell to below that of the controls. All three sheep recovered their appetites spontaneously within 48 hours after the cessation of dosing.

The Effect of Continual Dosing via the Drinking Water

The experiment was conducted in the same way as Experiments 1 and 2 and was continued for three consecutive days. Acetazolamide was added to the drinking water at the rate of 0.25 gm. per litre. With an average water intake of some 3 litres per day, this gave a dosage level of 15 to 20 mg. per Kg. per day.

Table 4

	Treated				Controls
	Day 1	Day 2	Day 3	Average	Average
Urine Volume (1) ..	1.08	0.95	0.95	0.99	1.03
Urine S.G.	1056	1054	1050	1053	1052
Na Output (m.eq.) ..	75	17	34	42	11
K Output (m.eq.)	576	496	500	524	601
Water intake (1)	3.61	2.00	3.12	2.91	3.20

Blood samples taken from the treated sheep at the end of the experimental period showed normal plasma electrolyte concentrations.

Discussion.

The results obtained are in conformity with those of Maren *et al.* in that a single dose of acetazolamide caused a marked increase in sodium excretion, especially over the first 6 hours, but the increase in water excretion was insignificant from a clinical point of view. This

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may be attributed to the power of urine concentration possessed by the ovine kidney (urine samples with an S.G. up to 1.065 were frequently encountered). The loss of effect on repeated dosing is clearly seen in Table 4 where the sodium output on the first day of treatment was threefold the average shown on the two subsequent days.

Numerous samples of urine from treated sheep were tested for the presence of sugar and albumen with negative results.

CONCLUSION

Acetazolamide would not appear to be a suitable diuretic for use in sheep as it fails to increase water excretion in normal sheep to any material extent despite its known action of increasing soidum excretion. Excessive oral dosing causes anorexia presumably through inhibition of the ruminal flora.

II. MERCURAMIDE WITH THEOPHYLLINE.

The preparation used was "Neptal" (M and B) described as an association of mercuramide and theophylline for intramuscular injection containing mercuramide 9.20% w/v and theophylline 4.5% w/v.

In the first trial a dose of 0.25 cc. Neptal i.m. was used. Of a group of four sheep, two were injected and two left as controls. The following week the groups were reversed. Full data were collected for six days after each injection. The diuretic action persisted for 72 hours. The average figures for the first four days are given below:—

TABLE 5

THE EFFECT OF 0.25 C.C. NEPTAL i.m.

		Days after injection			
		1	2	3	4
Water Consumption (litres)	Treated	2.79	2.91	3.72	3.32
	Control	2.72	2.70	3.23	3.33
Urine Volume (litres)	Treated	1.27	1.07	1.16	0.91
	Control	1.03	0.91	0.95	0.91
Urine S.G.	Treated	1040	1043	1037	1046
	Control	1047	1045	1046	1049
Blood Urea N (mg%)	Treated	26.7	30.3	30.4	—
	Control	26.2	26.2	26.2	—

All figures represent averages from four sheep.

As will be seen there was a mild diuresis reaching a maximum on the 3rd day and passing off on the 4th day as reflected by the differences in urine volume and S.G. between the treated and control animals.

Unfortunately blood samples were only taken during the first three days but the analyses indicated urea retention after treatment.

All four sheep showed glycosuria after treatment as shown by the "Test-Tape" (Lily) technique. Details are shown in Table 7. Simultaneous blood analyses revealed no increase in blood sugar level, indicating that the drug had interfered with glucose reabsorption.

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In view of the production of glycosuria and the indication of urea retention, together with an almost insignificant increase in urine volume it was apparent that "Neptal" would not prove a suitable diuretic for sheep. Tests were made with higher doses, namely 0.5 and 1.0 cc.. In both these tests two experimental animals and two controls were used.

TABLE 6

Dose c.c.	Group	Water Intake	Urine vol.	Urine S.G.	Blood Urea N.
0.5	Control ..	2.90	0.74	1052	31.8
	Treated ..	3.44	1.38	1032	43.9
1.0	Control ..	2.65	1.08	1046	31.5
	Treated ..	3.90	1.39	1032	55.5

Figures represent averages for two sheep over the period of reaction. See text.

In the first trial (dose 0.5 cc) the diuretic action lasted for nine days. The figures given in Table 6 represent the averages over this period. Both treated sheep again showed glycosuria and elevated blood urea nitrogen. The uraemia persisted for 7 days.

The animals receiving 1.0 c.c. showed diuresis for only four days and the urine output was no greater than when 0.5 c.c. was given. Again both sheep showed glycosuria and hyperuraemia, the maximum individual figure reached being 64.4 mg. urea N % on the second day after injection. The blood urea was normal on the eighth day.

TABLE 7

GLYCOSUREA FOLLOWING "NEPTAL".

	Days after injection					
Dose	1	2	3	4	5	6
1.0 c.c. ..	—	++	++	++	++	—
	—	+	++	+	—	—
0.5 c.c. ..	++	++	+	—	—	—
	—	+++	++	++	+	—
0.25 c.c...	—	—	—	Tr.	—	—
	—	Tr.	+	—	—	—
	—	++	+	++	+	—
	—	+	+	—	—	—

The sugar content of several samples of urine showing a + + test was determined by Meyer's modification of the Benedict test and found to be between 250 and 300 mg. %., i.e. in agreement with the "Test-Tape" result.

As the urine was not preserved over the 24 hour collection period, the true figure was probably considerably higher.

CONCLUSIONS

Mercuramide would not appear to be a suitable diuretic for sheep as it caused glycosuria and urea retention without appreciably increasing urine volume.

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III. MERSALYL WITH THEOPHYLLINE.

The preparation used was "Injection of Mersalyl B.P. (M & B)" containing 10% mersalyl and 5% theophylline.

TABLE 8

THE EFFECT OF 1 C.C. MERSALYL I.V.

	Days after Treatment			Controls Average
	1	2	3	
Urine Volume (litres) ..	1.64	1.46	1.40	1.37
Urine S.G.	1034	1040	1040	1040
Na Output (m.eq.)	80	28	23	20
K Output (m.eq.)	698	680	695	690
Cl. Output (meq.).....	194	203	165	184

There was no rise in blood urea in the treated sheep. One sheep showed a mild glycosuria on the second, third and fourth days after injection.

It will be noted that there appeared to be an initial increase in sodium loss followed by a compensatory retention.

CONCLUSIONS

Although mersalyl did not cause the same degree of glycosuria and urea retention shown by mercuramide, it caused only a 20% increase in urine volume over the first 24 hours after administration.

IV. HYDROFLUMETHIAZIDE.

The preparation used was "NaClex" (Glaxo) which is put up as tablets each containing 50 mg. of the active ingredient.

This drug is claimed to cause inhibition of the renal reabsorption of water, sodium and chloride, and to have little carbonic anhydrase inhibitory action. Consequently it is claimed not to cause acidosis nor to exhibit a progressive loss of efficacy on repeated administration.

TABLE 9

THE EFFEAT OF 50 MG. "NACLEä" TWICE DAILY GIVEN ORALLY FOR THREE CONSECUTIVE DATS

	Days of Treatment			Controls (Average)
	1st	2nd	3rd	
Urine Volume (litres) ..	1.52	1.47	1.26	1.13
Urine S.G.	1038	1037	1040	1047
Na Output (m.eq.)	50	14	22	6
K. Output (m.eq.).....	697	670	616	656
Cl Output (m.eq.).....	250	190	161	170
HCO_3 Output (m.eq.) ..	358	368	358	322
Water Intake (litres) ..	3.13	3.78	3.36	3.54

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After each spell of dosing, blood samples were taken from the treated and control animals and the following factors determined viz. Na, K, HCO_3, Cl and Urea N. No differences were found between the two groups. The urine of the treated sheep was consistently free of sugar.

CONCLUSIONS

The average urine output was increased over that of the controls by 35% on the first day of treatment but the response decreased progressively. A similar trend was seen in the Na, K and Cl outputs.

V. AMINOPHYLLINE

Aminophylline was administered intravenously at the rate of 250 mg. per dose given twice daily at 9 a.m. and 3 p.m. Each trial was conducted over 24 hours following the initial dose. The results are summarized in Table 10.

TABLE 10

	Urine Vol. (l)	Urine S.G.	Na Output	K Output	Cl Output	Water Intake
Controls	1.15	1041	2	623	149	3.5
Treated	1.44	1036	37	620	166	3.4

CONCLUSIONS

Despite the relatively large dose given, the urine volume was increased by only 25%. There appeared to be a selective increase in sodium and chloride excretion.

VI. GLUCOSE

The fact that glycosuria was not necessarily accompanied by polyuria in sheep had been noted in previous experiments on alloxan diabetes in that species. Figures over 16 sheep/days, when the animals had been passing urine containing from 2 to 3.8% glucose showed an average 24 hour urine output of 1.20 litres which is much the same as that of normal sheep acting as controls in the present series of experiments.

In order to confirm this observation, two sheep were each injected with 20 gm glucose in 100 c.c. saline intravenously. Their average urine output during the succeeding 24 hours was 1.04 litres as compared to 1.15 litres from the controls and 1.10 litres shown by the two experimental animals while acting as controls in a previous experiment. It is therefore evident that the treatment had little or no effect on urine output.

The blood sugar levels of the two sheep were raised from 43 and 55 mg. % immediately before the injection to 264 and 256 immediately after. Three hours later they had both returned to 55 mg %. The urine collected during these three hours contained 2.0 and 1.6% glucose respectively. The samples collected during the subsequent

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three hours both contained 0.2% glucose. The combined volumes of these two samples were 364 and 366 c.c. respectively. It will be seen, therefore, that the rate of urine output was not correlated to the degree of glycosuria.

VII. UREA

Two sheep were injected intravenously with 10 gm. urea in 100 c.c. saline. Both showed marked haemoglobinuria afterwards and blood samples showed haemolysis. Six hours after the injection the plasma of one sheep was clear of haemoglobin but that of the other was still coloured.

The blood urea levels were raised from an average of 66 mg.% before the injection to 128 mg.% immediately after. During the 24 hours after injection the sheep passed 1.29 and 1.93 litres of urine as compared to 1.10 and 1.08 litres respectively when acting as controls. The injection of 10 gm. of urea therefore raised the urine volume by 17% in the one case and 79% in the other but its use could not be recommended in view of the haemolysis. This might be overcome by reducing the concentration, but this would entail giving large volumes of fluid intravenously, a procedure often contra-indicated when duiretic action is required.

General Conclusions

None of the drugs tested appeared to give a clinically satisfactory diuresis.

Summary

1. A series of diuretic drugs was tested on sheep. These were:— acetazolamide, mercuramide, mersalyl, hydroflumethiazide, aminophylline and glucose and urea intravenously.
2. None of these drugs increased the average urine output by more than some 25%. No clinically satisfactory diuretic for sheep was found.
3. Large doses of acetazolamide caused anorexia. Mercuramide caused glycosuria and urea retention.

Reference

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THE USE OF A "NEGUVON-ASUNTOL" MIXTURE AS AN ANTHELMINTIC FOR SHEEP

C. M. Meldal-Johnson*, G. L. Muller** and R. J. Thomas.***

A number of reports has been published on the anthelmintic activity of the organic phosphorus compounds, "Neguvon" (Bayer L13/59) and "Asuntol" (Bayer 21/199) and these have been summarized by Reinecke and Schutte[7]. Most of these reports are concerned with the action of the drugs against parasites of cattle. In sheep, Gordon[4] stated that following prestimulation "Neguvon" was highly effective against *Haemonchus contortus* at 55 mg. per Kg. and against *Trichostrongylus colubriformis* at 110 mg. per Kg., but did not give consistent results against *Oesophagostomum columbianum*. "Asuntol" was highly effective against *H. contortus*, *T. colubriformis* and *O. columbianum* without prestimulation at 6 mg. per Kg., but at this dosage it was considered too toxic for normal use. Herlich and Porter[6] reported that "Asuntol" at dosage rate of 12.5—25 mg. per Kg. was very effective against *Haemonchus*, *Ostertagia*, *Trichostrongylus*,*Cooperia*, *Trichuris* and *Strongyloides* spp., but not against *Nematodirus* or *Bunostomum* spp., very slight toxic symptoms being observed in lambs at the higher dose. Stampa[12] reported that "Neguvon" was effective against *Haemonchus* and *Nematodirus* species, and that "Asuntol" was effective against *Haemonchus*, *Ostertagia*, *Trichostrongylus Nematodirus*, *Oesophagostomum* and *Strongyloides* species, but the exact dosage rates used were not specified. This author found the toxic levels for sheep to be 90—130 mg. per Kg. for "Neguvon" and 17.6—20 mg. per Kg., for "Asuntol" under South African conditions. Thus in sheep, "Asuntol" is the more efficient drug but the effective dose is too close to the toxic dose for routine use. To overcome this difficulty mixtures of the two drugs have been tested, and Stampa[12] used a mixture in the proportions of 10 parts of "Neguvon" to 1 part of "Asuntol". This mixture, used following prestimulation, was stated to be effective against *Haemonchus*, *Ostertagia*, *Trichostrongylus*, *Nematodirus*, and *Oesophagostomum* species, but less effective against *Strongyloides*, at a dosage rate of 50–60 mg. per Kg. The lowest toxic dose was found to be 110 mg. per Kg. in the dry area of the Karoo, and 80 mg. per Kg. in the Cape winter rainfall area.

In view of the scarcity of detailed reports on the effectiveness of the drugs in sheep, trials were carried out 3 centres in the Union using the 10:1 "Neguvon—Asuntol" mixture recommended by Stampa[12].

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Experimental Procedure and Results

Trials were carried out on naturally infested sheep, the efficiency of the mixture being assessed on the percentage reduction in faecal egg counts for each species. The McMaster egg counting technique[5] was used throughout, eggs of *Strongyloides*, and *Nematodirus* species being counted separately, and all other species being grouped as "strongyle eggs". The latter group was then divided into species on the basis of differential larval counts from faeces cultures[14]. The mixture used contained 10 parts of "Neguvon" to 1 part of "Asuntol", and was administered in water by mouth, after presttimulationwith copper sulphate.

Trial I

This trial was carried out in the King Williams Town area, a summer rainfall area, during October 1959. A group of 50 ewes grazing on artificial pasture was selected; of these 25 were dosed with 50 mg. per Kg. of the "Neguvon-Asuntol" mixture, and 25 were kept as undosed controls. The dosage was calculated on the individual weight of each sheep. Faeces sampling was carried out three times at 2-day intervals, before and after dosing, and further samples were taken 14 days after dosing. Mean pre- and post-dosing egg counts for each species are given in Table 1.

Table 1

(*a*) Dosed 50 mg. per Kg. "Neguvon-Asuntol".

Mean egg count	Nematodirus	Total strongyles	H.	T.	O.
Pre-dosing	28 e.p.g.	507	22	255	230
Post-dosing	18	122	2	53	66
% reduction in egg count	35	76	91	74	71

(*b*) Controls. Undosed.

Mean egg count	Nematodirus	Total strongyles	H.	T.	O.
Pre-dosing.....	4 e.p.g.	383	9	169	204
Post-dosing	4	321	6	125	188
% reduction in egg count	0	16	33	16	8

H. Haemonchus contortus — O. *Oesophagostomum columbianum.*
T. Trichostrongylus spp. — *N. Nematodirus* spp.
e.p.g.: eggs per gram of faeces.

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Trial 2

The second trial was carried out in the Eastern Transvaal at Ermelo during November, 1959. 30 Merino yearlings grazing on natural veld were divided into 2 groups of 15, of which 1 group received 50 mg. per Kg. of "Neguvon-Asuntol" mixture, the dosage being based on an average live weight of 66 lbs.; the other groups were kept as undosed controls. Faeces samples were taken 3 times at 2-day intervals before and after dosing. Mean pre- and post-dosing egg counts for each species are given in Table 2.

TABLE 2

(*a*) Dosed 50 mg. per Kg. "Neguvon-Asuntol".

Mean egg count	Total Strongylus	H.	T.	O.
Pre-dosing	563	488	100	15
Post-dosing	68	34	7	26
% reduction in egg count	88	92	93	0

(*b*) Undosed controls.

Mean egg count	Total Strongylus	H.	T.	O.
Pre-dosing	494	451	34	10
Post-dosing	401	365	23	13
% reduction in egg count	19	19	32	0

Trial 3

The third trial was carried out in the winter rainfall region of the Western Cape at George, using a group of 12 sheep maintained in a concrete floored pen. The sheep received a ration of lucerne hay and mixed cereals. Each sheep was administered 77 mg. per Kg. of the "Neguvon-Asuntol" mixture, no undosed controls being kept. Faeces samples were taken on the day of dosing and again 7 days later. Approximately one month later the sheep were redosed with 110 mg. per Kg. of the mixture and sampling repeated. Details of pre- and post-dosing egg counts and the percentage reduction in egg count for each species present are shown in Tables 3 and 4.

TABLE 3

Dosed 77 mg. per Kg., "Neguvon-Asuntol".

Mean egg count	S.	N.	Total Strongylus	H.	T.	O.	B.	Os.	Co.	Ch.
Pre-dosing	169	39	8,120	1220	4950	568	81	730	325	240
Post-dosing	124	12	1,720	0	1000	447	17	120	0	137
% reduction	27	70	79	100	80	22	79	84	100	43

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TABLE 4

Dosed 110 mg. per Kg. "Neguvon-Asuntol".

Mean egg count	S.	N.	Total Strongylus	H.	T.	O.	B.	Os.	Co.	Ch.
Pre-dosing	124	3	815	—	147	236	—	400	—	32
Post-dosing	97	0	138	—	3	78	—	24	—	34
% reduction ..	22	100	83	—	98	67	—	94	—	0

S. *Strongyloides papillosus*
B. *Bunostomum trigonocephalum*
O. *Ostertagia* species.
Co. *Cooperia* species.
Ch. *Chabertia ovina*

The results from all these trials are combined in Table 5, and the mean percentage reduction obtained for each species is shown.

TABLE 5

% Reduction	S.	N.	Total Strongylus	H.	T.	O.	B.	Os.	Co.	Ch.
King William's Twn.	—	35	76	91	74	71	—	—	—	—
Ermelo	—	—	88	92	93	0	—	—	—	—
Mossel Bay	27	70	79	100	80	22	79	84	100	43
	22	100	83	—	98	67	—	94	—	0
Mean	25	68	81	94	86	53	79	89	100	22

DISCUSSION

Similar results were obtained at all three centres, and thus the results may be regarded as generally applicable throughout the Union. The use of higher doses in trial 3, namely 77 and 110 mg. per Kg. did not appear to cause any marked increase in the efficiency of the mixture as compared with the lower dose of 50 mg. per Kg. used in trials 1 and 2. While the undosed controls in trials 1 and 2 showed some fluctuation in egg count, these variations were not very wide, and the reductions in egg count obtained in the treated groups must be attributed mainly to the action of the drug.

Varying criteria have been used to evaluate the efficiency of anthelmintics based on reduction in egg count and those adopted by Riek and Keith[9] appear the most satisfactory. These authors considered a reduction in egg count of 80–100% to be highly effective, 60–80% effective and below 60% not effective. Judged by these criteria the 10:1 "Neguvon-Asuntol", mixture used in these trials was highly effective against *H. contortus* and *Trichostrongylus* spp. at 50 mg. per Kg. and against *Ostertagia*, *Cooperia*, and *Nematodirus* spp. at 77–110 mg. per Kg. It was effective against *Bunostomum* spp. at 77 mg. per Kg., but not effective against O. *columbianum*, C. *ovina*, or S. *papillosus*. These results agree with those of Stampa[12] in respect of *Haemonchus*, *Trichostrongylus* and *Ostertagia* spp. However, the high efficiency against

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Nematodirus, *Oesophagostomum* and *Stongyloides* spp. recorded by this author was not confirmed in these trials. A high efficiency was only obtained against *Nematodirus* spp. at 110 mg. per Kg., while the results obtained against *O. columbianum* were extremely erratic, ranging from 0% to 71%, and this cannot be considered at all satisfactory. The rage of effectiveness in these trials, however, is very similar to that recorded by Gordon[4] and Herlich & Porter[6] for "Neguvon" and "Asuntol" separately.

Thus the range of activity of this mixture corresponds approximately to that of micro-fine phenothiazine,[13],[1],[2]. Both drugs are highly effective against *Haemonchus*, *Trichostrongylus*, *Ostertagia*, and *Cooperia* species. Phenothiazine had the advantage of a high activity againts *O. columbianum* and *C. ovina*, while the "Neguvon-Asuntol" mixture has a high activity against *B. trigonocephalum*, and would be the drug of choice against this species. "Neguvon-Asuntol" suffers from the disadvantage that prestimulation is necessary, and its action is therefore less reliable than that of phenothiazine, but it would appear to offer a very useful alternative for use with phenothiazine, particularly where a broad spectrum of activity is required as in the winter rainfall areas of the Union.

No signs of toxicity were observed in these trials, confirming that 50 mg. per Kg. is apparently well within the safety margin, even when dosage is not accurately assessed on individual body weight. Stampa[12] recorded toxic symptoms at 110 mg. per Kg., but in the present trials no toxicity was observed even at this dosage. However, the sheep receiving this dosage were maintained indoors on a ration consisting mainly of lucerne hay, and this may account for the absence of toxic symptoms, as toxicity is apparently closely related to diet[9],[12]. It is of some interest that a dosage rate of 50–110 mg. per Kg. of the mixture, giving a dose of 5–10 mg. per Kg. "Asuntol", caused no toxic symptoms, yet Gordon[4] reported occasional fatalities with doses as low as 6 mg. per Kg. On the other hand Herlich and Porter[6] used up to 25 mg. per Kg. and obtained only very slight toxic symptoms at the highest dose rate. It is apparent that some variation occurs in the toxicity of these organic phosphorus compounds and Montgomery(see Riek[8]) has suggested that this variation may be due to climatic or nutritional differences. Some confirmation for this suggestion is given by the work of Stampa who found that sheep in the winter rainfall area were more susceptible than those in the dry Karoo. It is therefore advisable that considerable care should be exercised in the use of these drugs until more information is available in regard to the conditions which influence their toxicity.

Some comments may be made on the interpretation of the efficiency of anthelmintics based solely on reduction in faecal egg count. This method is widely used because of the ease with which it can be carried out, and the saving in cost which results from the fact that slaughtering is unnecessary. However, the limitations of the method must not be overlooked. Firstly, a considerable fluctuation in egg count may occur from day to day, due partly to natural variations in output and also to the limitations of the sampling technique,[3],[10],[11]

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Secondly, the possibility of spontaneous elimination of infection or "self-cure" must be taken into consideration. In order to minimize these errors it is essential that the comparative pre-dosing and post-dosing egg counts should represent the mean of a series of samples, taken from as large a group of animals as possible over a period of several days. In addition, adequate numbers of undosed controls should be kept to eliminate the possibility of "self-cure" giving false results. In the trials recorded above, variations of up to 30% occurred in successive egg counts for individual species, despite the fact that that these represented mean counts for up to 25 animals. To be of significance, therefore, the reduction in egg count in treated groups should be considerably greater than this figure, and this is one reason why a reduction in egg count of less than 60% is considered not effective. It is therefore apparent that the reliability of trials of this nature is greatly increased if these two principles are observed, namely, the use of a series of pre-dosing and post-dosing egg counts based on reasonable numbers of animals, and the inclusion of adequate controls.

Summary

(1) Three field trials of the efficacy of a 10:1 "Neguvon-Asuntol" mixture against sheep parasitess are reported.

(2) It is concluded that the drug is highly effective against *Haemonchus* and *Trichostrongylus* species at a dose of 50 mg. per Kg. and against *Cooperia*, *Ostertagia* and *Nematodirus* species at 77–110 mg. per Kg. in all cases following pre-stimulation with copper sulphate. The higher dose is, however, rather close to the toxic dose and 50 mg. per Kg. is the normally recommended dose. It was effective against *Bunostomum trigonocephalum* at 77 mg. per Kg., but not effective against *Chabertia*, *Oesophagostomum* or *Strongyloides* species.

(3) The range of activity is thus similar to that of micro-fine phenothiazine and offers a useful alternative to phenothiazine, its chief limitations being the necessity for pre-stimulation, and a lack of activity against *O. columbianum*. It will probably be the drug of choice against *B. trigonocephalum*.

(4) In view of the variations in toxicity of the organic phosphorus compounds reported in the literature, some caution is necessary in the use of these drugs until more evidence is available regarding the factors which affect toxicity.

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BOOK REVIEW

FOOD POISONING—Elliot B. Dewberry.

4th Edition, London. Leonard Hill Ltd. 1959. pp. 404 + xvi, plus index; 40 full and 16 text illustrations.

This is an excellent work in which the subject matter has been dealt with most thoroughly. It deals with food-borne infection and intoxication due to Salmonellae, Staphylococci, metallic salts, poisonous plants, poisonous fungi, fish and shell fish, Cl. *botulinum* and Cl. *welchii*. There is a chapter on laboratory investigations, one on inspection of canned foods and one on food hygiene (principles and methods of prevention). The book should be of great interest to food hygienists, toxicologists and bacteriologists.

L. W. van den H.

VETERINARY DRUGS IN CURRENT USE—R. Seiden.

New York, Springer Publishing Co. 1959, pp. 128. Price $2.25.

This well printed, soft covered publication is designed to render information instantly available to the practitioner on his rounds or the student working in a clinic, in the form of a quick reference pocket book. Drugs used in the prevention and control of disease in farm animals, including poultry, dogs and cats are arranged in alphabetical order. Common synonyms are given and cross-indexed; all the animal diseases for which the drugs are recommended are briefly described and cross-indexed.

The descriptions of drugs are concise and include official name, synonyms, pharmacologic grouping, physical and chemical properties, uses, warnings, antidotes, doses, and cross-references.

Every practitioner or student will immediately recognize the utter practicability of this little book.

The author reveals his own wide practical experience in the selection of drugs which are in current use all over the Western world and in the presentation of only the essential facts concerning them.

In the reviewers opinion, this publication will be a most useful acquisition to every veterinary surgeon's library, no matter what his interest or special field of endeavour happens to be.

K. v. d. W.

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THE CONTROL OF INTERNAL PARASITES OF CATTLE WITH A 10:1 MIXTURE OF NEGUVON AND ASUNTOL.

S. Stampa.*.

Introduction

Riek and Keith[3] found that Neguvon (Bayer L13/59) gave good control of the following parasites of cattle at safe dosing rates: *Haemonchus placei*, *Ostertagia ostertagia*, and *Oesophagostomum radiatum*. It also controlled *Bunostomum phlebotomum*, *Cooperia* and *Trichostrongylus* spp. but only to a lesser extent. Furthermore, Herlich & Porter[1] determined that Asuntol was very effective against Haemonchus, Ostertagia, Cooperia, Trichostrongylus and Trichuris, but not effective against Bunostomum. Stampa[5] recommended for the control of internal parasites of sheep, that use should be made of a 10:1 mixture of Neguvon and Asuntol, as this mixture controlled a wider range of species at safe dosing rates than either remedy alone. Reinecke and Schutte[2] could not demonstrate the advantage of mixing Neguvon and Asuntol for the control of internal parasites of cattle. They favour the use of Asuntol only, which, according to their experience, controls all species of importance, with the exception of Bunostomum, at 10–15 mg./kg.

The aim of the work described in this paper, was to test the efficacy and safety of the 10:1 Neguvon/Asuntol mixture, for the control of internal parasites of cattle, which, according to the writer, provides the best results in sheep.

Materials and Methods

A 9.1 per cent solution of Neguvon plus 0.91 per cent suspension of Asuntol was used in all but one experiment, in which a miscible oil, containing 45.5 g. Neguvon and 4.55 g. Asuntol per 100 c.c. was tested undiluted. Working methods were the same as those used in the experiments with sheep[5]. Faecal egg counts were made according to the Gordon Whitlock method. In order to determine very small egg counts, which are frequently found after dosing, 4 g. of manure was mixed with 56 ml. sugar solution and 1.5 mg. of this mixture was examined. The lowest possible egg count was, therefore, 10 e.p.g. When two counts were made before and after dosing, as was the case in most experiments, the lowest possible average was 5 e.p.g. The eggs of Haemonchus, Cooperia, Trichostrongylus and Oesophagostomum were counted together without trying to differentiate between these species. The number of eggs to be accounted for by each of these different species of worms, was calculated on a percentage basis according to larval identifications from faecal cultures. Naturally infested animals carrying a mixed infestation were used in all experiments. They were kept on infested pasture before and during the time of observation.

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TABLE 1. PERCENTAGE REDUCTION IN FAECAL EGG COUNTS

* Very slight infection before dosing.
** Emulsion.
— Not applicable.
0 = 0–59% reduction in worm egg count after dosing.
\+ = 60– 79% reduction in worm egg count after dosing.
++ = 80– 89% reduction in worm egg count after dosing.
+++ = 90–100% reduction in worm egg count after dosing.

Name of Farm	Avon	Moria	Moria	Yelverton	Avon	Dandee	Dandee	Dandee	Yelverton
Number of Animals	7	6	5	6	6	6	6	6	4
Dose mg./kg.	30–40	40	40	55	60	60	60	60**	93.5
Sodium Bicarbonate premedicated	Yes	Yes	No	Yes	Yes	Yes	No	No	Yes
Haemonchus	—	+++	+++*	—	—	+++*	+++*	+++*	+++
Cooperia	—	+++	+++	—	—	+++	+++	++	+++
Trichostrongylus	—	+++	+++	—	—	—	—	—	+++*
Oesophagostumum	—	+++	+++*	—	—	+++*	—	—	+++
1–4 unspecified	+++	—	—	+++	—	—	—	—	—
Bunostomum	0	+++	0	—	+++	+	+++	+++	+++

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RESULTS: *A. Efficacy*

The percentage reduction in faecal egg counts is shown in table 1. (Page facing).

In the experiment "Avon", no controls were kept. In the other experiments, untreated controls showed no significant increase or decrease in eggs per g. during the observation period.

The results against Bunostomum are not as consistent as those obtained against other parasites. Although no Bunostomum eggs were found in the manure samples of some animals after dosing, (thus representing a 100 per cent reduction in faecal egg counts), in other animals the reduction in the output of Bunostomum eggs was only small or even nil. Larval cultures confirmed this observation. A further experiment with sheep produced similar results.

TABLE 2

REDUCTION OF BUNOSTOMUM EGGS BY DOSING SHEEP WITH NEGUVON/ASUNTOL AND PHENOTHIAZINE

Eartag No.	E.p.g. before dosing	E.p.g. after dosing	Reduction per cent	Treatment
Y 51	200	0	100	70 mg./kg. Neguvon/Asuntol
Y 76	533	300	43.5	
S 76	267	280	0	
S 77	133	0	100	
S 78	333	0	100	
Y 96	267	0	100	
Total:	1733	580	66.6	
G 19	467	0		25 g. Phenothiazine (820–925 mg./kg.)
G 43	0	20		
G 70	200	40		
G 88	0	20		
Total:	667	80	88.0	
B 1	267	867		Untreated Controls.
B 2	0	333		
B 60	0	67		
B 69	200	200		
B 96	333	200		
Total:	800	1667	Increase: 108.5 per cent.	

A considerable increase of e.p.g. will be noticed in the untreated controls. This however, does not explain the variable reduction of Bunostomum eggs by Neguvon/Asuntol.

B. Toxicity:

The efficacy trials revealed that 60 mg./kg. is an adequate dosage. Toxicity trials were conducted in order to show that (A) this dosage is well tolerated and (B) to what extent a 50 per cent higher dosage, i.e. 90 mg./kg. is tolerated by animals kept under Asuntol, indicating also age, conditions, locality and nutrition. In table 3 the results of toxicity tests with 90 mg./kg. Neguvon/Asuntol are shown, and in table 4 those with Neguvon A at varying dosage rates.

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TABLE 3. TOXICITY TESTS WITH 90 mg./kg. NEGUVON/ASUNTOL

Number of Animals	Age	Condition	District	Food	Reactions
38	3–4 months ..	Fair to good	Vryburg, C.P.	Green grassveld	None
5	4 months	Fair	Koppies, O.F.S.	Green grassveld and concentrates	None
5	4–6 months ..	Poor	Acornhoek, Transvaal	Green grassveld	None
5	6–12 months ..	Poor	Koppies, O.F.S.	Partly dry grassveld and concentrates	None
5	8 months	Fair	Louis Trichardt, Transvaal ..	Dry grassveld	One light
52	12–15 months..	Fair to good	Vryburg, C.P.	Green grassveld	One light
10	1–2 years.....	Fair	Acornhoek, Transvaal	Green grassveld	None
Total: 120.					

Five further aninals of the same age, but kept on veld and concentrates on the farm near Louis Tichardt, received 99 mg./kg. Of these animals two reacted severely, one slightly, but all survived.

TABLE 4. TOXICITY TRIALS WITH NEGUVON A

Number of Animals	Age	Condition	District	Food	Dosage mg./kg.
6	6–8 months ..	Poor	Koppies, O.F.S.	Green grassveld and concentrate	80
5	1–2 years.....	Fair	Tweeling, O.F.S.	Partly green grassveld	80
3	Adult	Fair	Tweeling, O.F.S.	Partly green grassveld	80
5	6–8 months ..	Very poor	Koppies, O.F.S.	Green grassveld and concentrate	70
1	Very old	Very poor	Koppies, O.F.S.	Green grassveld	60
53	Adult	Fair	Louis Trichardt, Transvaal ..	Dry grassveld	60
18	Adult	Very poor	Louis Trichardt, Transvaal ..	Dry grassveld	60
6	6–8 months ..	Very poor	Koppies, O.F.S.	Green grassveld	60
6	1–2 years.....	Good	Warmbaths, Transvaal	Dry grassveld	60
10	1–2 years.....	Poor	Acornhoek, Transvaal	Green grassveld	55
35	½–1½ years	Fair	Tweeling, O.F.S.	Partly green grassveld	50
64	1½–3 years	Fair	Tweeling, O.F.S.	Partly green grassveld	50
Total: 212					

There were no reactions in the latter case.

As a result of this experimental work, a preliminary recommendation was drawn up as follows. Animals of less than 4 months of age were not included.

150–200 lbs. live weight	3.8 gr. a.i. — 42–56 mg. kg.
200–300 lbs. ,, ,,	5.23 gr. a.i. — 39–58 ,, ,,
300–400 lbs. ,, ,,	7.6 gr. a.i. — 42–57 ,, ,,
400–500 lbs. ,, ,,	10.0 gr. a.i. — 44–56 ,, ,,
500–600 lbs. ,, ,,	12.36 gr. a.i. — 45–55 ,, ,,
Over 600 lbs. ,, ,,	14.72 gr. ai.i — up to 54 mg./kg.

To date approximately 100 kg. a.i. which is enough for approximately 7,000–10,000 head of cattle, has been used by farmers according to this dosing recommendation. This includes pregnant and lactating cows, animals regularly dipped or sprayed, of different breeds and ages, and animals kept in the Transvaal, Orange Free State, Natal, the Eastern Cape Province, South West Africa and Southern Rhodesia. The only adverse effect observed and brought to notice was a slight drop in the milk yield of a number of Jersey cows; this lasted for 2 days only; thereafter production was normal again. There was no taste or smell recognizable in the milk after dosing. This is not surprising since less than 0.2 per cent of the total amount of Neguvon taken orally is excreted in the milk.[4] Whether measurable quantities of Asuntol are excreted in the milk after oral administration is not known.

Conclusion

It can be concluded from the findings presented on this paper that 40 mg./kg. of the 10:1 mixture of Neguvon and Asuntol, provided good control of Haemonchus, Cooperia, Trichostrongylus, and Oesophagostomum. The efficacy against Haemonchus and Oesophagostomum is substantiated by the findings of Riek and Keith[3] who proved that Neguvon alone controls these parasites at 44 mg./kg.

The results are not clear-cut as far as the control of Bunostomum is concerned. It appears that Neguvon/Asuntol fails to control Bunostomum spp. at 40 mg./kg. At 60 mg./kg. Bunostomum is controlled in cattle from 60–100 per cent.

Bunostomum was better controlled in the experiment "Moria," when Neguvon/Asuntol was given after premedication by a 10 per cent sodium bicarbonate solution. In the experiment "Dandee," this was reversed. It must be assumed, therefore, that these results represent the variability of the efficacy against Bunostomum and not the influence of premedication with sodium bicarbonate. No improvement in the efficacy of Neguvon/Asuntol by the premedication of sodium bicarbonate could be demonstrated. This is in contrast to the earlier findings with sheep[5] where a premedication with a 10 per cent copper sulphate solution improved the efficacy against the parasites in the small intestine.

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Although the remedy appeared to be slightly less effective against Cooperia, in the experiment "Dandee", in the group which received it in the form of an undiluted 50 per cent emulsion, this difference was rather small and, unless confirmed by further trials, cannot be regarded as significant. As 90 mg./kg. was well tolerated in different areas of South Africa, the Neguvon/Asuntol mixture provides a safety margin, broad enough to recommend it also for the control of internal parasites of cattle. The remedy is apparently tolerated less by animals kept on dry grass pasture.

Summary

(1) A 10:1 mixture of 40 mg./kg. is shown to be effective against Haemonchus, Trichostrongylus, Cooperia, and Oesophagostomum, but is not effective against Bunostomum.

(2) Bunostomum is controlled at 60 mg./kg.

(3) Premedication with sodium bicarbonate did not influence the efficacy of the remedy.

(4) The remedy is well tolerated under a variety of conditions, but is apparently more toxic when cattle are kept on dry grass pasture.

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SOME OBSERVATIONS ON THE USE OF SUCCINYL CHOLINE CHLORIDE (SUXAMETHONIUM) IN HORSES WITH PARTICULAR REFERENCE TO THE EFFECT ON THE HEART.

C. F. B. Hofmeyr*

The advent of muscle relaxants on the clinical scene was heralded by the use of curare, which is the active principle in South American arrow poison, and which acts by paralyzing the neuro-muscular end-plate. It has been used, although sporadically, for more than a decade in veterinary medicine.

As its prolonged action (which can be shortened by the use of cholinergic drugs) is a disadvantage, the search for muscle relaxants went on and has resulted in the elaboration of a short acting relaxant—succinyl choline chloride (or iodide). This drug depolarizes muscle and is destroyed by the cholinesterase of the body. In view of experience elsewhere with this drug in horses it was decided to conduct a series of clinical trials on equine animals.

Review of Literature

Stowe[11] mentioned the intravenous use of succinyl choline chloride in "over 30 horses". The dose varied from 0.08 mg./Kg.;—1 mg./Kg.; it was injected in 3 to 10 seconds, caused collapse in 20 to 30 seconds, led to apnoea for ½ to 2½ minutes and paralysis for 2½ to 10 minutes. No cyanosis was observed. Large doses could cause death, which could be prevented by artificial respiration. He stated further: "During prolonged apnoea there are concurrent abnormalities in the electrocradiogram". The same author[12] elsewhere reported similar findings.

Jones[7] in discussing general anaesthetics in the horse and ox stated that muscle relaxants will materially assist surgery.

Hansson[4] cast about 500 horses without accident using succinyl choline iodide. He used it together with 6% Nembutal. He listed doses and their effects from 0.13 mg./Kg. to 0.30 mg./Kg. The only complication was respiratory arrest. A case receiving 0.3 mg./Kg. exhibited moderate cyanosis, and started respiration in 1½ minutes. He found 0.18 mg./Kg. to be the optimum dose in Thoroughbreds and 0.13 mg./Kg. in draft horses. Hansson[5] further reported on the use of this drug on 660 horses and 140 cattle in a total of 1,000 castings. The mode of administration was the same as before and the doses given were 0.17 mg./Kg. for warm blooded horses, 0.13 mg./Kg. for cold blooded horses and 0.02 mg./Kg. for cattle. He found that muscular function returned in five minutes. He applied artificial respiration if apnoea lasted longer than 45 seconds but did not regard it essential even in marked overdosage. The usual period of apnoea

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was found to be ½ minute. Most of the horses were American Standardbreds. The same author[6] again reported on the use of this drug in horses and cattle, and, in addition, on dogs and pigs. He stated that the therapeutic tolerance in the horse is high and a complete block is brought about at 0.60 mg./Kg. with restoration in six to seven minutes.

Ammann[1] used Cellocurin, a proprietary preparation of succinyl choline chloride. He administered it intravenously in 10 seconds and took precautions that the horse did not injyre its mouth and teeth. Apnoea lasted 1 to 1½ minutes. The subjects rose within 10 to 15 minutes. Celocurin was administered at the rate of 0.17 mg./Kg., usually combined with a barbiturate in the same syringe. He directed attention to the fact that the neutralization of succinyl choline chloride depended upon the serum concentration of pseudo-cholinesterase. If this was low then paralysis persisted longer.

Larsen[8] used suxamethonium (succinyl choline chloride) in more than 500 large animals and combined it with a barbiturate. In the horse muscular fibrillation started in 20 to 30 seconds. Apnoea and collapse lasted ½ to 1 minute. First diaphragmatic, then intercostal breathing, then hyperventilation were noted. He also mentioned that suxamethonium is hydrolized by serum cholinesterinase and pseudo-cholinesterases. As the horse has high cholesterinase levels, recovery took place in three to four minutes, but in dogs, where they are much lower, apnoea might last 20 to 30 minutes.

Dietz, Krause and Sattler[3] stated that succinyl choline chloride had its main application in the horse and dog in veterinary medicine.

Stevenson and Hall[10] observed the effects of the drug in horses under thiopentone-cyclopropane-oxygen anaesthesia. Intravenous use of 0.17 mg./Kg. suxamethonium caused a rise in blood pressure of 50 mm. Hg. without causing apnoea. With a dose of 0.34 mg./Kg. the rise in blood pressure was 100 mm. Hg. The pulse rate increased 30% and 125% respectively. They concluded by saying that it was clear that suxamethonium casting was not without risk in healthy animals.

Neal and Wright[9] described the use of succinyl choline chloride as a casting agent in 20 horses, at the dosage rate of 0.18 mg./Kg. The latent period was 20 seconds (range 10 to 35 seconds). Fasciculations lasted 15 to 30 seconds. The horse then sank to the ground. Duration of apnoea was from one to nearly three minutes. Auscultation of the heart was alarming. Within 15 seconds after falling gross abnormalities developed: bradycardia with intervals of up to 6 seconds between beats, then periods of tachycardia of 180 beats per minute. Variations in tone and regularity were evident. They did not report any cardiac fatalities but were hesitant about advising this drug in any animal with an impaired heart.

Tavernor" mentioned "striking electrocardiographic changes." He had one death in his series, after 85 mg. succinyl choline chloride had been administered to a gelding weighing 850 lb. Primary cardiac arrest occurred; there was no period of apnoea. Post-mortem examination showed fatty changes in the heart.

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Material and Methods

The present series consisted of 33 experimental animals—31 horses and two mules—subjected to intravenous succinyl choline chloride (suxamethonium) administration at the rate of 0.17 mg./Kg. (three cases received 0.16 mg./Kg. and two 0.18 mg./Kg.) for the purpose of general clinical assessment. The animals were all in good general condition, of riding or draft type, and all were over 12 years old, some much older. These investigations were done over a period of months whenever cases and time made such investigations possible.

No particular attention was paid to the heart, until one subject (No. 4) died. Subsequently electro-cardiograms were recorded by means of lead III in seven cases: viz. Nos. 15, 16, 19, 20, 31, 32, 33. Amongst these was one further death (No. 32). In two cases 30 mg. atropine sulphate was administered intravenously in an attempt to determine whether stimulation of the vagus played a rôle, as it had been stated that cardiac arrest could occur due to this cause after injection of succinyl choline chloride in man.

At no stage was recovery assisted by artificial respirations or any other means, the purpose being to assess the effects of succinyl choline chloride as thoroughly as possible.

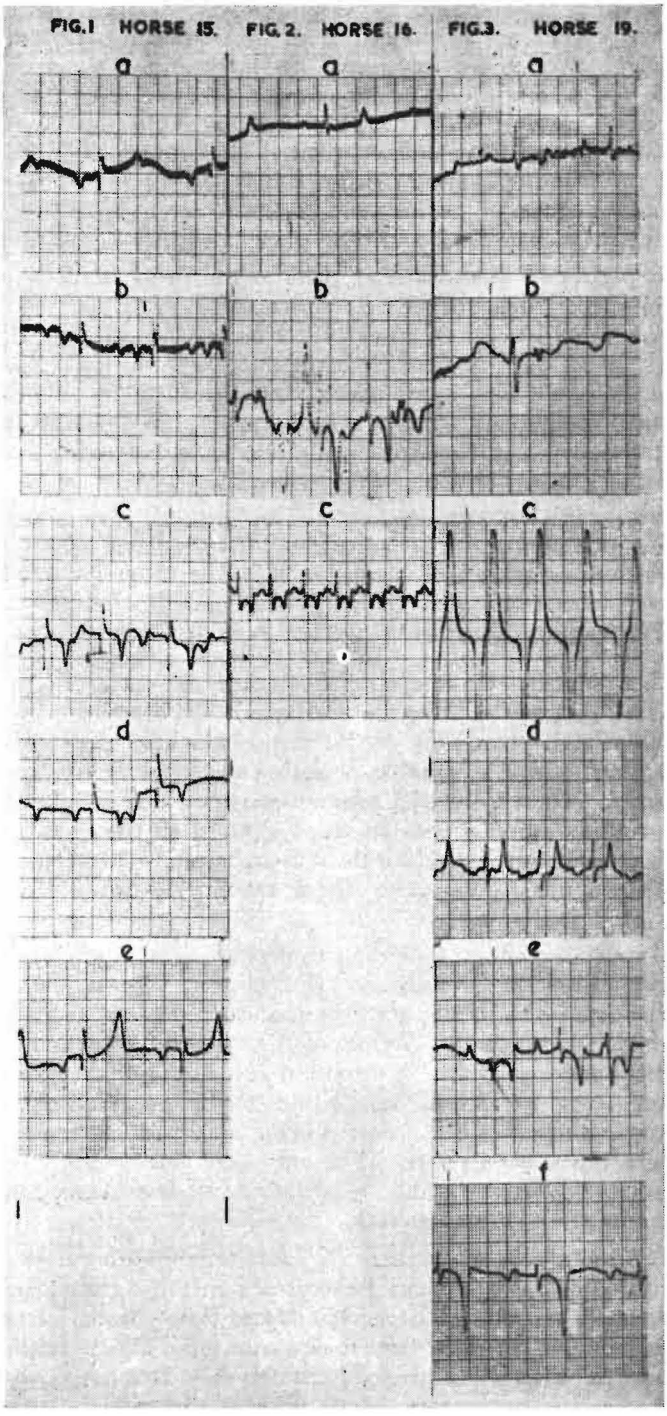
General Observations

When succinyl choline chloride is injected intravenously at the dosage rate indicated above, the first symptoms seen are muscle fasciculations as well as moving of the membrana nictitans over the eye. This happens within a variable period, but almost always well under 30 seconds. Then follows a sudden collapse, when the animal falls to the ground and rolls onto its side. The mean time between injection of succinyl choline chloride and collapse is 30 seconds (range 12 to 76 seconds). Two cases did not collapse, nor did they show apnoea.

All muscular effort, including respiration, is arrested at this stage in almost all cases. There were five exceptions amongst 31 subjects that collapsed. The mean period of apnoea (if present) is 55.8 seconds (range 14 to 112 seconds). When respirations return, this is heralded by rather inco-ordinated contraction of abdominal and thoracic respiratory muscles as well as dilation of the nostrils. At first the respiratory volume is small, but quickly attains normality while passing through a hyperpnoea phase. The animal is able to get up within a minute or two after this. The mean period of recumbency was 213.9 seconds (range 77 to 464 seconds).

In contrast to the findings of some other authors, cyanosis is evident in some cases. It can be expected in those cases that do not breathe for 90 seconds and longer. It has been shown by cases that remained apnoeic for much less than a minute. This is probably due to associated severely impaired circulation. This was particularly noticeable in the two mules. It is notable that the mules clinically showed much less resistance to succinyl choline chloride than horses,

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collapse being generally earlier. The number of mules is too small, however, to permit of any definite conclusions. (Detailed obversations on each case may be had on request).

In the majority of cases there is tachycardia, and in more than hslf (18 of the 28 cases so examined) cardiac arrhythmias. The recorded electro-cardiograms, of which representative reproductions from all seven cases are given in figures 1 to 7, together with subjoined explanatory text, provide the available data regarding the cardiac reactions obtained in this study. In this connection the work of Brooijmans[2] on electrocardiaography in the horse and cow was consulted: Dr. J. M. Combrink of the Department of Medicine of the Medical Faculty contributed materially to helpful discussions on the E.C.G. tracings.

Discussion and Conclusion

The fact that two deaths had occurred in 33 subjects is contrary to expectations, as various authors[4],[8] had reported on the apparent safety of the drug. These authors, as well as most of the others who have used succinyl choline chloride, did not draw attention to any cardiac effects. The subjects used in this investigation were all cases in which some form of cardiac impairment would not be regarded as unusual. None on ausculation at rest revealed marked cardiac aberration. The cases that died were closely similar, the one collapsing in 54 seconds and the other in 45 seconds; the mean time being 30 seconds for all cases. Hence no particular general sensitivity to the action of the drug is indicated.

One case (No. 4) showed apnoea for 108 seconds (mean time of all cases being 55.8 seconds). This was rather protracted and yet not

Fig. 1. (Horse No. 15)

(*a*) Tracing before injection of succinyl choline chloride (s.c.c.). Somatic shivering.
(*b*) After injection of s.c.c. and immediately before collapse. Tachycardia.
(*c*) Ventricular extrasystole and T wave changes. Ventricular block.
(*d*) Reversal of T waves (often associated with myocardial injury).
(*e*) Restoration.

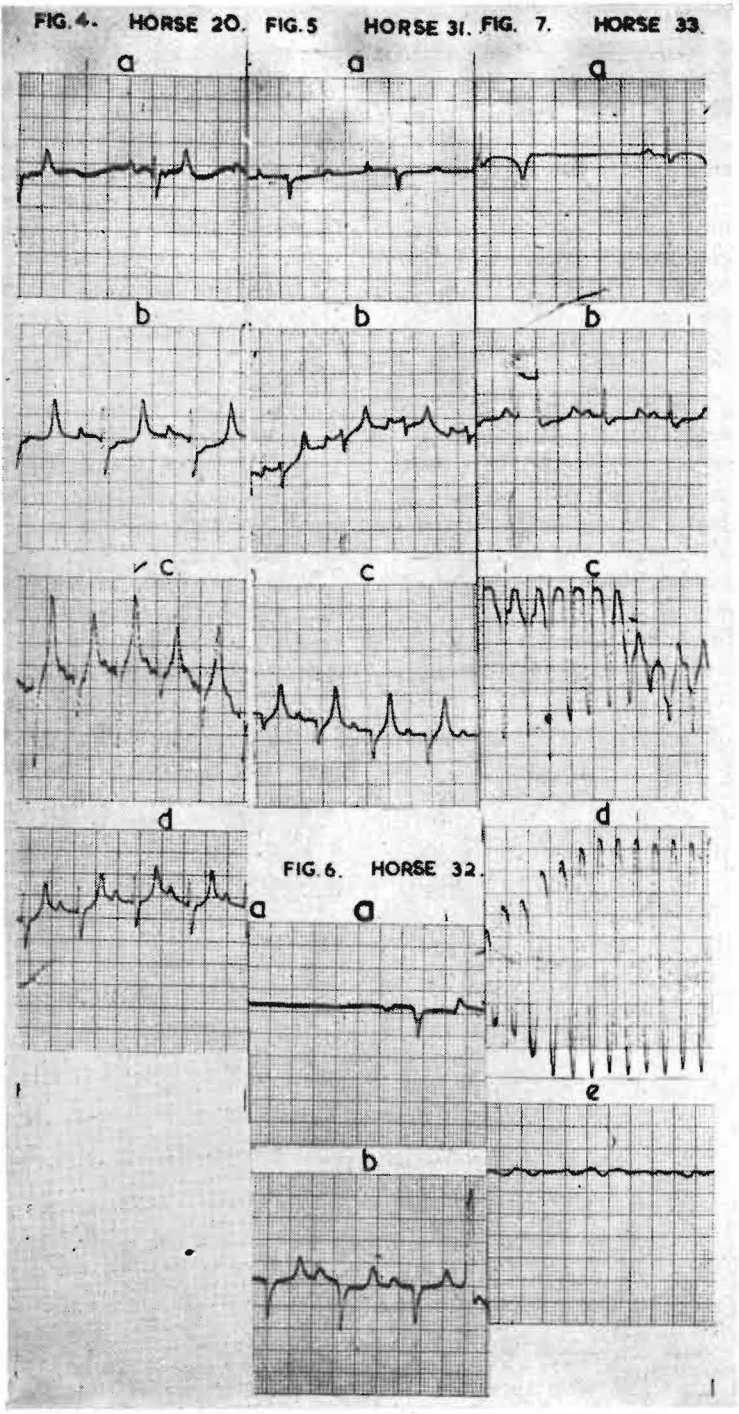
Fig. 2. (Horse No. 16)

(*a*) Before injection of s.c.c.
(*b*) Within 45 seconds of the injection of s.c.c. First supraventricular tachycardia and then polytopic ventricular tachycardia.
(*c*) At 105 seconds supraventricular tachycardia (200 p.m.) with signs of myocardial ischaemia.

Fig. 3. (Horse No. 19)

(*a*) Sinus rhythm before s.c.c. injection.
(*b*) 45 seconds after collapse. Cardiac arrest (fibrillation) and ventricular extrasystole after a further 30 seconds.
(*c*) Ventricular tachycardia after a further 15 seconds.
(*d*) Supraventricular tachycardia after a further 15 seconds.
(*e*) Ventricular tachycardia alternating with supraventricular arrhythmia.
(*f*) 3 Minutes 30 seconds after administration of s.c.c. Suggestion of superficial myocardial injury.

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long enough, the author feels, to account for death, especially as powerful respirations were in evidence just prior to that. The same applies to Case 33. The period of apnoea was not recorded, as attention was occupied by the electrocardiograph at the time, but the case breathed well before exitus, so that the above remarks would apply here as well. Both were, without a doubt, cases of primary cardiac arrest. Unfortunately a histopathological examination of the myocardium was not made in either case, but the heart showed sub-epicardial and sub-endocardial haemorrhages macroscpically.

Case No. 33 had had atropine sulphate (30 mg.) intravenously before administration of succinyl choline chloride but case No. 4 did not.

Stowe's[11] statement regarding abnormalities in the electrocardiogram during prolonged apnoea creates the impression that the apnoea is held responsible for cardiac signs. Of the seven cases examined by E. C. G. in the present series, only one did not show abnormal tracings after succinyl choline chloride injection, while the periods of apnoea were 31, 15, 24, 25, 45, seconds respectively, i.e. all less than the observed average period.

Hansson[6] noted tachycardia after administration of succinyl chloride. This he explained in that contraction of the abdominal muscles had lead to increased rate of filling of the right atrium and a tachycardia via Bainbridge's reflex. This theory is not tenable, as tachycardia occurs before other manifestations, such as muscle contractions. Furthermore, there is not only an increase in rate but also

FIG. 4. (HORSE NO. 20)

(*a*) Before use of s.c.c. and atropine.
(*b*) After injection of 30 mg. atropine sulphate intravenously. Sinus tachycardia.
(*c*) After injection of s.c.c., at collapse. Supraventricular tachycardia.
(*d*) Within 30 seconds of collapse: return to normal.

FIG. 5. (HORSE NO. 31)

(*a*) Before injection of s.c.c.
(*b*) After injection of s.c.c. and before collapse.
(*c*) Within 15 seconds of collapse.

These E.C.G.'s show only acceleration of heartbeat and more clearly marked oscillations after the use of succinyl choline chloride.

FIG. 6. (HORSE NO. 32)

(*a*) Before use of s.c.c.: sinus arrhythmia and arrest.
(*b*) After use of s.c.c.: ventricular extrasystole with improvement of sinus ayrhthmia.

FIG. 7. (HORSE NO. 33)

(*a*) Before administration of s.c.c. or atropine sulphate.
(*b*) After 30 mg. atropine sulphate intravenously. Sinus trachycardia.
(*c*) 45 seconds after injection of s.c.c. Ventricular fibrillation and supraventricular tachycardia.
(*d*) Ventricular fibrillation.
(*e*) 3 Minutes after injection of s.c.c.: cardiac arrest and death.

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modification in character of the heartbeat, as is illustrated by most of the E. C. G.'s shown.

Stevenson and Hall's[10] observations lead them to reject the concept of asphyxia as playing a likely rôle and to suggest stimulation of the post-ganglionic nerve fibres with consequent release of adrenalin: such an action can provoke cardiac irregularities and there is no need to postulate a direct effect on the cardiac centre or heart muscle. These statements cannot be wholly acceptable in view of the E.C.G. evidence presented, which suggests actual injury to the myocardium.

Neal and Wright[9] express amazement that other workers did not emphasize the cardiac changes and that many apparently did not observe them. No cardiac fatalities have been reported. These authors are reluctant to advise the indiscriminate use of this drug.

Tavernor[13] shares the feelings of the previous authors and mentions "striking E.C.G. changes". With one death in his series due to primary cardiac arrest, he stresses the importance of great attention to the circulatory system whenever this drug is employed.

The present author finds himself in considerable sympathy with Neal and Wright and with Tavernor. It is very difficult indeed to explain the remarkably good results of Hansson[4,5,6] with horses and cattle. He does not state whether his cases were all relatively young or not. Were this so, it is more likely that the myocardium would be normal than in older cases. It is possible that this drug easily exposes a weakness in a heart subjected to previous strains. Or is it conceivable that the simultaneous use of a barbiturate, as Hansson employed, could in some unexplained way act as myocardial safeguard?

It is realized full well that the interpretation of the E.C.G. requires both knowledge and extensive experience. A great deal remains to be discovered with regard to electrical examination of the equine heart. It is, therefore, not intended to attempt a detailed analysis and discussion. Suffice to state that there appears to be important evidence of the influence on the cardiac conductive mechanism as shown by extrasystoles, ventricular blocks, and other conduction disturbances. Similarly changes in the T waves strongly suggest myocardial injury.

The fact that atropine pre-medication at adequate dosage level—as indicated by the well marked and typical atropine sinus tachycardia—did not prevent death suggests that the vagus does not play a significant rôle in the cardiac arrest following succinyl choline chloride.

In conclusion, clinicians must be warned gravely against the indiscriminate use of succinyl choline chloride or similar drugs in horses, because of the danger of causing heart injury, which may or may not be fatal.

A further question arises at the present stage of our knowledge. Is the use of this drug at all justified in the race horse? As the slightest cardiac impairment will show itself in a race, can we be sure that myocardial injury caused by the use of this drug will not prejudice the racing career of the patient? This is an important consideration—very much more so in the Thoroughbred than in the hack or casual jumper. An additional consideration is the possible injury to lips and teeth and to the hips and pelvis as result of the sudden collapse.

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Summary

(1) Succinyl choline chloride was used on 31 horses and two mules, at the rate of 0.17 mg./Kg. intravenously.

(2) Collapse occurred in 31 cases, in a mean time of 30 seconds after injection of the drug. Apnoea occurred in 26 of the 31 cases that collapsed, and lasted an average of 59 seconds. The mean period of recumbency was 214 seconds.

(3) Two horses died in cardiac arrest.

(4) E.C.G. studies, done on seven cases, demonstrated effects on the cardiac conduction mechanism and indicated myocardial injury.

(5) Warning is issued against indiscriminate use of the drug; apprehension is expressed with regard to possible long term effects, *inter alia* in race horses.

Acknowledgments

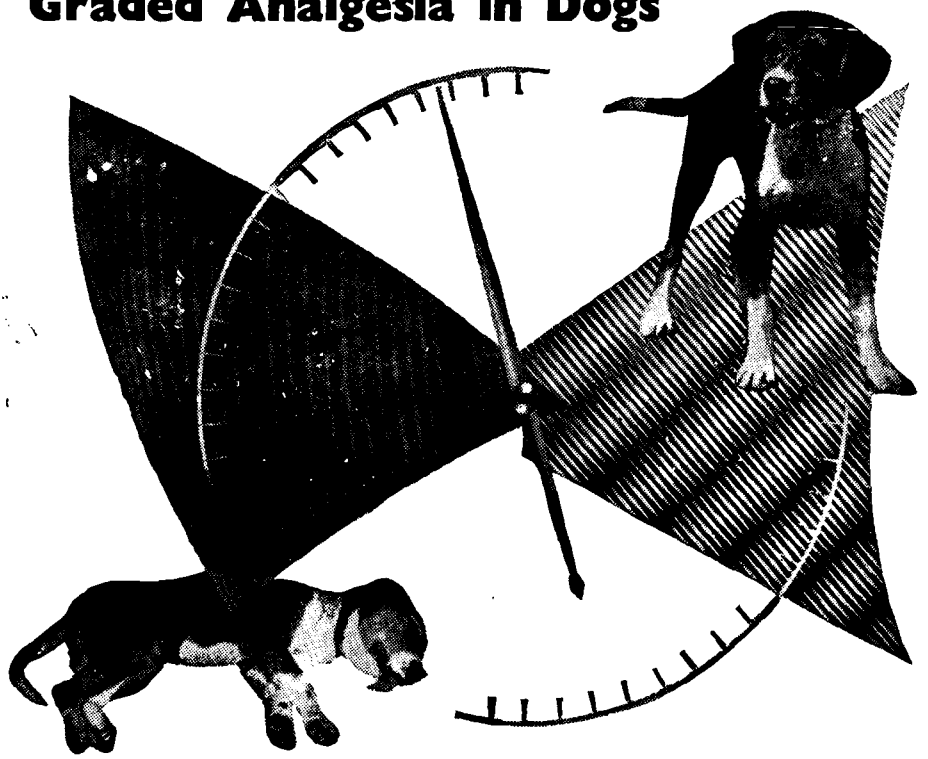
The author has great pleasure in acknowledging the assistance of the following: Professor K. van der Walt, Department of Medicine of this Faculty for loan of the E.C.G.; Dr. J. M. Combrink (mentioned in the text) and Mr. D. de Vos, Chief Technical Officer in the author's department. The author is also indebted to the Director of Veterinary Services for permission to publish this article; further, to Messrs. Maré and de Bruyn who respectively mounted and photographed the tracings. Last but not least, the author is grateful to Messrs. Burroughs Wellcome for supplying the Anectine (Succinyl choline chloride) used in this experiment.

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REPORT ON A TRIAL WITH M & B 5062A IN CANINE BABESIASIS.

K. van der Walt*

Following successful clinical trials with M&B 5062A (3:3[1] diamidinocarbanilide di-isethionate) in the treatment of redwater (*B. bovis.*) in the British Isles, this drug was submitted to the Director of Veterinary Services at Onderstepoort for trials in the treatment of canine babesiasis (*B. canis* infection).

The drug was supplied as a solution in multi-dose rubber-capped bottles containing 50 c.c. each, and labelled M&B 5062A solution; 50% W/V; 500 mg. in each c.c.

Canine Babesiasis (Biliary fever).

This is a very common tickborn disease of dogs with a wide distribution in South Africa. The disease is caused by a pearshaped endoglobular protozoan parasite, (*Babesia canis*) and is transmitted by the dog ticks *Haemaphysalis leachi* and *Rhipicephalus sanguineus.*

The disease is characterised by an incubation period of 10–21 days in naturally infected dogs. Animals show a sudden rise in temperature (105°–106°) accompanied by rigors, anorexia and listlessness. The disease generally follows an acute course; most of the untreated animals succumb after 4–8 days.

There is a progressive anaemia which may become marked after about 72 hours and may be followed later by haemoglobinuria and jaundice. Apart from the severe anaemia, animals show liver dysfunction which, with myocarditis, often contributes to early death.

In early cases, treatment is most effectively carried out with phenamidine isethionate 5% solution. In advanced cases supportive treatment with blood transfusions, liver protecting agents and haematinics is essential.

Methods

All the recorded cases were presented at the Veterinary Faculty's outpatient department for treatment. The majority of animals were hospitalized until regarded as fit to go home.

In every case blood smears were made on admission and daily thereafter to follow the incidence of parasites in the erythrocytes.

On admission, venous blood was collected in oxalate and a full haematological investigation was undertaken, to assess the severity of the case at the time of treatment.

Where necessary, blood transfusions and/or general supportive treatment were given according to experience gained in many thousands of cases.

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Administration of M&B 5062A.

The 50% solution was diluted with sterile physiological saline to a 10% solution in order to facilitate the computation of the dose. In all cases the estimated dose was injected subcutaneously in the neck region after taking the normal precautions for this procedure.

Reaction to M&B 5062A.

At no time during the trial was there any evidence of pain at the time of injection or of a local reaction afterwards.

Even at a dosage rate of 50 mg./kg. sick animals showed no apparent sign of discomfort and at no time were the animals observed to vomit. Vomiting, due to a fall in bloodpressure, is one of the usual reactions following the administration of phenamidine or one of the quinuronium compounds.

Dosage rate.

From evidence supplied by the manufacturers gained with treatment of *B. bovis* infection in cattle, it appeared that a dosage rate of 5 mg./kg. body weight should be sufficient. As the trial progressed, however, the dose was increased and cases were treated with 7.5 mg., 10mg., 20mg., 30mg., 40mg., and 50mg./kg. body weight.

Analysis of cases.

Forty dogs (30 males, 10 females) were treated with M&B 5062A. The animals were representative of all the breeds normally found in an urban community. The ages varied from three months to seven years but the majority was under 12 months. Body weight varied from 4.5 kg. to 43 kg. the average being slightly less that 10 kg.

Analysis of the haematological picture shows a wide variation, but usually when an owner decides to present a case for treatment the animal has a red blood cell count of about 2.5 ml. cells/cu m.m with a corresponding haemoglobin and packed cell volume picture. These cases are usually fairly ill, showing listlessness, anorexia, tachycardia, tumor splenis and pale mucous membranes. At this stage recovery normally takes place with routine treament.

Should an animal be presented in a state of medical shock, severe anaemia with dehydration and collapse, a transfusion of whole blood with standard A.C.D. mixture as anticoagulant is given as a routine before specific treatment is undertaken.

Results of treatment.

Of the 40 treated animals 30 recovered after treatment with M&B 5062A alone. In all these cases clinical recovery appeared somewhat slower than would have been expected with our routine treatments, the animals taking about twelve hours longer to regain their appetites and about 3 days before they became completely normal. These recoveries took place at all levels of dosage, ranging from 7.5 mg. to 50 mg./kg.

It is interesting to note that 17 cases still had a positive bloodsmear on the second day and 6 of these were still positive on the third day.

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In two cases infection persisted until the fourth day. Five out of the nine cases of relapse showed a positive smear for more than one day.

There were 9 cases of relapse, occuring on or about the 9th day after the first injection. Two (3813 & 3921) of the nine cases relapsed more than once and in both cases other drugs were used either at the first or second relapse. One of these cases (3921) subsequently died.

Case 4443 had another attack of biliary-fever 24 days after being treated the first time. It is difficult to assess whether this was a relapse or a fresh infection. The animal recovered after being treated with phenamidine the second time.

Four cases of relapse (cases 3676, 3921, 3956, & 4251) were again treated with M&B 5062A during the relapse and they recovered normally. In these four cases the initial dosage rates did not appear to have had any influence on the incidence of relapses as it occurred at dosage levels of 7.5 mg. 15 mg. 20 mg. & 40 mg./Kg.

Two cases (3799 & 3921) died in spite of treatment at initial dosage levels of 10 & 15 mg./Kg. Case 3799 was in a state of shock on presentation and was transfused with 180 cc. whole blood. On the 3rd day, when the bloodsmear was still positive for Babesia, the animal received 5% phenamidine. In our experience this animal would have recovered with routine treatment.

Case 3291 had relapsed twice. The first relapse which occurred 9 days after the initial attack was treated with 30 mg./Kg. M&B 5062A and the animal recovered slowly. Eleven days after the first relapse the dog was presented with a second relapse. In spite of the administration of 40 mg./Kg. M&B 5062A the animal became progessively worse. The bloodsmear remained positive. A bloodtransfusion and trypan blue solution was given intravenously. The animal died in spite of this treatment. In our experience the animal would have recovered if routine treatment had been employed.

Of the treated animals, six (Nos: 3291, 4143, 3575, 3392, 3801 & 3799) received blood transfusions. Four of these recovered.

Discussion

(Details regarding breed, age and sex of each patient, haematological findings treatment, and course of the disease may be had from the author.)

Of the 40 cases treated with M&B 5062A, 30 animals (75%) recovered after a single injection, which indicates that the drug does have a curative value in *B. canis* infection.

The results, however, are not as encouraging as those obtained with *B. bovis* infections in Britain. In spite of increasing the dose tenfold, results were unpredictable and compare unfavourably with the drugs currently used as specifics for *B. canis* infections in South Africa.

At the present stage it does not seem advisable to use M&B 5062A either as a routine or alternative treatment for biliary fever in dogs.

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SUMMARY

(1) A short description of the clinical features of biliary fever in dogs in South Africa is given.

(2) At a dosage rate of up to 50 mg./Kg. bodyweight the drug appears safe and causes no discomfort.

(3) An analysis of the results obtained in 40 cases is presented. At all dosage levels employed the results were variable and unpredictable.

TREATMENT OF ANIMALS POISONED BY ALDRIN, DIELDRIN AND ENDRIN FORMULATIONS.

(Press Release by courtesy of the Shell Company of Africa Limited.)

With the increase in the use of aldrin, dieldrin and endrin insecticide formulations the possibility of animals being poisoned by these materials also increases. Animals, unlike humans, cannot be warned of these dangers but have to be physically restrained from coming into contact with the insecticides. As this is not always possible one must know how to treat such poisoned animals.

This subject of pesticide poisoning of animals is fully covered by R. S. Corner in his "Landers Veterinary Toxicology" published by Bailliere, Tindall and Cox, London, 1957. We would however, like to make additional comments on the treatment which should be administered to animals poisoned by aldrin, dieldrin or endrin formulations.

Symptoms of poisoning by these insecticides are generally nausea, vomiting and convulsions. Vomiting is not always present especially in cases of large quantities of the insecticide having been consumed. As aldrin, dieldrin and endrin react on the central nervous system convulsions are usually present in most cases.

If ruminants or horses are poisoned, barbiturates should not be administered. In the case of the former, gases may be trapped in the rumen since they are lying down and in the case of horses, pneumonia may be induced by lying down. Treatment in the above cases should be similar to that administered to cattle suffering from milk fever. An injection of a soluble calcium salt such as Calcium Borogluconate can be given. In the case of cows the dosage would be about 300 millilitres of a 25% solution and correspondingly smaller doses for sheep and goats etc.

In the case of smaller animals such as cats and dogs barbiturates can be given. The approximate dose of pheno-barbitone for these animals is between one fifth and one seventh of a grain per pound of body weight.

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DERMATITIS IN THE BLACK RHINOCEROS (*Diceros bicornis*) DUE TO FILARIASIS.

K. C. A. Schulz* and E. B. Kluge.**

The cause of the frequent occurrence of ulcerative wounds behind the shoulder of black rhinoceros in the Hluhluwe and Mkuzi game reserves of Zululand has been the subject of speculation for many years[1]. The seasonal appearance of the lesions, coupled with the fact that they were only seen in adult animals, led to the belief that they were associated with secondary sex skin glands which became active during the breeding season. A similar condition has been described in the rhinoceros in the Ambolseli National Park in Kenya[1,2,3]. In Zululand only the black rhinoceros is affected and not the white (*Ceratothium simus*), despite the fact that the two species are often in close proximity. The condition appears in animals of both sexes.

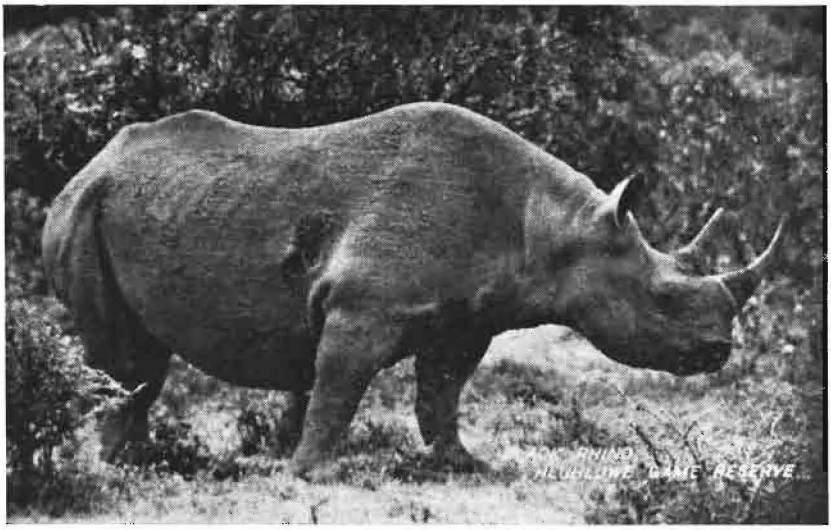


Fig. 1. Photo of a black rhinocerus taken in the Hluhluwe Game Reserve showing the lesion described.
Published by kind permission of the Natal Parks, Game and Fish Preservation Board.

The most conspicuous lesions always occur behind the point of the shoulder and are some six inches in diameter. During the summer they are reddened and accompanied by pruritis. Rubbing and scratching causes further damage and secondary infection is common. During the winter the lesions recede and become almost unnoticeable. Similarly affected areas may be found on other parts of the body but these are seldom as obvious as those behind the shoulder.

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An investigation of the condition has now been made possible through the collection of specimens by one of the present authors (E.B.K.).

The pathological findings vary according to the age of the lesion and the season in which the specimen was taken. Three distinct stages can be differentiated, viz. florid, intermediary and dormant.

Macroscopically the florid stage is characterized by exfoliation, erosions, pustule and crust formation, ulceration and haemorrhage. Round the wound the skin is corrugated by variable sized nodules and depressions and has a mottled appearance due to depigmented areas and scar formation. (Fig 2). Here larvae, nymphae and adults of *Amblyoma hebraeum* and *Rhipicephalus maculatus* and adult *Dermacentor rhinocerinus* may be found attached. Nodular eruption of the skin, as described in the Indian elephant (*Elephas maximus* L.)[4,5] has not been seen.



Fig. 2. Clearly indicates the roughened and mottled appearance of the affected skin of the black rhinoceros.

On microscopic examination the granulation tissue of the ulcers is seen to contain very numerous microfilariae, in adjacent areas these are difficult to demonstrate. In intermediate and quiescent lesions they could not be found even after a most careful search. Their

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morphological characteristics such as the bluntly rounded head, curved and pointed tail and granular body are clearly visible. The parasites lie in irregular serpentile coils and are not provided with a sheath (fig. 3). They are situated in the superficial lymphatics and tissue spaces and have never been observed in blood vesels. Not infrequently microfilariae are in close proximity to the ulcerating surface and would be easily accessible to insects settling on the wounds. They are usually enclosed by inflammatory cells: plasma cells and lymphocytes predominating with occasional neutrophiles and eosinophiles present. Many of these cells exhibit necrobiosis. In a few instances microfilariae are seen in the vicinity of well defined encapsulated, calcified nodules, possibly the remains of dead helminths or trhombosed blood vessels. No sections of the mature filarid worm are seen in these areas.

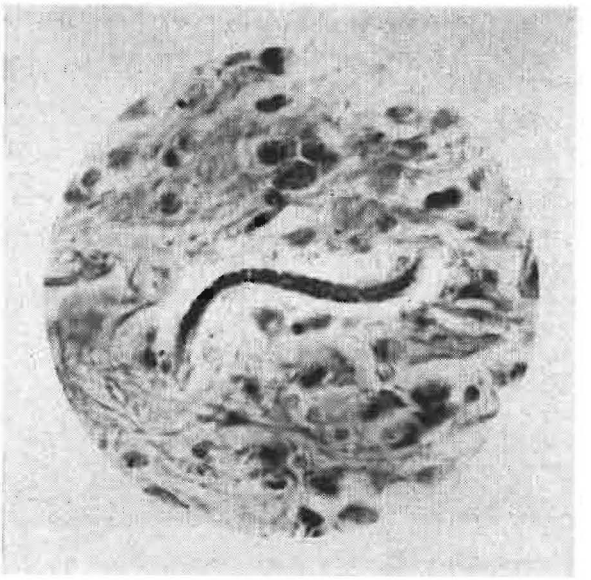


Fig. 3. The serpentine projection of the free lying microfilariae. No cellular reaction is seen.

Since in most active areas fragile, vascular granulation tissue is present, trauma is prone to cause bleeding very readily. Even in places where no alterations of the epidermis are manifest, there is a definite engorgement of blood vessels and haemorrhage subjacent to the surface epithelium. These changes probably account for the distinct red colour of the affected skin.

The intermediary (subacute) stage is characterized by marked hypertrophy and hyperplasia of the epithelium accompanied by pronounced desquamation (erosion) and crust formation. The most salient feature however, is the presence of superficially situated mature filariae, principally female worms. The uterine tubes contain either embryonated eggs or well developed larvae. The adult filarid lies

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either free (fig. 4*a*) or is enclosed in a cystic structure lined by proliferating and desquamating epithelium and an inflammatory cellular exudate. From the epithelial lining long branching rete pegs extend into the adjacent stroma. No free microfilariae are present. These changes closely conform to those of "summer-sores" described in cattle[6]. The corium is definitely less vascular and cellular than in the florid stage. Well defined and extensive haemorrhages with an increase of eosinophiles in their vicinity are observed, the shape and distribution closely resembling those of burrows seen in other helminthic affections.

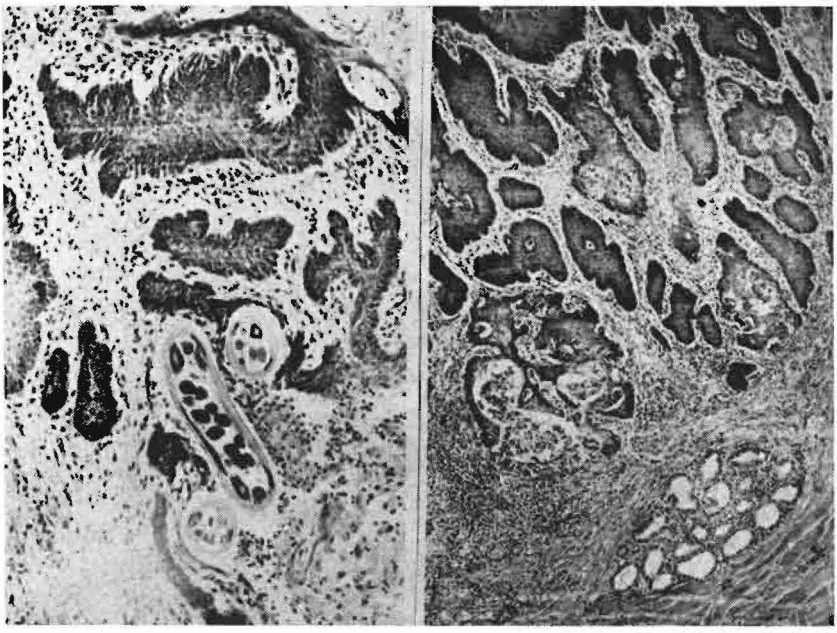


Fig. 4. (*a*) Superficially placed filarid (sections) and hyperplastic epithelium is evident. (*b*) Note the pronounced epithelial and inflammatory changes and the slightly affected sweat gland.

The genus of the infecting parasites could not be determined from the fragments of microfilariae and adult filarids in the sections.

Dormant or quiescent lesions do not show the presence of microfilaria. Occasionally a section of an adult helminth is detected. In some foci of the "dormant" lesion the eosinophile is the predominating cell, whereas in others lymphocytes and histiocytes appear in great numbers. There is an apparent reduction in the cell content but the collagen fibres appear more prominent.

The changes in the epidermis vary according to the amount of damage present. There is always exfoliation. In more severely affected areas hyperplasia of the epithelium with either clubbing or elongation of the rete pegs and a variable degree of parakeratosis or hyperkeratosis are evident. Sometimes distinct papilliform structures

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are formed. In localized areas signs of malignancy become apparent, characterized by infiltrative growth of the epithelium extending for some considerable depth into the underlying vascular granulation tissue, or totally denuded ulcers may be formed. Occasionally the epithelium is distinctly acanthotic, pearl formation being evident. In one case streptothrix-like organisms and epithelial changes typical of Senkobo disease in cattle could be demonstrated[7].

All sections show more or less the same type of chronic inflammatory reaction, differing only in degree and extent. The cell content consists of lymphocytes, histiocytes, plasma cells, fibroblasts, relatively few neutrophiles and eosinophiles and occasional multinucleated giant cells. Inflammation occurs principally around and sometimes within the superficially placed blood vessels and dilated lymphatics, and to a lesser extent between the dense collagenous connective tissue fibres, where it is sparsely but diffusely distributed. The cellular reaction extends to a variable depth into the dermis often well beyond the deeply seated sweat glands. In the majority of these glands catarrhal changes are apparent, with some periglandular infiltration and occasionally even a purulent adenitis. There is no indication of any excessive secretory activity of these glands, although the superficial changes are very marked (fig. 4*b*).

These findings leave little doubt that the primary cause of the lesion is a filarid, as yet unidentified. Periodic exacerbation of such filarial infestations is known to occur in other species. It is possible that the microfilariae are transmitted by arthropods during the florid stage of the lesion. There is no evidence to support the popular belief that the condition is associated with the seasonal activity of secondary sex skin glands.

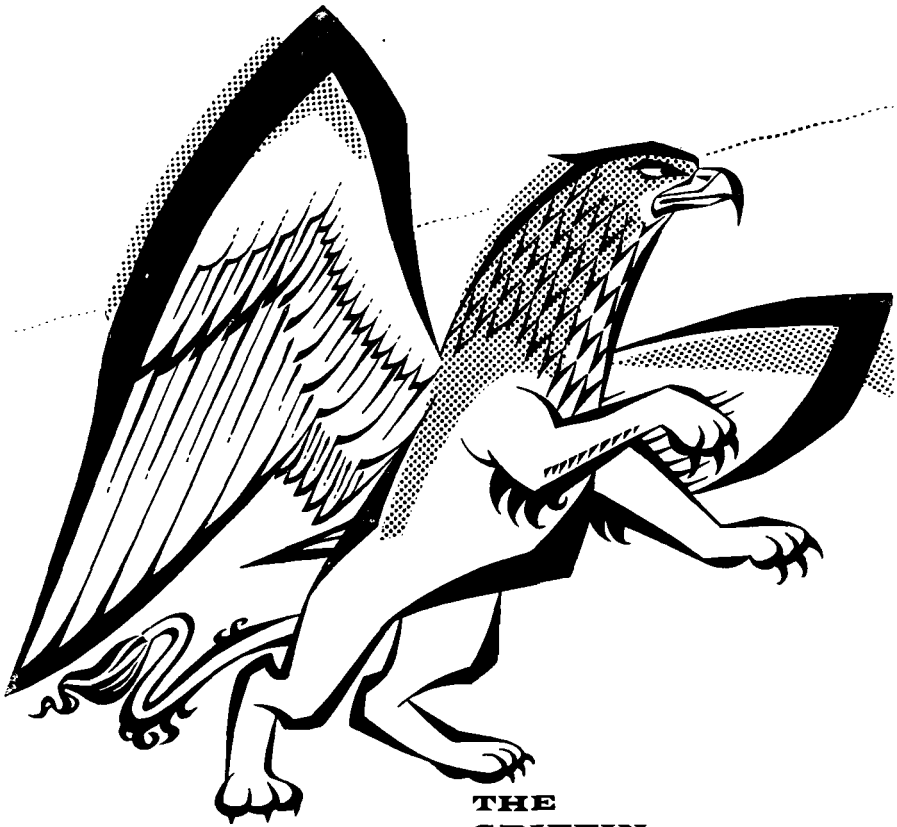
Summary

Cutaneous lesions frequently observed in the black rhinoceros (*Diceros bicornis*) and showing seasonal exacerbation have been shown to be due to a filarid infestation.

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THE GRIFFIN

This is called a GRIFFIN because it is a winged quadruped. This kind of wild animal is born in Hyperbosean parts, or in the mountains. All its bodily members are like a lion's, but its wings and mask are like an eagle's.

T. H. White *The Book of Beasts*

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THE OCCURRENCE OF A CYST OF SARCOCYSTIS LANKESTER 1882 IN THE CEREBELLUM OF AN OX.

R. D. Bigalke and R. C. Tustin.*

Sarcocystis is a protozoon found mainly in striated muscle fibres of vertebrates. Initial development is intracellular, but with growth of the cyst, the muscle fibre may degenerate leaving it lying free in connective tissue. Sarcocystis also occurs in the Purkinje fibres of the heart, and Scott[8] mentions briefly that it has been found in the lips, skin, submucous connective tissue of the intestine and dura mater of the brain. Wenyon[10] also records its occurrence in the skin.

The parasite found in cattle is known as *Sarcocystis fusiformis* Railliet, 1897[7]. It is very common in cardiac muscle. Thick and thin walled cysts, the majority of which were not visible macroscopically, were described by Fukushima.[3] In histological preparations he found that the banana-shaped spores were 8.5 to 12 microns in length and 3 to 4 microns in width. According to Knuth and du Toit[4] Sarcocystis spores measure 9 to 12 microns in length and 1 to 5 microns in width. Compartments commonly occur, but are not invariably present.

Description

In this paper it is desired to record the occurrence of a cyst closely resembling that of Sarcocystis (*vide supra*), by one of us (R.C.T.), in a haematoxylin-eosin stained routine section of the cerebellum of an ox. The animal had died from foreign body pneumonia on a farm in the Vryburg district of the Northern Cape Province. We were unable to demonstrate the cyst again in serial sections cut from the same block of tissue in which the cyst was found originally. Serial sections made from other portions of the brain did not reveal the presence of cysts.

In Figure 1 it will be noticed that the cyst is situated at the transition of the granular stratum of the cortex and the medulla of the cerebellum. Its shape is more or less elliptical, and is 142 by 77 microns in size. In Figure 3 the cyst wall and the septa of the compartments can be seen as thin lines of less than one micron in thickness. No

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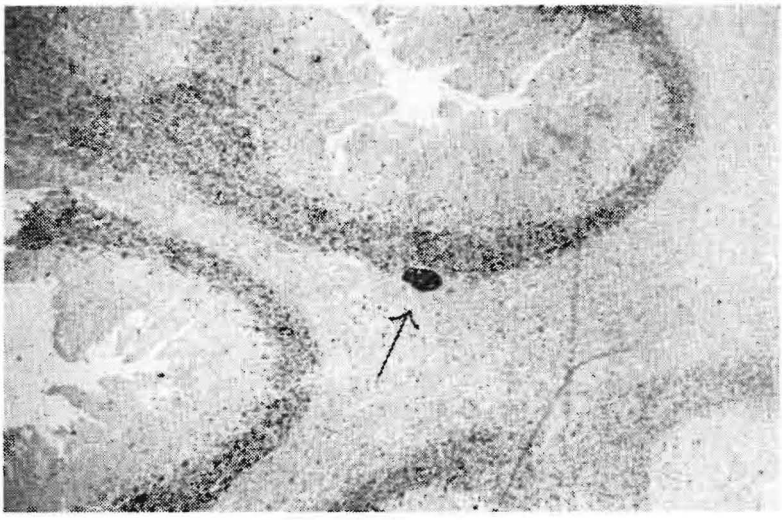


Fig. 1 Sarcocystis cyst in cerebellum of an ox .x30.

cyst wall nuclei are visible. The cyst is less compact and not as spindle-shaped as most Sarcocystis cysts are in cardiac and skeletal muscles. This is probably due to the less resistant nature of the surrounding nervous tissue. No cellular reaction on the part of the host is present.

The spores vary in shape from spherical, ovoid and elliptical to typical banana shape, depending upon the plane on which they have been cut. The banana-shaped spores measure 8.8 to 10.2 microns in

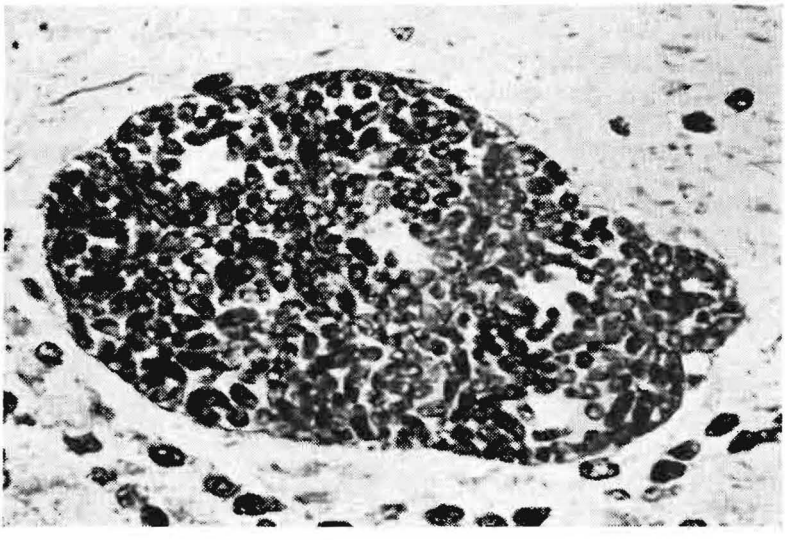


Fig. 2. Same cyst as in Fig. 1. x 480.

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length and 2.9 to 3.7 microns in width, with an average size of 9.8 by 3.3 microns. They stain a purple colour. The inner structure of the spores is not very distinct. Darkly staining portions probably represent the nuclei.

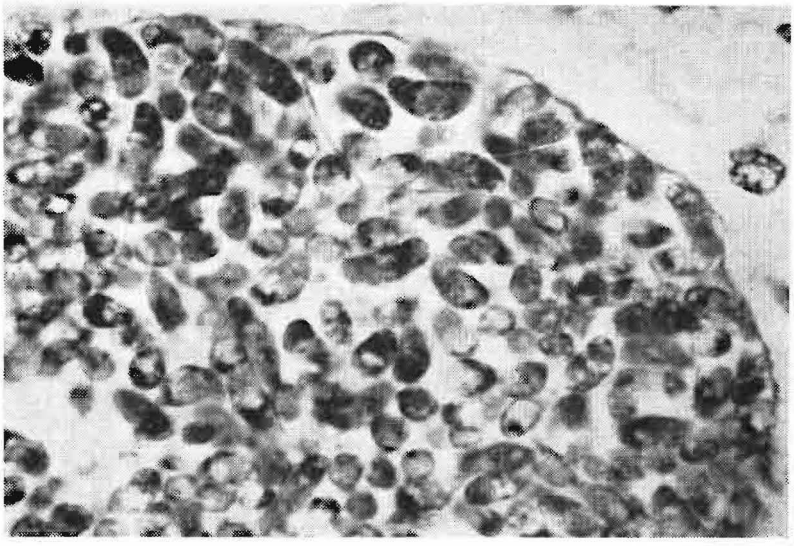


Fig. 3. Same cyst as above showing cyst wall, portions of compartmental septa and spores. x 1200.

In sections from the myocardium of this animal, stained with haematoxylin-eosin, a few typical thin walled Sarcocystis cysts were observed. They were comparable in size to the cyst in the cerebellum but were more spindle-shaped and compartments were not present. The average size of the spores was 9.1 microns in length and 2.5 microns in width.

DIFFERENTIAL DIAGNOSIS.

The following parasites were taken into consideration in the differential diagnosis:—*Besnoitia besnoiti* (Marotel, 1912), *Toxoplasma gondii* Nicolle and Manceaux, 1909, M-organism Frenkel, 1953 and *Encephalitozoon cuniculi* Levaditi, Nicolau and Schoen, 1924.

B. besnoiti is a parasite of cattle. Mature cysts measure up to 600 microns[5], and are spherical in shape. Compartments do not occur. An occasional cyst may be bi- or trilobular in appearance due to multiplication of cyst organisms in different vacuoles in the host cell. Nuclei are present in the cyst wall, which is homogenous and varies from 8 to 35 microns in thickness. In fixed preparations the crescentic cyst organisms measure from 5 to 5.5 microns in length by 1.5 to 2.2 microns in width. Cysts have not been observed in the brain. *Besnoitia jellisoni* Frenkel, 1953 and *Besnoitia bennetti* Babudieri, 1931, the aetiological agents of murine and equine besnoitiosis, are so similar to *B. besnoiti* that their morphological features need not be mentioned here.

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T. gondii is found in the brain, eye and cardiac and skeletal muscles of a variety of animals. The spherical cysts are 30 to 100 microns in size and the cyst wall is less than 1 micron in thickness[1]. There are no compartments. The cyst organisms are crescentic in shape and they are 4 to 7 microns in length and 2 to 4 microns in width[9].

M-organism: This is a temporary name for a parasite found by Frenkel[1] in the brain of *Microtus modestus* (probably *Microtus pennsylvanicus modestus* Baird, 1957) in the U.S.A. The lobulated cysts measure from 100 to 500 microns in size. The walls are less than one micron in thickness and compartments are present. The crescentic cyst organisms are 2 by 5 microns in size.

E. cuniculi has been found in the brain of mice and rabbits. The rod-shaped parasites are 2 by 2.5 microns in length and 0.5 by 1.2 microns in width[6]. The size, the complete absence of a cyst wall[2], and the fact that it stains Gram-positive make *Encephalitozoon* distinct from the other parasites mentioned here.

Conclusion

Comparison of the morphological characteristics of the cyst described above with those of the organisms considered in the differential diagnosis, makes it evident that we have been dealing with a Sarcocystis cyst.

Summary

(1) The occurrence of a Sarcocystis cyst in the cerebellum of an ox is recorded.

(2) The differential diagnosis of the cyst is discussed.

Acknowledgements

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AN OUTBREAK OF BACTERIAL ICTERUS IN SHEEP

R. C. Tustin*, T. F. Adelaar** and C. Cameron***.

A report was received from the State Veterinarian, Dundee, Dr. H. F. Strydom, during January 1960, of an outbreak of suspected geeldikkop (*Tribulosis ovis*) in a flock of sheep in the Dannhauser district. As tribulosis is unknown in Natal an investigation was carried out in the hope that some light might be shed on the aetiology of this condition. The disease, however, was shown not to be geeldikkop.

History

Early in January 1960, 300 Merino sheep were moved about 15 miles to a high-lying farm in the Drakensberg foothills in the Dannhauser district. Three weeks later the sheep were dosed with a proprietary worm remedy containing arsenic and nicotine and were placed in a new camp. Five or six days after having been dosed, deaths commenced. Over a hundred sheep died within a period of two weeks. A change was made in the grazing within two days of the first deaths but this made no appreciable difference to the course of the disease. The investigation was carried out during the terminal stage of the outbreak, when only a few deaths occurred. The mortality rate amongst affected sheep was practically a hundred per cent. The farm had carried sheep for many years.

Symptomatology and Post-Mortem Appearance.

Affected sheep lagged behind the flock with their heads down and showing rapid respiration. Some had a slightly bloody, and others a yellowish coloured discharge from the nostrils. Fever was present in some of the affected animals. According to the owner all sick sheep had shown an icterus. A clinical icterus was present in the eight cases examined. Approximately ten per cent. of the affected sheep showed a swelling of the ears and exposed areas of the head. After progressively becoming weaker, affected animals died within four to five days of commencement of symptoms.

Neither age nor sex affected the course of the disease. Many of the ewes were pregnant, but no abortions occurred.

The owner had treated five of the sick sheep by injecting a proprietary preparation containing sulphadimidine; at the time of the investigation this treatment definitely had prolonged the course of the disease.

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Eight post-mortem examinations of natural cases were performed and the following is a resumé of the pathological anatomical findings:

Generalised icterus; petechiae (sometimes ecchymoses) of the subcutis, mucosa of abomasum and of small and large intestine, urinary bladder, tongue, endocardium, epicardium, pulmonary artery (in one case) and mesenteric lymph nodes; atony of the forestomachs; bile stasis, degeneration and focal necrosis of the liver; mild nephrosis; unilateral but sometimes bilateral fibrino-necrotic pleuritis and pericarditis and a corresponding necrotic pneumonia of the apical and cardiac lobes; mild trichostrongylosis, haemonchosis, oesophagostomiasis and *Oestrus ovis* infestation.

The most striking changes were: the icterus, the fat being pigmented a canary yellow colour; the pneumonia and pleuritis, which were either only unilateral or, alternately, more severe on the right side; and the pigmentation and focal necrosis of the liver. The foci of necrosis in the liver were about one to two millimetres in diameter, irregular in shape and were distributed unevenly throughout the organ. Their number was never great.

In one case blood clots were present in the small and large intestines.

DIAGNOSIS

On the farm a tentative diagnosis of foreign body pneumonia was made, the icterus being considered to be due to the action of a haemolytic bacterium. This conclusion was reached after consideration of the following points: the farm had been grazed for many years without undue mortality in either cattle or sheep; history of dosing; the gangrenous pneumonia, predominantly right-sided in the cases examined; icterus and liver lesions; absence of any poisonous plants likely to cause an icterus*; hyperthermia shown by some of the affected sheep; the fact that a parenteral injection of a sulphonamide appeared to have some therapeutic value; and the multiple haemorrhages.

Once the farmer's attention had been directed to the pneumonia he reported that every animal he had subsequently examined had shown similar lesions.

Laboratory examination of a variety of specimens taken from two sheep (nos. 1 and 2) killed *in extremis* yielded the following results.

1. *Chemical analysis.*—A trace of arsenic was found in the ruminal contents of both sheep and in the liver and kidneys of one. The lungs (pneumonic areas) of both were negative for arsenic. Acute arsenical poisoning could thus be excluded.

The copper content of the two livers was within normal limits (360 and 338 p.p.m. Cu on a dry basis). In enzootic icterus values of 1,000 p.p.m. or more are not unusual. In geeldikkop usually a slight

*Dr. L. E. W. Codd, Assistant Chief, Division of Botany, reported that the veld was typical for that region and no poisonous plants, which were likely to have caused icterus, were encountered.

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rise in liver copper is found, though the blood copper values in geeldikkop may be as high as those seen in enzootic icterus. The difference in liver copper values may be due to the more acute course of enzootic icterus[1].

2. *Haematology.*—Examination of bloodsmears revealed mild *Gonderia ovis* infection—not considered to be of any significance—and anaemic changes. Red cell volume and haemoglobin determinations on blood samples from sheep no. 2 and from two sick sheep (nos. 3 & 4) yielded similar indications. Two of the sheep had a moderate leucocytosis. These anaemic changes are considered to be due to verminosis and nutritional factors, the more so in view of the findings in experimental cases. More significant are the figures for the van den Bergh and transaminase activity tests given in Table 1.

TABLE 1

	Sheep No. 2	Sheep No. 3	Sheep No. 4
van den Bergh (mg. %)			
Direct	6.30	0.40	1.30
Indirect	4.90	0.50	3.60
Total	11.20	0.90	4.90
Transaminase activity ..	Grossly elevated	Normal	Markedly elevated

3. *Histology.*—The histological picture confirmed the macroscopic findings. In acute cases the most striking changes occurred in the lungs and liver. The lungs showed a necrotic pneumonia with infiltration of round cells and aggregations of neutrophiles. Numerous small indistinct Gram negative cocci were present in some sections. Focal necrosis, bile stasis, cloudy swelling and mild fatty changes were seen in the liver, as well as pigmentation by bile of some Kupffer cells, normal and necrotic hepatic cells. The foci of necrosis were irregular in distribution and varied in size from a relatively few cells to many; the necrotic cells showed shrinkage, an eosinophilic cytoplasm and pyknosis or karyolysis of the nuclei. A leucostasis and a few small foci of neutrophiles were noted in the liver of one case.

In one case, which had been treated with one injection of sulphadimidine during the early stage of the disease and which had died approximately 45 days after infection, the liver showed a focal increase in connective tissue and odd cells or groups of cells were necrobiotic. A few small aggregations of neutrophiles with necrosis of the hepatic cells in their vicinity, and a leucostasis were present. Large pigment bearing macrophages were grouped especially around the central veins and portal tracts. Pigmentation of some parenchyma cells and a mild bile stasis occurred. A purulent bronchopneumonia was present. The spleen showed a purulent splenitis which was manifested by a severe increase in the number of neutrophiles.

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4. *Virological Examination of Specimens obtained from Living and Dead Sheep.*—These proved to be negative for Rift Valley fever and Wesselsbron fever by intracerebral injection of baby mice.

5. *Bacteriological Examination.*—An identical Gram negative organism was isolated in pure culture from the pneumonic areas of the lungs of sheep numbers 1 and 2. As yet it has not been identified.

A short description of the organism follows.

(*a*) *Morphology.*—A gram negative, pleomorphic organism with coccoid, diplococcoid and coccobacillary forms occasionally forming short filaments. No branching has been observed.

(*b*) *Cultural Characteristics.*—Grows with difficulty on serum agar but better growth is obtained on blood agar. No growth occurs on ordinary agar. Colonies develop after 48 hours on blood agar and are about one millimetre in diameter, are more or less flat, translucent and slightly mucoid. Colonies tend to become confluent. In serum broth, growth is slow and a slight powdery sediment is formed after 48 hours.

It is aerobic, and has an optimum growth temperature of 37°C.

(*c*) *Biochemical Reactions.*—Litmus milk is not coagulated. Nitrites are formed from nitrates. It is catalase positive, indol positive, MR negative and H_2S negative. Coagulated serum is not liquefied. Acid is formed in glucose and in maltose but no other sugars are fermented.

In view of these results it was decided to test the pathogenicity of the organism by means of a series of experiments in order to confirm the diagnosis.

SUMMARY OF EXPERIMENTAL WORK

All animals in the experiments described below, except those which were dosed with cultures or received an inoculation of a culture filtrate, received injections of bacterial suspensions in normal saline obtained from cultures grown aerobically on blood tryptose agar for 48 hours at 37°C. The density of the suspensions used was equivalent to number 5 tube of Brown's Standard Opacity tubes which is equal to 1,894 million *Escherichia coli* bacteria per millilitre.

1. *Laboratory Animals.*

The organism is pathogenic for mice, guinea pigs and rabbits. Death usually occurs after 24 to 72 hours after intravenous or subcutaneous inoculation. In more chronic cases a focal necrosis of the liver similar to that seen in sheep is observed. The organism may be recovered from the heart blood.

2. *Sheep.*

(*a*) *Routes of Infection*:

A sheep that received an intratracheal injection of 2 ml. of the bacterial suspension did not show any symptoms. A similar result was obtained after dosing 4 ml. of the suspension to another sheep.

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Subcutaneous and intravenous injections of the suspension proved to be pathogenic for sheep and the course of the disease could more or less be controlled by the dosage used.

Two sheep which received 2 ml. of a bacteria-free culture filtrate subcutaneously did not show any symptoms. The filtrate was obtained by growing the organism in serum broth for 48 hours and filtering through a Seitz filter.

(*b*) *Course*:

Of two sheep which received intravenous injections of 2 ml. and 1.5 ml. respectively of the bacterial suspension, one died within 24 hours and the other within 48 hours.

Three sheep which received injections of 2 ml. subcutaneously died within 48 to 72 hours. Of three sheep which received injections of 1 ml. subcutaneously one died after seven days and one after nine days. The other did not show any symptoms except lameness in the leg in which it was inoculated.

(*c*) *Symptoms*:

No fever was observed except in one sheep which showed a transient rectal temperature of 106.2 °F about eight hours after inoculation.

In the majority of cases the inoculation of the suspension subcutaneously into a leg resulted in a lameness of that leg. The lameness developed usually after approximately 24 hours and persisted until death. One sheep referred to above showed lameness but no other symptoms.

Within 24 to 48 hours of inoculation, depending on the size of the dose, the sheep showed inappetence and weakness, which progressed to prostration and death.

Two sheep which lived for seven and nine days respectively after inoculation both developed a clinical icterus on the fourth day. The icterus became progressively more severe until death.

The sheep which lived nine days showed a break in the wool on the eighth day.

(*d*) *Clinical Pathology*:

Specimens of blood were taken from each sheep immediately before inoculation. They were subsequently bled once a day. The blood picture of a control sheep which received the same treatment as the experimental sheep but was not inoculated, remained unaltered.

The salient points of the clinical pathology revealed in the representative tables of the blood analyses below are:

(*a*) that the blood picture is virtually undisturbed except for a slight leucocytosis,

(*b*) that there was considerable hepatocellular damage, and

(*c*) that this was followed a day or two later by considerable kidney damage.

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Table 2

Blood Analysis

Sheep 7576. Died approximately 60 hours after inoculation.

	Day	
	1 (prebleed)	2
Erythrocyte sedimentation rate (50° Wintrobe) ..	0	0
Red cell count (millions per cu. mm.)	8.43	8.46
Red cell volume (%)	42	40
Haemoglobin (g. %)	11.80	11.00
White cell count (per cu. mm.)	8,200	7,900
Blood urea nitrogen (mg. %)	18.40	20.20
van den Bergh (mg. %) Direct	0	0.45
Indirect	0	0.45
Total	0	0.90
Transaminase Activity	Normal	Normal

Table 3

Blood Analysis

Sheep 8118. Died approximately 72 hours after infection.

	Day		
	1 (prebleed)	2	3
Erythrocyte sedimentation rate (50° Wintrobe)	0	0	0
Red cell count (millions per cu. mm.)	8.40	8.44	8.33
Red cell volume (%)	40	41	41
Haemoglobin (g. %)	11.40	12.20	11.80
White cell count (per cu. mm.) ..	7,100	7,400	8,800
Blood urea nitrogen (mg. %) ..	20.20	22.10	34.90
van den Bergh (mg. %) Direct ..	0	0	0.90
Indirect ..	0	0	2.70
Total	0	0	3.60
Transaminase Activity	Normal	Slight rise	Considerable rise

Table 4

Blood Analysis

Sheep 8094.* Killed *in extremis* seven days after infection. (Sheep was not prebled.)

	Day		
	4	6	7
Erythrocyte sedimentation rate (50° Wintrobe).....	0	—	0
Red cell count (millions per cu. mm.)	6.58	6.26	6.42
Red cell volume (%)	30	27	28
Haemoglobin (g. %)	9.45	8.45	9.15
White cell count (per cu. mm.) ..	9,400	11,600	10,300
Blood urea nitrogen (mg. %)....	11	—	37.6
van den Bergh (mg.%) Direct ..	2.20	2.45	3.40
Indirect ..	1.65	1.65	1.95
Total	3.85	4.10	5.35
Transaminase Activity.....	Very high	Very high	Still high

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TABLE 5

Blood Analysis

Sheep 4920*. Died nine days after infection.

	Day							
	1 (Prebleed)	2	3	4	5	6	7	8
Temperature (°F)	104.8	103.2	103.7	103.8	103	104	104.2	103.7
Erythrocyte sedimentation rate (50° Wintrobe)	3	4	4	6	10	6	—	10
Red cell count (millions per cu. mm.)	6.01	5.99	6.27	6.30	6.01	5.81	—	5.89
Red cell volume (%)	35	35	39	38	36	33	—	31
Haemoglobin (g.%)	9.80	9.45	11.00	10.20	9.45	9.15	—	9.15
White cell count (per cu. mm.)	8,300	8,600	8,800	12,400	13,100	12,600	—	11,600
Blood urea nitrogen (mg. %)	12.9	9.2	14.7	18.4	35.0	40.5	—	123.0
van den Bergh (mg.%) Direct	0	0	1.55	4.90	4.80	5.90	5.40	6.80
Indirect	0	0	1.40	3.80	4.65	6.40	9.30	7.90
Total	0	0	2.95	8.70	9.45	12.30	14.70	14.70
Transaminase Activity	Normal	Normal	High	Higher	High	High	High	High

*Full details regarding transaminase activity, bilirubin and blood urea nitrogen of sheep numbers 4920 and 8094 are given by Malherbe[3]. (See figures 5 and 6, pages 168 and 169 of this journal.)

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(e) *Pathology:*

At the site of inoculation a very mild localised cellulitis with a small amount of pus formation was present in the majority of cases.

Animals dying of the acute infection showed the usual lesions associated with a septicaemia.

In the two more chronic cases, numbers 8094 and 4920, the changes observed were: Marked generalised icterus; subcutaneous haemorrhages; bile stasis and small foci of necrosis in the liver; hyperaemia and oedema of the lungs; petechiae of the mucosa of the abomasum and of some lymph nodes in one case; slight tumour splenis and a moderate nephroses.

(f) *Histology:*

The histological examination confirmed the macroscopic findings. The most striking changes occurred in the liver.

Sheep which died within 72 hours showed cloudy swelling, mild to moderate fatty changes and necrosis of odd liver cells.

Fatty changes, cloudy swelling, bile stasis, bile pigmentation of some liver cells and necrosis of odd cells and groups of liver cells were seen in the livers of cases of longer duration. Sheep 4920 showed a severe nephrosis.

3. *Recovery of the Bacterium from Experimental Sheep.*

Reisolation of the bacterium after experimental inoculation into sheep was not attempted in every case and when attempted was not always successful. The organism was, however, reisolated from the lymph nodes of one sheep showing clinical icterus, and laboratory animals died of an acute infection after receiving injections of organ emulsions from another.

Discussion and Conclusions

Icterus of bacterial origin is relatively uncommon in animals. That the aetiology of the icterus in the outbreak under review was due to a bacterial infection, has been proved conclusively by the facts that the same organism was isolated from the lungs of two affected sheep and that this organism, after culture, when injected parenterally into several sheep, caused an icterus and similar liver lesions.

The origin of the bacterium, however, remains speculative. It is not, as far as has been ascertained, one of the common pathogens. It seems improbable that it leads a saprophytic existence, as its optimum temperature on artificial media is 37°C and it requires the presence of serum or, preferably, blood for optimum growth. It seems doubtful that it is a common commensal. Cases of foreign body pneumonia in sheep are frequently encountered and this is the first outbreak of icterus, associated with a pneumonia, to which our attention has been directed. It is possible that the organism may be a variant strain or a mutant of a commensal occuring in the respiratory tract of sheep. The dosing fluid, which required dilution with water, or the bottle used during the dosing process, may have been responsible for the spread of the infection.

The history of dosing and the predominantly right-sided pneumonia may have been coincidental.

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Robinson[3,4] described an outbreak of bacterial icterus in sheep due to a Corynebacterium infection following blue tongue vaccine inoculation. He considered that the icterus was due to haemolysis; a haemoglobinuria was present in cases which were produced with the organism. Ryksen encountered cases of icterus after inoculation against blue tongue and enterotoxaemia[5]. In the experimental cases which were produced with the present organism no change was observed in the erythrocytic blood picture nor was haemoglobinuria seen, although anaemic changes were present in some of the natural cases. It is considered that the icterus is primarily of hepatic origin, this being confirmed by the van den Bergh tests and the histological picture. The results of the transaminase tests also indicate liver damage.

That the infection becomes septicaemic and affects the parenchymatous organs, the liver and kidney in particular, is confirmed by the chemical tests and the facts that the organism was reisolated from lymph nodes of one case and that the liver and other organs presumably contained the organism in another.

In contrast to Robinson's findings only a mild localised cellulitis is produced at the inoculation site when the organism is inoculated subcutaneously. No abscess formation was noticed.

Summary

(1) About 30% of a flock of 300 sheep died showing icterus and pneumonia. 10% of the affected animals showed photosensitisation. A description of the symptomatology, pathology, bacteriology and experimental work is given.

(2) It has been conclusively proved that a Gram negative bactrium, as yet unidentified, was responsible.

(3) Experimental subcutaneous inoculation of the organism into sheep resulted in either an acute septicaemia or a more portracted illness of which hepatic damage and icterus were the salient features. The course could more or less be controlled by the size of the dose. The organism is also pathogenic for rabbits, guinea pigs and mice, the only experimental animals tested.

Addendum

Since submitting this article for publication further outbreaks in sheep have been encountered. The pneumonia, however, was not a consistent feature. An identical bacterium has been isolated from affected sheep. These outbreaks will be reported upon later.

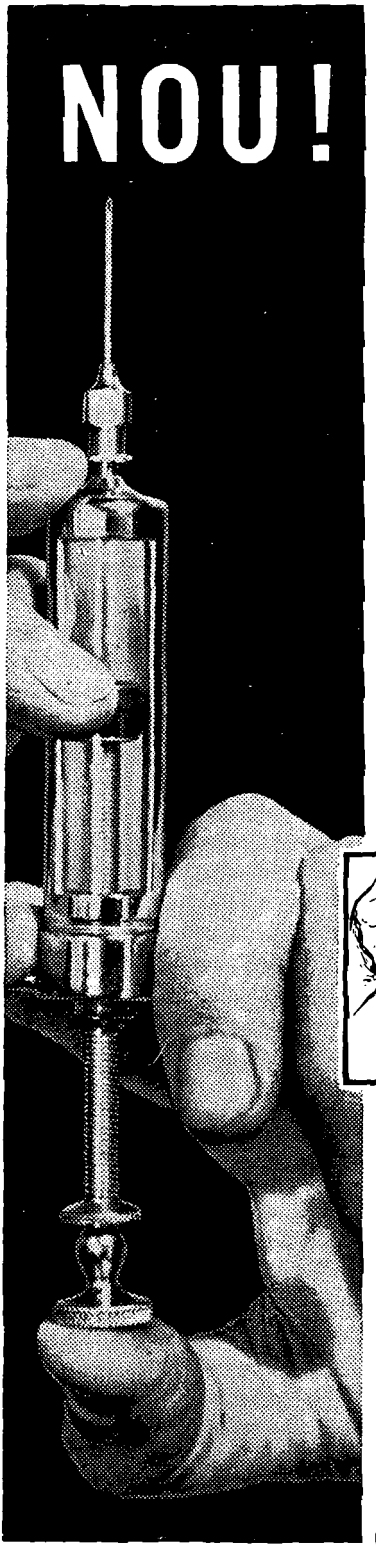
Acknowledgements

The Director of Veterinary Services is thanked for permission to publish this article. Thanks are also due to Dr. W. D. Malherbe and Mr. W. Haupt for the blood analyses.

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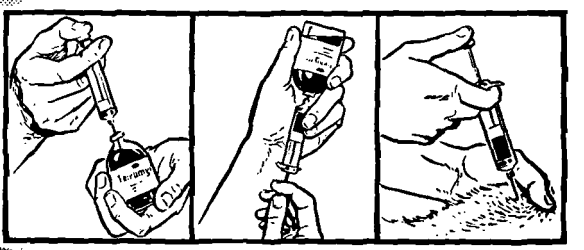
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REACTIONS TO HEARTWATER IMMUNIZATION SHOWN BY CATTLE

G. D. Sutton*

During the latter half of 1958 a number of Afrikander, Friesland, Red Poll and Sussex cattle ex Armoedsvlakte, Vryburg, were immunized against Heartwater at Onderstepoort. The group consisted of 141 cows, and 69 calves ranging from one week to three months old. The infective dose consisted of 5 ml. infected sheep blood as issued by Onderstepoort. This blood is frozen and stored on dry ice. The animals were temperatured daily between 8 a.m. and 10 a.m. The normal temperatures ranged from 101°F to 102°F except for a few calves which showed temperatures of 101°F to 103°F. A sharp rise in temperatures to over 103°F was regarded as a reaction and treatment was given immediately. This consisted of an intramuscular injection of "Terramycin Soluble Powder" (Animal Formula) given at the rate of 50 gms. for cows and 20 gms. for calves irrespective of weight.

REACTIONS IN THE COWS

Of the 141 cows injected, 18 showed no temperature reaction. Of these, two died. One died three days after the injection, the cause of death could not be established, but heartwater was excluded. The second collapsed and died of peracute heartwater on the 13th day after injection, without showing any rise in temperature.

The remaining 123 cows reacted on the following days after the injection.

11th day — 1	19th day — 13
12th day — 8	20th day — 4
13th day — 1	21st day — 3
14th day — 17	22nd day — 4
15th day — 17	23rd day — 0
16th day — 15	24th day — 2
17th day — 21	25th day — 0
18th day — 15	26th day — 2

Five of these reactors died. Their history was as follows:—

(1) Reacted on the 17th day after injection and was treated. The temperature dropped and the animal appeared to be recovering but died on the 19th day without showing any rise of temperature.

(2) Reacted on the 17th day after the injection, was treated but died the next day.

*Division of Veterinary Services, Onderstepoort.

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(3) Reacted and treated on the 19th day after the injection. The temperature dropped and the animal appeared to be recovering but died on the 21st day without showing any further rise in temperature.

(4) Reacted and treated on the 20th day. The temperature continued to rise. It was treated a second time on the 21st day but died on the same day.

(5) Reacted and treated on the 21st day. The temperature dropped. It remained ill without showing any temperature rise and was treated again on the 23rd day. There was no rise in temperature but it died on the 25th day.

RESPONSE TO TREATMENT

The following types of response to treatment were exhibited by the 118 reactors which survived.

Those requiring one treatment

87 cows were treated on the day of the reaction. Their temperatures dropped and they showed no further symptoms.

Those requiring two treatments

Four cows treated on the day of reaction still showed elevated temperatures the following day. A second treatment was followed by recovery.

Two cows relapsed on the second day after treatment and required a second injection.

Twenty-two cows showed delayed relapses after treatment. The intervals between the initial treatment and the second rise in temperature were as follows:

2 days — 8 cows
3 days — 5 cows
4 days — 6 cows
5 days — 1 cow
10 days — 1 cow
11 days — 1 cow

All were given a second treatment on the day the temperature rose for the second time and recovered thereafter.

Those requiring Three Treatments

Three cows required three treatments. The intervals between elevations of temperature were as follows:—

Cow	1	2	3
Days between 1st and 2nd treatment	5	13	2
Days between 2nd and 3rd treatment	2	2	2

The losses amongst the cows were therefore 6 out of 140 or 4.3 per cent.

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REACTIONS IN THE CALVES

Of the 69 calves, 13 showed no temperature reaction or other symptoms.

The remaining 56 calves reacted on the following days after the injection.

7th day — 1	17th day — 4
10th day — 0	18th day — 5
11th day — 0	19th day — 2
12th day — 0	20th day — 6
13th day — 6	21st day — 4
14th day — 5	22nd day — 2
15th day — 10	23rd day — 3
16th day — 7	

They were all treated on the day of reaction and showed the following responses:

In fifty calves a single treatment was followed by permanent recovery.

Two calves required treatment on two consecutive days.

Four calves showed a secondary rise in temperature between 5 and 7 days after the first reaction.

There were no deaths among the calves.

Summary of Reactions

The reactions can therefore be summarised into 7 types viz:—

Type of Reaction	Number of cases	
	Cows	Calves
1. No reaction, no treatment	16	13
2. Reaction controlled by one treatment	87	50
3. Initial reaction requiring two treatments	4	2
4. Initial reaction treated. Second reaction 2–11 days, later successfully treated	24	4
5. As for group 4 but followed by a third reaction 2–6 days later	3	0
6. Died despite treatment	5	0
7. Died before treatment given (no temperature reaction)	1	0
Total	140*	69

*The cow that died of other causes is not included.

THE INFLUENCE OF BREED

These cattle came from herds which had been under close supervision for some 30 years and had been reared under identical conditions. The work therefore offered an excellent opportunity to examine any breed variations in susceptibility to heartwater which may exist. An analysis of the results reveals the following:—

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SUMMARY OF REACTIONS ACCORDING TO BREED.

ADULT CATTLE

Breed	Reaction Died	No reaction Died	No reaction Lived	One treatment Lived	Two treatments Lived	Three treatments Lived	Total each Breed
Friesland	2	0	5	22	8	—	38
Afrikander ..	1	1	5	20	5	1	33
Sussex	1	—	5	26	8	1	41
Red Poll	1	—	1	19	7	1	29
Total reactions	5	1	16	87	28	3	141

CALVES

Breed	No Reaction Lived	One Treatment Lived	Two treatments Lived	Total each Breed
Friesland	3	3	5	11
Afrikander	3	14	—	17
Sussex	6	21	—	27
Red Poll	1	12	1	13
Total reactions	13	50	6	69

No differences could therefore be seen between the various breeds represented.

SUMMARY

Details of the reactions of 141 cows and 69 calves to artificial infection with Heartwater and treatment with Terramycin are given.

No breed difference in susceptibility to Heartwater could be established between Afrikanders, Friesland, Sussex and Red Poll cattle.

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CANINE ANTI-HAEMOPHILIC GLOBULIN DEFICIENCY—A CASE REPORT

H. G. Purchase and* H. B. W. Greig.**

A haemophilia-like disease in greyhounds was first reported by Merkins[1] in 1938, according to Field *et al.*[2], who also described the condition in dogs.

Graham *et al.*[3] described it in Irish Setters. They showed it to be identical with human haemophilia and that the defect in dogs could be corrected by injection of human or bovine plasma protein Cohn Fraction I. The chief symptoms are subcutaneous haematomata and haemarthroses. Brinkhous and Graham[4] did breeding experiments with a number of male and female haemophilic dogs. They found the haemophilic gene to be sex-linked, the heterozygous female being the carrier. They also discovered that the homozygous female was a typical haemophilic (a condition which has only been described three times in women[5,6]). Spontaneous mutation accounts for about 1/4 to 1/3 of the cases of human haemophilia; whether this occurs in dogs or other animals is not known.

Haemophilia-like disease has also been described in the pig[7,8] but no recent studies seem to have been reported.

An interesting situation exists in respect of the horse. Bell *et al.*[9] reported that normal horses have a deficiency of anti-haemophilic globulin while Soulier and Larrieu[10] were not able to confirm this. Sjliön[11] found a Christmas factor (Factor IX, Plasma Thromboplastin Component, P.T.C.) deficiency in normal horses. Ollendorf[12] re-examined the question, investigating thirty-five apparently normal horses at the Danish State Serum Institute, Copenhagen. He found that eleven out of thirty-five had various coagulation defects including anti-haemophilic globulin deficiency and Factor IX deficiency. He showed, however, that in the same horse, in vitro testing reveals marked variability of thromboplastic activity from time to time.

History.

The patient is a twelve months old male golden brown cocker spaniel, whose pedigree is unfortunately unavailable. It was bred in South Africa. A large swelling developed on the forehead seven to eight months before being seen at the clinic. The swelling gradually decreased in size over a period. At intervals thereafter swellings developed on the hind legs, forelegs and side. As far as is known, these haematomata were not always associated with a history of trauma.

Condition on Admission.

The dog was thin, but very alert and interested in his surroundings. His mucous membranes were pale and he had a tachycardia with a

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TABLE 1. RESULTS OF LABORATORY EXAMINATIONS

	Test	Units	Date 28/9	3/10	3/10	5/10	15/10	Normal Values and Remarks
1.	Blood Smear		-ive	-ive		-ive	-ive	For Babesia canis.
2.	Bleeding Time	Mins	3.5					2–3
3.	Coagulation Time	Mins	10					4–10
4.	Prothrombin Time	Secs	8.2					10–17
5.	Sedimentation Rate	m.m.	2.5				5	0–5
6.	Packed Cell Volume	%	21.7				32	39±5.8
7.	Haemoglobin Concentration	Gm %	6.3			10.4	12.2	13±1.8
8.	Erythrocyte Count	103/cu.mm.	3.0				5.7	6.1±1.0
9.	Leucocyte Count	106/cu.mm.	21				9.6	11.3±3.3
10.	Differential Count:							
	Neutrophils	%		76.6		79.3	66.6	74±7
	Lymphocytes	%		15.0		11.3	17.3	20±5.4
	Eosinophils	%		2.6		5.0	12.6	2±2
	Monocytes	%		6.0		4.6	3.3	4±3
11.	Transaminase S.GO–T	King	50					Normal
	S.GP–T	King	48					Normal
12.	Mean Cell Volume	Cu. μ	72				57	59±8
13.	Mean Cell Hb. Conc.	%	29				38	34±4.5
14.	Faeces Examination		+				+++	For Ancylostomes

Blood Transfusion Given

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slight systolic murmur and a tonsillitis. Over the left scapula there was a large haematoma, about 200 c.c. in volume, which was not attached to the underlying tissue. There was a line of petechiae at the gingival border opposite the lower left carnassial tooth. The teeth had a soft brown surface deposit.

Apart from the above, nothing else abnormal was found on clinical examination.

Diagnosis.

After completing the examinations (28th September, 1959) shown in Table 1, a provisional diagnosis of an idiopathic haemorrhagic state was made. This was accompanied by tonsillitis, severe anaemia and verminosis. The results of laboratory examinations carried out on the dog during his stay at the clinic are summarized in the table.

(Page facing).

The haemorrhagic state could have been due to defective capillary haemostatic function or to a coagulation defect. The clotting time, at the upper limit of normal, suggested the latter; the bleeding time was normal. A thromboplastin generation test was carried out and the results are shown in Table 2.

TABLE 2

RESULTS OF BIGGS AND DOUGLAS THROMBOPLASTIN GENERATION TEST

Reaction	1	2	3	4	5
Source of: Adsorbed Plasma	N. Human	Dog	N. Human	Dog	Equal parts Dog + human (Haemophiliac)
Serum	N. Human	Dog	Dog	N. Human	N. Human
Platelets ..	N. Human	N. Human	N. Human	N. Human	N. Human
Incubation Time	Substrate Clotting Times (Seconds)				
1 minute	35.6	31.6	10.0	33.2	47.0
2 minutes	24.4	25.8	10.8	27.6	41.2
3 minutes	17.0	26.6	12.0	32.0	44.0
4 minutes	13.4	26.0	13.8	30.8	38.0
5 minutes	14.8	26.8	15.0	31.6	39.6
6 minutes		26.4		33.6	
7 minutes		22.7			
8 minutes					

Column (1) shows the control normal thromboplastin generation.

Column (2) indicates a severe abnormality with the dog's components in that the substrate clotting time remained long, even after seven minutes: i.e. no significant amount of thromboplastin was formed.

Column (3) The adsorbed plasma was substituted with normal human adsorbed plasma and resulted in normal thromboplastin generation.

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Column (4) Human normal serum was substituted for the dog's serum with no improvement, indicating that the defect lay in the dog's plasma.

This defect could either be due to factor V or Anti-Haemophilic Globulin (A.H.G.) deficiency. As discussed below in factor V deficiency the prothrombin time is lengthened. In this case it was normal. Therefore, the diagnosis of an A.H.G. deficiency was made—a condition corresponding to haemophilia in man.

As confirmation of this, the dog's plasma was used in equal quantities with human haemophilic plasma. The result is shown in Column (5). The dog's plasma was unable to correct the abnormal times obtained when using plasma from a human case of haemophilia.

Treatment.

A blood transfusion was given on the fifth day and the dog received 54 mgm. of iron intramuscularly and 50 mgm. of Vitamin B12 every –06 days. The response to this therapy can be seen in Table 1.

The tonsillitis did not respond to the usual antibiotics. Erythromycin was, however, found to be the one of choice after sensitivity tests had been done on cultures from tonsillar swabs. This treatment improved the tonsillitis, but did not cure it.

No vermifuge was given until 18th October, 1959, because the animal was so weak and the worm burden was not significant.

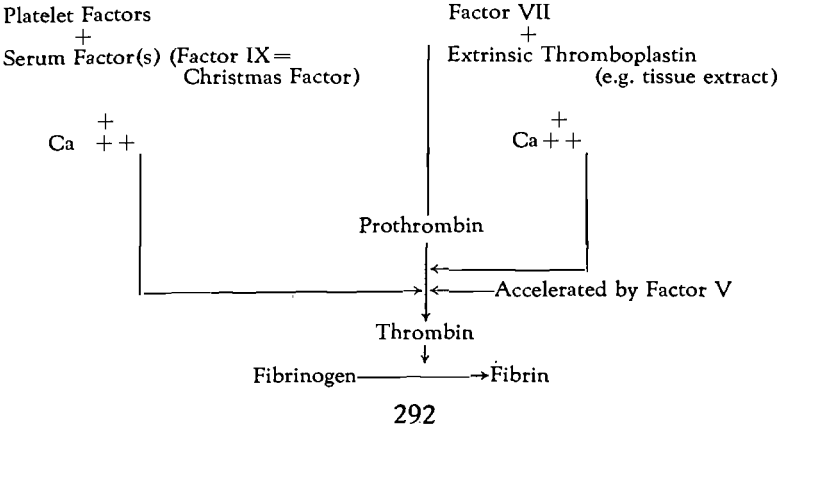
During the stay in hospital the dog put on one kilogram bodyweight and on discharge appeared clinically normal.

Of interest is the fact that the haemorrhagic state has apparently been controlled for about 6 months by this single transfusion; a second transfusion was given in April, 1960.

DISCUSSION

Figure 1 shows the most widely held current views on coagulation theory.

FIGURE 1. OUTLINE OF CURRENT THEORY OF COAGULATION.



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Any deficiency of the factors shown on the right hand side of figure 1 will result in an abnormal prothrombin time ("prothrombin index"). Thus a deficiency of Factor VII, found in conditions of Vitamin K deficiency and following administration of the coumarin group of drugs, results in a long prothrombin time. A deficiency of Factor V (which as far as the authors are aware has not been reported in animals of economic importance) will also result in a long prothrombin time. Theoretically, a deficiency of prothrombin itself will result in a long prothrombin time, but this is found very rarely as a primary disease; it may occur in acute liver failure.

For long it was believed that the intrinsic thromboplastin of blood was weak. Biggs, Douglas and MacFarlane[13] did a signal service to the study of coagulation defects when they showed that the intrinsic thromboplastin was as powerful as any extrinsic thromboplastin, but its generation takes several minutes in shed blood. From this observation was born the Thromboplastin Generation test[14] which is, par excellence, the test for evidence of deficiencies of any of the factors shown on the left side (Intrinsic System) of Figure 1.

In the Thromboplastin Generation test a suspension of platelets (to supply several platelet factors), diluted serum (to supply serum factor IX), adsorbed plasma (to supply plasma factors V and A.H.G.) and calcium chloride solutions (M/40) are incubated together. At minute intervals during the incubation, samples are removed from the mixture and transferred to normal citrated plasma and recalcified simultaneously. The time taken for the plasma to clot is noted. In effect this last stage is the same as a prothrombin time test, only, instead of using an extract of brain as a thromboplastin, the plasma thromboplastin generated in the reaction mixture is used. As normal plasma is used as substrate, containing normal amounts of prothrombin, Factor V and Factor VII, the clotting time of this substrate is a measure of the thromboplastin added from the generating mixture.

Adsorbed plasma is normal citrated plasma which has had its prothrombin and Factor VII or its precursor removed by treatment with alumina (aluminium hydroxide). This removal of prothrombin is necessary as the presence of prothrombin in the generating mixture interferes with the test. After adsorption Factor V and A.H.G. are still present (i.e. they are not adsorbed).

The thromboplastin generation test can detect deficiencies of serum factors, plasma factors or platelet factors. By suitable mixing experiments, using sera or plasma with known deficiencies, the nature of the defect in the test plasma or serum can be defined. It will be noted that A.H.G. and Factor IX (Christmas Factor) deficiencies cause defective thromboplastin generation but do not affect the prothrombin time, while Factor VII seems to have no function in the thromboplastin generation test but a deficiency of this factor causes an abnormal prothrombin time. In Factor V deficiency in both tests are abnormal.

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TABLE 3

SUMMARY OF RESULTS OF THE PROTHROMBIN TIME TEST AND THE THROMBOPLASTIN GENERATION TEST IN CERTAIN COAGULATION DISORDERS

Disease or Deficiency	Result of	
	Prothrombin Test	Thromboplastin Gereration Test
Haemophilia ..... (A.H.G. deficiency)	Normal	Plasma defect
Factor IX deficiency .... (Christmas Disease)	Normal	Serum defect
Factor VII deficiency	Prolonged	Normal
Factor V deficiency	Prolonged	Plasma defect

The introduction of the thromboplastin generation test has been an advance of the greatest moment for the satisfactory diagnosis of coagulation disorders. Unfortunately, it is not a simple test and requires some skill for successful performance. It is not a test which lends itself to sporadic performance and hence it is best carried out in centres which are called upon to perform the test frequently. With modification, it can be used to measure the magnitude of the deficiency in question. It does not seem to have been widely used as yet in veterinary medicine.

Treatment of anti-haemophilic globulin deficiency is by fresh plasma or fresh blood transfusion. Stored or freeze-dried plasma is of no value as A.H.G. is labile and destroyed by storage or freeze drying. In emergency, i.e. post-operatively or following injury, large amounts of fresh plasma (or blood—but plasma is better as there is little danger of circulatory overload) should be infused as rapidly as possible. The aim is to raise the A.H.G. levels in the blood to above the level (about 5–10% of the normal) at which coagulation proceeds normally.

SUMMARY

The literature on haemophilia-like diseases in domestic animals is reviewed briefly. A case of haemophilia (anti-haemophilic globulin deficiency) in a spaniel is described. An outline is given of the modern theory of blood coagulation and the most important recent advance in the diagnosis of coagulation defects, the thromboplastin generation test of Biggs and Douglas, is discussed in some detail.

ACKNOWLEDGEMENTS

Thanks are due to the Director of Veterinary Services, and to the Professor of Medicine, Onderstepoort, for permission to publish this case report. Thanks are also due to the Director and Deputy Director, South African Institute for Medical Research, Johannesburgh, for facilities and encouragement.

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ACUTE MERCURY POISONING IN A DOG.

R. C. Tustin*, J. A. Minne,** T. F. Adelaar**.

This case of mercury poisoning in a dog is of interest as it was caused by the inhalation of mercury vapour: a relatively rare occurrence in animals. The dog and several chickens were brought to this laboratory for post-mortem examination, following the deaths of another dog, about 200 chickens, and five people.

History

It is alleged that the mercury had been used in an attempt to extract gold from some supposedly gold-bearing material, by placing the mixture on a stove in a crowded kitchen in which the animals also were present. The dog died approximately 72 hours after exposure to the mercury vapour. Unfortunately the symptomatology of the case could not be obtained.

Post-mortem Examination.

The following changes were most noticeable:—Generalised venous congestion and cyanosis; hyperaemia and a severe oedema of the lungs; nephrosis; severe congestion of the liver; mild hyperaemia of the mucous membrane of the stomach and intestine; chronic purulent conjunctivitis; severe congestion of the meningeal vessels, and a dirty greyish colour of the brain substance. The stomach contained about five millilitres of bile and the intestines a small amount of semi-solid ingesta.

The most striking changes present were the congestion of the meningeal vessels and liver, the discoloration of the brain, and the hyperaemia and oedema of the lungs.

At the time of post-mortem examination a diagnosis of "acute poisoning, aetiology to be determined" was made, and material taken for chemical tests.

Histopathology.

The macroscopic lesions were confirmed histologically. The most apparent changes occurred in the kidney and liver. The kidney showed a severe nephrosis, characterised by cloudy swelling especially of the proximal convoluted tubules, fatty degeneration which was most marked in the ascending limbs of Henle, and the formation of albuminous casts. Fatty degeneration and cloudy swelling were also present in the liver.

Chemical Analyses.

The organs of the dog contained the following amounts of mercury per 100 gm. of the fresh material:

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Liver, 0.6 mg.; lung, 0.6 mg.; kidney, 3.6 mg.; brain, 14.0 mg.; the stomach, intestines and their contents together, 0.1 mg. and the blood a trace.

Ten chickens were examined. The organs were removed, pooled according to type, and the fresh material analysed. The liver contained 1.4 mg.%, brain 0.5 mg.%, stomach contents 2.0 mg.% and the kidney 1.3. mg% mercury.

Discussion.

Mercury has a freezing point of —39°F and a boiling point of 30°6 F. The metal is appreciably volatile even at ordinary temperatures. According to Smith and Jones[1], acute mercurial poisoning results chiefly from the accidental ingestion of mercuric chloride, or of the organic mercurial fungicides. It may also accur as the result of excessive absorption of mercuric chloride antiseptic solution, or possibly of mercury iodide used as a counter-irritant. They consider when mercury poisoning is encountered in domestic animals, it usually results from the feeding of animals, chiefly swine, with grain which has been set aside for use as seed and treated with an organic mercurial fungicide.

Fröhner[2] on the other hand, states that most cases of mercurial poisoning are caused by medicaments given internally: the extremely toxic properties of the substance often being underestimated. He states, too, that poisoning by the inhalation of mercury vapour should also be taken into account and cites two cases; one where the crew of a ship and all the animals on board succumbed after mercury had escaped into the hold, and the other where cats in a thermometer factory developed mercurial poisoning.

Petrelius[3] described the case of aerogenous mercury poisoning from Harbitz in Norway where 24 cattle were concerned. To one of these mercury ointment (Ung. resolvens) had been applied to the udder. In the course of one month 12 animals became sick, eight of which died: the rest recovering. The organs contained so high a mercury content that mercury poisoning was thought certain. He stated that no conclusive evidence had been produced to prove that mercury poisoning occurred aerogenously and then described a series of experiments which proved that it could, in fact, occur. Briefly his method was to cover the hind legs of a horse on several occasions with "grey ointment," which contained about 30% mercury. Sheep and cattle placed in the same stall developed symptoms of mercury poisoning: some of the sheep eventually dying. The animals showed dyspnoea, coughing attacks, anorexia, marked languor, and lay down most of the time. Some showed fever, diarrhoea and albuminuria.

Garner[4] states that in acute cases the post-mortem findings are gastro-enteritis and acute parenchymatous nephritis. After inhalation of mercury vapour there may also be oedema of the lungs, hydrothorax, hydropericardium, subpial oedema and haemorrhages in the epicardium and endocardium.

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Among the domestic animals, ruminants are considered to be the most susceptible to mercury poisoning. Poisoning by inhalation is rare, and in the case under review the fact that the intestines contained such small amounts of mercury, and the other organs relatively larger amounts, does point to a route of intoxication other than by ingestion.

The discoloration of the brain is of interest, as a similar picture is often seen in lead poisoning.

Summary.

The pathology of a case of mercury poisoning by the inhalation of metallic mercury vapour is described. The most apparent lesions were congestion of the meningeal vessels and liver, a greyish discoloration of the brain, nephrosis and hyperaemia and oedema of the lungs.

Acknowledgements

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UTERINE FIBROMA IN A BITCH

C. F. B. Hofmeyr*

Fibroma in the uterus of the bitch occurs very rarely, according to the author's experience and judging from the paucity of references in the literature.

The importance of the present case lies in the fact that clinical and operative appearances indicated a very grave prognosis due to a strong suspicion of malignant neoplasia. This is intended as a warning that unless actual metastasis can be demonstrated, the surgeon should await the opinion of the pathologist before giving a hopeless prognosis or worse still, carrying out euthanasia.

Subject.

The patient was an aged black Alsatian bitch.

Anamnesis.

The dog was noticed to lose condition while developing an increased abdominal circumference. As she had been in oestrum some time before, this was regarded as a sign of pregnancy. As impending abortion was suspected, the bitch was presented for examination.

Examination.

Although the animal was well cared for, her condition was poor. General clinical examination was negative except for fluid in the abdominal cavity and a very large tumour considered to be part of either the tip of the uterus or the spleen.

Diagnosis.

Neoplasia of either the uterus or spleen, associated with ascites. A grave prognosis was given, operation advised and agreed to.

Operation.

Sagatal (M & B) anaesthesia was employed and blood given by intravenous drip. The abdomen was entered through an ample incision in the linea alba. About two litres of wine-coloured fluid escaped. A sudden fall in blood pressure was feared and vasopressors were held in readiness but their use was not necessary. The tumour delivered through the incision proved to be ovoid in shape, smooth surfaced, slightly flattened and having a length of about 20 cm. A panhysterectomy was then performed and the abdomen explored for metastases, but none was found. The wound was closed in the usual manner.

The patient stood the operation surprisingly well and made an uneventful recovery.

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A specimen was sent to the Department of Anatomy and Prof. H. P. A. de Boom reported as follows: Tumour—fibroma with considerable necrosis in the centre. It is suggested that it might have arisen from the fallopian tube.

Discussion.

It is very well known that neoplastic malignancy causes loss of condition. The additional clinical findings of a hard mass in the abdomen and ascites appeared to leave little room for doubt that the growth was cancerous.

At operation these fears were reinforced especially when the wine-coloured fluid escaped with the suggestion of ulceration of the tumour or possibly of its secondaries.

It appears that the loss of condition must be ascribed to toxaemia emanating from the necrosed tumour tissue.

This operation was done more than a year ago and the bitch is in excellent condition.

Summary.

A case of uterine fibroma in a bitch, closely simulating a malignant tumour clinically, and the successful surgical removal of the tumour, are described.

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BILATERAL MANDIBULAR FRACTURE IN A BOVINE

D. H. G. Irwin.*

Introduction.

Freudenberg[2] considers fracture of the lower jaw rare in large animals. He found seven cases in 5,000 equine surgical cases, and adds that satisfactory healing takes place in about six weeks. Barrairon and Lebailly[1] describe a case of comminuted fracture of the lower jaw in a horse which they treated successfully. No previous record of bilateral mandibular fracture in the bovine could be found.

The Subject and History.

This was a two-and-one-half year old grade Afrikander heifer. She was about four months pregnant, and in good condition until meeting with an accident some 14 days previously, when she fell heavily on her lower jaw whilst attempting to leap a fence. She had drunk water, but had neither eaten nor ruminated since the accident.

Examination.

Upon examination on 22/7/59 the heifer was in poor condition and mildly dehydrated. The tongue protruded from the mouth, which hung open, and both mouth and tongue were soiled with sand. The tongue was retracted in response to pain stimuli. The left ramus was fractured immediately rostrad to the first premolar, and the right ramus between premolars 2 and 3. The mucous membrane was broken at these sites, and swelling was present. The rumen was atonic and contained little ingesta. Rectal faeces were scanty and dryish. The heifer was highly spirited however, and determinedly resisted handling.

Surgical Treatment.

On 22–23/7/59 supportive treatment was undertaken to correct fluid balance, one litre of physiological saline with 5% dextrose daily being administered.

On 24/7/59 250 mgm chloropromazine ("Largactil", May & Baker) was injected intramuscularly an hour and a half prior to the operation, and the left flank (rumenotomy site) prepared for surgery.

Under lignocaine ("Xylotox", Pharmaceutical Mfg. Co.) local infiltration the rumen was exposed as for rumenotomy and its wall sewn to the lips of a 10 cm. skin incision. (Tge second stage in producing a ruminal fistula was delayed until the following day.) The heifer was then anaesthetized with 10% chloral hydrate solution, with ½ gr. atropine sulphate, intravenously. Since premolars 1 and 2 on the left and premolar 3 on the right side were loose, they were extracted. The fracture was set and a cast of plaster of paris applied.

The following day a circular portion of the exposed ruminal wall was excised to complete the fistula, and artificial feeding begun.

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The Feeding Program.

Professor Clark of the Department of Physiology kindly advised the author on the nutritional aspects of the case. Accordingly the following materials were administered via the fistula: one litre of freshly collected sheep ruminal ingesta, 0.5 Kg. sugar, 20 ml. glacial acetic acid, 1 Kg. of the usual Onderstepoort ruminant concentrate meal, 1.3 Kg. lucerne-hay meal along with 4.5 L. water. It was found later that a cylindrical equine vaginal speculum fitted the fistula, and this was left *in situ* as a semi-permanent funnel. The outer opening was plugged with cottonwool to exclude air and encourage the correct ruminal digestive processes.

At the time of the first feeding, a second feed of concentrate and hay-meal was set to soak in about 5 litres of water. The second feed was given about 9.30 p.m. but because the rumen was too full to take the full amount, half the lucerne hay-meal was omitted from the feed. The rumen was topped up twice daily with water between meals. The feeding proceeded as described above for the next three days, but on the 29th the rate of rumen emptying was very slow, whereupon 15 ml. glacial acetic acid diluted in about 3 litres of water, was administered and feeding omitted. Rumen level was still static on 30th, so acetic acid dosing was repeated. On the 1st August the indigestion had not improved so 500 ml. sheep ruminal ingesta plus 12 L. water was administered, with a dramatic improvement by the next day, when feeding was resumed.

For the next two days, 3rd and 4th August, the dosing of sheep's rumen contents was repeated, and on the 5th the heifer had diarrhoea. Her general appearance, however, improved quite noticeably. The

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diarrhoea was treated by discontinuing the dosing of rumen contents. By 8th August the faeces were normal. All went well until 14th August when constipation was noticed. This was treated with 0.5 Kg. sugar and 15 ml. propionic acid suitably diluted. Ruminal emptying was delayed and on 15th 1 L. sheep rumen content was dosed. Although digestion was proceeding well on a diet of ± 700 gm. concentrate meal and 850 gm. lucerne hay meal, on 16th the heifer seemed to be loosing bloom. On 19th 300,000 i.u. vitamin A and 3 ml. Plebex (Wyeth) were injected. On 20th 1 L. rumen content was dosed along with about 500 gm of ground feed. On 21st about 800 gm feed and 9–10 L. water were well used and all appeared to be going well. The heifer was in good flesh, and bloom was evident again.

Additional nursing treatment and observations.

Soiling of the flank occurred from time to time when ingesta overflowed through the fistula. This was handled by washing and drying and then applying vaseline—no skin damage occurred. The nose needed cleansing on several occasions since the heifer was not able to protrude the tongue to do it herself.

On 3rd August the plaster was reinforced with more plaster of paris. The mouth was irrigated when feed was noticed to have accumulated there—a fact to which attention was drawn by restricted chewing movements.

On 21st it was decided that the cast would be removed on the 23rd.

On 22nd August the heifer was found dead in her box.

Post-mortem Examination.

The cause of death could not be established. The pathological anatomy included sub-epicardial and sub-endocardial petechiae, an creased volume of animniotic fluid and chronic hypertrophic cholecystitis. A small quantity of ruminal contents was found in the buccal cavity. The left ramus was more firmly healed than the right, with satisfactory osteoid development present in both.

Discussion.

The case offered several academic and clinical points of interest. First, it showed that a ruminant may be kept alive for 44 days whilst being unable to ruminate, and pick up considerable condition in the process. It had been hoped that there would have been no attempt to regurgitate ingesta, since the feed was ground before being administered. As stated above, regurgitation did occur. Perhaps the feed was not ground sufficiently fine to preclude this. The use of artificial saliva was considered. It was felt, however, that saliva would be formed and swallowed without the stimulus of chewing.

The plaster casting was satisfactory. The author would be inclined to use a similar cast in this type of lesion, provided the fracture

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was recent (up to 3 days.) In cases of delay, however, metal plating and injection of blood (in the absence of possible infection) to provide a new fracture heamatoma, would be combined with the cast.

It is disappointing to have lost the case when success was so near, particularly as the cause of death could not be determined.

Summary.

A case of bilateral compound fracture of the horizontal rami in a range heifer and the treatment are described and discussed.

ACKNOWLEDGEMENT

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VERSLAG VAN UITBREKINGS VAN GEPROKLAMEERDE VEESIEKTES IN DIE UNIE VAN SUID-AFRIKA

1. Gedurende die Week eindigende 29 Julie 1960

Siekte	Plaas of Plek	Distrik	Geografiese ligging	Opmerkings
B.W.D.	Sarnia	Pinetown	3030D	
B.W.D.	Hillcrest	Pinetown	3030D	
Hondsdolheid	Bosley 477	Soutpansberg	2924B	
Hondsdolheid	Stella Dorpsgronde	Vryburg	2427D	Meerkat
Kwaadaardige Buffel	Mzinene 526	Hlabisa	3129B	
Gonderiose Milde Bees-	Khaku Location			
Gonderiose	No. 39	Sibasa	3023C	
Gonderiose	Marseille 82	Sibasa	3024A	
Gonderiose	Kleinplaas 415	Sibasa	2924B	
Slapsiekte	Scattergood	Kuruman	2328B	Perde
Tuberkulose	Amanzimtoti Eksperimenteleplaas (Bantoe Sake)	Umlozi	3031B	Bees
Tuberkulose	R. Milbank "Alvin"	Camperdown	3030D	Bees
Tuberkulose	Suid-Ooshoek	Wepener	2630D	Voëls

2. During the Week ending 5th August, 1960

Disease	Farm or Place	District	Geographical Location	Remarks
Benign Bovine Gonderiosis	Penington Butchery	Umzinto	3031B	
Gonderiosis	Nhlwati 525	Hlabisa	3129B	
Gonderiosis	Sibasa D/A	Sibasa	3023C	
Gonderiosis	Lambane D/A	Sibasa	3023D	
Mange	Ndakana Loc. 8	Nqamakwe	2733B	Goat
Mange	Busila	Willowvale	2833B	Goat
Mange	Dube Loc. 14A	Kingwilliamstown	2733C	Goat
Mange	Qibira Loc. 37	Middeldrift	2733C	Goat
Mange	Zihlahleni Loc.25	Middeldrift	2733C	Goat
Mange	Qumanco Loc.	St. Marks	2732D	Equine
Mange	Cofimvaba Loc.	St. Marks	2732D	Equine
Rabies	Kruis	Boshof	2529C	Bovine
Rabies	Mons	Vrede	2928D	Bovine Histological Positive
	No. of Outbreaks			
Lumpy Skin	1	Worcester	1934D	

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3. GEDURENDE DIE WEEK EINDIGENDE 12 AUGUSTUS 1960

Siekte	Plaas of Plek	Distrik	Geografiese ligging	Opmerkings
B.W.D.	Stilwater	Vryheid	3028D	
Brusellose	Hella Hella	Richmond	3030C	
Hondsdolheid	Rhodesdene Voorstad, Kimberley	Kimberley	2429D	Hond Hist. susp.
Skurfte	Mbozisa 7	Mqanduli	2832D	Bk.
Skurfte	Mvezo 15	Umtata	2832D	do.
Skurfte	Oxobo	Idutywa	2833A	do.
Skurfte	Sundwana	Idutywa	2833A	do.
Skurfte	Nqadu	Willowvale	2833B	do.
Skurfte	Three Crown No. 22	Glen Grey	2732C	do.
Skurfte	Oitsi Location	St. Marks	2732D	Perd
Skurfte	Numcuzo Loc.	St. Marks	2732D	Perd
Tuberkulose	Williams	Bothaville	2628B	Beeste
Tuberkulose	Tweespruit	Thaba Nchu	2730A	Beeste
Tuberkulose	Rosebank Entstof stasie	Wynberg	1835A	Beeste

4. DURING THE WEEK ENDING 19th AUGUST, 1960

Disease	Farm or Place	District	Geographical Location	Remarks
Anthrax	Mona 706	Nongoma	3128D	
Anthrax	Skoongesig	Kroonstad	2728C	Porcine
Anthrax	Welkom	Hoopstad	2529B	Bovine
B.W.D.	Bonnie Home	Bethlehem	2829A	
Foot and Mouth	Sheila 220	Letaba	3125A	
Mange	Kwenxura Location 36	Mqanduli	2832D	Goat
Mange	Baziye Loc. 6	Umtata	2832D	Equine
Mange	Gosforth P187	Peddie	2734A	Goat
Rabies	Waterval 4	Rustenburg	2726C	
Rabies	Fabrici Suburb	Kimberley	2429D	Civet Cat Hist. Pos.
Scab	Mafeking Kaffir Village	Mafeking	2526D	Ovine
Tuberculsis	Sunwin Park	Camperdown	3030D	Bovine
Tuberculosis	Greenmeadows (B.M. Brokensha)	Pinetown	3030D	Bovine
Tuberculosis	Oldenburg	Ladybrand	2730A	Bovine

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5. GEDURENDE DIE WEEK EINDIGENDE 26 AUGUSTUS 1960

Siekte	Plaas of Plek	Distrik	Geografiese ligging	Opmerkings
B.W.D.	Grasmere	Vereeniging	2727B	
B.W.D.	New Germany	Pinetown	3030D	
B.W.D.	Tangaat	Inanda	3130C	
B.W.D.	Glemaholm Pluimveeplaas	Pietermaritzburg	3030C	
Hondsdolheid	Doornkop	Maraisburg	2532D	Meerkat
Miltsiekte	Suidwestelike hoek van die Oostelike Caprivi strook		2319A & B	
Miltsiekte	Hillside	Kei Road	2733D	
Milde Bees-Gonderiose	Reinshaw	Umzinto	3031B	
Skurfte	Laer Tongwane	Butterworth	2833A	Bok
Skurfte	Caba Loc. 10	Tsomo	2733B	Bok
Skurfte	Mjingini	Kentani	2833C	Bok
Skurfte	Ntshingeni	St. Marks	2732D	Bok
Skurfte	Tolifyeni Loc. 13	Kingwilliamstown	2733C	Bok
Skurfte	Kokstad dorpsgrond	Mt. Currie	2931A	Perd
Skurfte	T.A. 58	Pietermaritzburg	3030C	Perd
Tuberkulose	Lodewykslust 54	Piet Retief	3028B	Bees
Tuberkulose	Koppieskraal 129	Potchefstroom	2727C	Bees
Tuberkulose	Nooitgedacht 56	Klerksdorp	2627D	Bees
Tuberkulose	Kikoesvlei	Darling	1834A	Bees
Tuberkulose	Rosebank	Wynberg	1835A	Bees
Tuberkulose	J. J. Swart se Melkery	Upington	2821C	Bees 2 Positief

6. GEDURENDE DIE WEEK EINDIGENDE 23 SEPTEMBER 1960

Siekte	Plaas of Plek	Distrik	Geografiese ligging	Opmerkings
B.W.D.	Swartland Pluimveeplaas (Moorresburg)	Malmesbury	1834B	
Hondsdolheid	Debeers	Kimberley	2429D	Hond susp. geval 3463
Miltsiekte	Tabora	Reitz	2828D	Bees
Miltsiekte	Melkboschfontein deel van Knoffelfontein	Taungs	2428C	Bees
Knopvelsiekte	Graspan	Christiana	2528C	Bees
Skurfte	Pretoria B	Weenen	3029C	Perd
Skurfte	Cicilia (White)	Weenen	3029C	Perd
Skurfte	Nqwati 28	Umtata	2832D	Perd
Skurfte	Pongola 516	Ubombo	3228C	Bok
Skurfte	Mangwazana 515	Ubombo	3228C	Bok
Skurfte	Nkungavini 509	Ingwavuma	3228A	Bok
Skurfte	Mucwasu 15	Elliotdale	2832D	Bok
Skurfte	Bufana 14	Elliotdale	2832D	Bok
Tuberkulose	Grootfontein	Philippolis	2531A	Bees
Tuberkulose	Bothas Hill	Camperdown	3030D	Bees

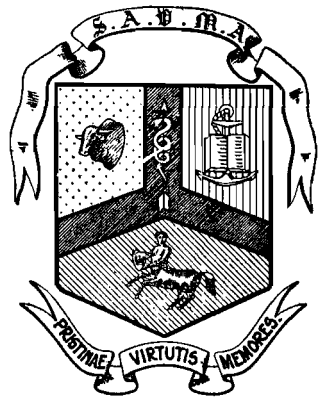
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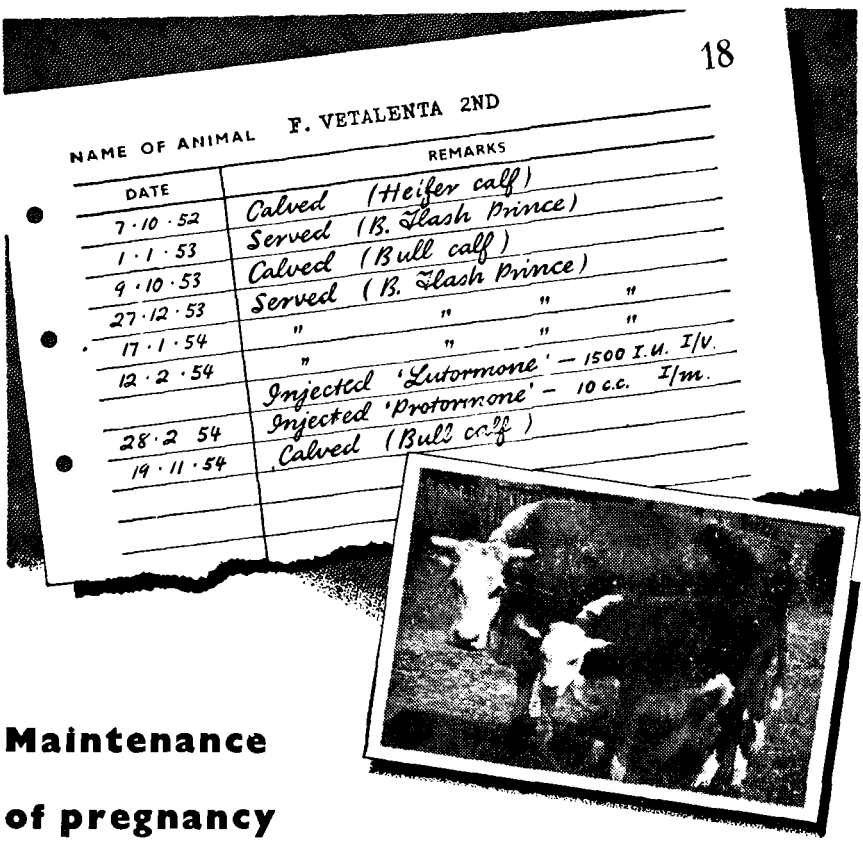
TYDSKRIF

VAN

DIE SUID-AFRIKAANSE
VETERINÊR-MEDIESE
VERENIGING

KONGRES-UITGAWE

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Maintenance of pregnancy

Mild luteal deficiency in the cow results in abortion or resorption of the fœtus. Until recently the common method of correcting this condition was to inject 'Lutormone' brand Luteinising Hormone at the time of service and then to give further doses until the cow was at least 12 weeks in calf.

Workers at The Wellcome Veterinary Research Station, Frant, have now demonstrated a more reliable method. This is the administration of 'Lutormone' at the time of service followed 14-16 days later by one injection of progesterone ('Protormone').

'Protormone' is also of value in marked luteal deficiency, for example persistent ovarian cysts. In cases where two doses of 'Lutormone' have failed to correct the condition a response may often be elicited with 'Protormone'.

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'PROTORMONE' BRAND

INJECTION OF PROGESTERONE (VETERINARY)

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MODERN NARCOSIS IN HORSES, CATTLE AND SWINE

O. Dietz*

Ten to fifteen years ago the treatment of the individual animal held first place in veterinary practice in Europe. Especially the horse was privileged when suffering from surgical conditions, because of its economic importance. Surgical diseases in cattle and swine were treated only when very valuable. Chloral hydrate was then the general anaesthetic for horses, cattle and swine, in the clinics of veterinary faculties as well as under conditions of practice. Significant changes have occurred since, in the whole field of veterinary medicine. The activity of the veterinary surgeon working in the country has shifted more and more to prophylaxis, however, without eliminating curative treatment completely. During the rapid progress of motorization, and intensification of cattle and swine breeding, the emphasis of veterinary practice has shifted towards these species. Concurrently with this purely economic revolution, veterinary surgery received repeated stimuli, particularly in the field of anaesthesia—the state we call unconsciousness and insensibility which is necessary for each surgical intervention for the patient's, as well as for the operator's sake. This progress in the field of general anaesthesia differs by no means from that in human medicine. The minor differences that do exist are due to the nature of the subject and the circumstances under which the veterinary patient must be anaesthetized. It is no problem to apply narcosis, if there are suitably trained assistants. In practice, modern anaesthesia means choosing methods which are comparatively harmless for the patient, which the practising veterinary surgeon can apply without any assistants, which inhibit reflexes, and which are suited to the operation, and will last for the necessary length of time. This progress in modern narcosis, we owe in the first place to the pharmacologists and pysiologists.

Now, what constitutes "modern" anaesthesia in veterinary medicine? There are three fundamentally different methods of narcosis or muscle relaxation, which can be used to supplement one another. They are: firstly, intubation in closed or half-open system, secondly, the potentiated narcosis and thirdly, application of muscle-relaxants as an adjuvant to narcosis. In applying these methods the veterinary surgeon is not confronted with one species, as in human medicine, but with a number of species, each reacting differently to the various narcotics, analgesics, sedatives and muscle-relaxants. The intermediary metabolism of our domestic animals is different, and so is their vegeta-

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tive reactivity and sensitivity of the central nervous system to these drugs. Similarly, there are differences in sensitivity to the muscle-relaxants.

Good progress has been made, especially with the barbiturates. The N(nitrogen)-methylated barbiturates are being displaced by the thio-barbiturates. And perhaps just now veterinary medicine stands at the beginning of a completely new method of narcosis—that of steroid narcosis (FRITSCH, KRAUSE, FREY)—where a state of narcosis resembling physiological sleep is attained by natural substances, the hormones. The choice of an anaesthetic depends upon requirements, and varies according to the species, state of the animal, the type of operative intervention, and whether applied in a clinic or in practice. Those methods of anaesthesia will now be considered which are applicable today in clinics, as well as in practice, and which have been developed latterly by adaptation of older methods.

I. ANALGESICS FOR HORSES

(1) *Sedation in the standing position*

Minor operative interventions require sedation in the standing horse. Morphine hydrochloride has not yet been surpassed by any other analgesic for this purpose. It can be applied intravenously in a dosage of 0.15–0.3 g. without supplementary treatment or at a dosage rate of 0.08–0.2 g. in combination with 8.0–12.0 g. chloral hydrate in 10 per cent solution. The analgesic effect of morphine in this combination is good. The observed side effects, such as slight motor unrest and vasodilation—especially venous—scarcely influence the intended operative intervention such as cautery and castration of animals in the standing position.

The effect lasts 30–45 minutes at most, which is sufficient to complete the surgical intervention without undue hurry. A similar state of analgesia for operating on the standing animal can be obtained by the combination of the analgesic Polamivet (Hoechst)—Methadone —similar to morphine in its effect, and chloral hydrate.

To Polamidon, a 2–dimethylamino-4,4–diphenylheptanon(5)–hydrochloride, a substance similar to atropine in its vagus-suppressing effect is added, and the combination is known as Polamidon C or Polamivet. Twenty–thirty millilitres Polamivet and 8–12 g. chloral hydrate are given intravenously in the standing animal. Over the last years substitution of Megaphen (Bayer) or Propaphenin (VEB Hydrierwerke Radleben) (i.e. chlorpromazine) for morphine or Polamivet in this combination with chloral hydrate has been tried repeatedly. The chloral hydrate was potentiated by this combination with the strongly sedative phenothiazine derivative, but the general analgesia was insufficient.

(2) *Chloral hydrate narcosis*

Chloral hydrate, applied by Liebig in 1832 for the first time, and introduced into therapeutics in 1869, is generally the best narcotic up to the present for use in clinics as well as in practice. Chloral

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hydrate causes strong hypnosis and sleep; it diminishes the sensibility and the reflexes and produces muscular relaxation. The reflexes, however, are not completely abolished. For intra-abdominal operations it has this disadvantage that the abdominal muscular pressure is scarcely suppressed. Although chloral hydrate anaesthesia cannot be regulated, its dangers for the patient lie more in slight underdosage than in overdosage. The intravenous infusion of a 10 per cent solution of chloral hydrate at the rate of 4.0 g. per 50 Kg. body weight is the best for general use. The rectal or peroral application is only used exceptionally nowadays.

The present importance of chloral hydrate lies in its use as a basal narcotic. Two methods have been developed. The first consisted of deepening the chloral hydrate narcosis, which cannot be regulated, by inhalation of ether (partly chloroform, too) in open system, which can be regulated. This combination led to induction of basal narcosis by means of chloral hydrate, followed by ether-oxygen inhalation narcosis, in half open or closed systems. Such complicated equipment is necessary for these methods of narcosis, that only clinics have them at their disposal.

On the other hand potentiated narcosis, with chloral hydrate as basal narcotic, combined with phenothiazine derivatives only, or with phenothiazine derivatives, analgesics, and chloral hydrate, have stood the test in clinics as well as in private practice.

(3) *Chloral hydrate and ether-oxygen inhalation narcosis*

About 100 years ago the anaesthetizing effect of ether was discovered, and somewhat later, the first complete anaesthesia was obtained by means of ether. Even for the horse, useful anaesthesia can be obtained by ether-steam inhalation with special equipment (BECKER and VÖLKER and others). This method of narcosis was introduced with chloral hydrate for casting animals without risk and for avoiding excitation. The ether-steam and the ether-oxygen-steam was conducted into the lungs directly by a naso-pharyngeal tube put into the trachea. The ether consumption amounted to 30–40 ml. per minute.

The development of endotracheal intubation narcosis in half open and closed systems in human medicine, suggested the use of similar equipment for narcosis in animals as well. The technical details and the method of using the apparatus are described in many publications. According to the construction of the apparatus the following methods of narcosis can be conducted: (1) circulation narcosis (closed system) with ether-oxygen; (2) narcosis in half-open system with ether-oxygen; (3) narcosis in half-open system with ether and fresh air. Finally, pure oxygen inhalation is possible. Artificial respiration, with the help of a pair of bellows, is possible in cases of cessation of the spontaneous respiration, in the half-open as well as in the closed systems. The equipment consequently allows the application of muscle-relaxants, especially of preparations of the Succynilcholine (Dekamethonium) group. In Europe the preparations Lysthenon (Stickstoffwerke, Linz), Succicuran (VEB Deutsche Hydrierwerke

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Radleben) and Celocurin (Vitrum, Stockholm) proved to be the best. In the Berlin Clinic we used Succicuran. Spontaneous respiration is not influenced by a dose of 10 mg. per 50 Kg. body weight. The effects last only 5–7 minutes and complete relaxation of the skeletal musculature can be obtained. However, spontaneous respiration ceases at a dosage level of 15–20 mg. of Succicuran per 50 Kg. body weight, because of subsequent paralysis of the diaphragmatic musculature, which lasts for 10–15 minutes. This method allows intrathoracic intervention in the horse, although an absolute indication for it does not exist in this animal, except in cases of foreign body obstruction in the thoracic part of the oesophagus, diaphragmatic rupture, or for the purpose of experimental investigation. The consumption of ether in the closed system with ether-oxygen is considerably reduced when compared to the old ether-steam inhalation narcosis. It amounts to approximately 50 to 55 ml. within 10 minutes, i.e. no more than 200 ml. ether are consumed for an operation lasting 30 to 45 minutes on the average. Anaesthesia performed in this way in the horse is completely satisfactory. The operation can proceed painlessly and is well-tolerated. Neither circulation nor respiration is unduly affected.

Instead of using chloral hydrate as basal narcosis Megaphen or Propaphenin (chlorpromazine) and Polamivet may be administered as premedication. Fifteen to twenty minutes before casting the animal, 10 to 15 ml. Megaphen (0.05 per cent) and 15 to 20 ml. Polamivet are administered intravenously. This premedication causes a reduction in ether consumption of about 40 ml. per 10 minutes. The casting and the intubation may be facilitated considerably by the additional administration of a muscle-relaxant (for example Succicuran, 10 mg. per 50 Kg. body weight). Further improvements are to be expected soon, in inhalation anaesthesia with ether-oxygen in the horse, although its use is mainly restricted to clinics.

In Austria, in Switzerland, in the Northern and Anglo-American countries inhalation narcosis with nitrous oxide, Cyclopropane, trichlorethylene (Trilene) and Fluothane is in use already. In Germany experience has been restricted to nitrous oxide.

(4) *The chlorpromazines and potentiated narcosis in the horse*

The introduction of the chlorpromazines effected considerable progress in veterinary surgery as well. The chlorpromazines have a highly sedative effect, and inhibit both sympathetic and parasympathetic systems; in addition they depress the metabolism thus lowering body temperature, and have anti-emetic qualities. Chlorpromazine itself has no narcotizing effect, but it raises the effects of different narcotics and analgesics. In Germany several preparations of this group are known at present, for example Propaphenin (VEB Deutsche Hydrierwerke Radleben), Megaphen (Bayer), Decentan (Merck), and Combelen (Bayer). During recent years three main indications have crystallized. Firstly, these preparations are used alone as so-called „tranquillizers", secondly for potentiating narcosis, and finally they are used purely therapeutically in psychoses. The chlorpromazines and derivatives do not seem to be suitable as sedatives for the horse when

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used alone. In the horse certain centres of inhibition may be released, causing excitation to a greater or lesser degree. In this species, therefore, the most important indication is in the field of potentiated anaesthesia. The first notes on potentiated narcosis originate from WESTHUES. He administered 10 ml. of the 0.5 per cent. solution of Megaphen, 10 to 15 to 20 ml. Polamivet and chloral hydrate intravenously to horses of average weight. The elimination of pain was much better than with chloral hydrate alone. OWEN and NEAL had similar results. Experience over the last 5 years showed that the dosage given by WESTHUES did not meet the requirements of every case.

In potentiated narcosis the risk is diminished considerably, and the narcosis itself is well tolerated. At the same time reflexes are abolished to a marked degree. This method thus commends itself, especially to the surgeon working without assistance. The dosage level—with Polamivet premedication—as advocated by GRUNER, closely meets all requirements. Before applying hobbles 0.3–0.5 mg. per Kg. body weight of Propaphenin (that is 5 to 10 ml. of the 2.5 per cent solution for immediate use per 500 Kg. body weight) and 15 to 20 ml. Polamivet per 500 Kg. body weight are injected intravenously. Then chloral hydrate in 10 per cent. solution is administered until the animal collapses. The usual dose of 4.0 g. chloral hydrate in 10 per cent solution per 50 Kg. body weight may be administered without any danger, if a long lasting narcosis with a minimum of reflexes is desired. The duration of this potentiated anaesthesia is about 80 to 120 minutes, and the elimination of pain is complete within the first 40 to 60 minutes. The muscular relaxation is particularly good within the first 30 minutes After this narcosis the animal sleeps for 1 to 3 hours, as a rule a disadvantage, especially under conditions of private practice. Complete absence of reflexes does not occur, especially when neurectomies are performed. Compared to the sole use of chloral hydrate this potentiated narcosis (Propaphenin-Polamivet-chloral hydrate) distinguishes itself by better elimination of pain, by longer duration of anaesthesia, and by better muscular relaxation, according to GRUNER. During narcosis and postnarcotic sleep the body temperature falls 1.5 to 5°C. The head must be rested lower than the body and the patient must be covered during the period of postnarcotic sleep. According to our experience the potentiated narcosis is one of the safest and most humane methods in the horse. Even patients with severely taxed circulation—for example in laparotomies or in cases of ileus—rarely develop alarming complications.

(5) *Barbiturate anaesthesia in the horse*

Recent experimental use of barbiturates only did not bring about the desired results. The pre- and postnarcotic excitability could not be eliminated; besides, these preparations were too brief in their action. In this connection the experiments with Rapidorm performed by SELINICK, TEUSCHER, and WEHNER and those with Narkovet made by WESTHUES are to be mentioned. Though WESTHUES combines Narkovet (consisting of five parts of Evipan sodium and one part

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of Luminal sodium) with Polamivet and Novalgin, excitability was not avoided completely. Anaesthesia for a short period of 5–10 minutes only could be obtained. The additional use of chloroform was necessary for operations lasting a longer time.

The experiments conducted hitherto to combine barbiturates and chloral hydrate as tried by SILBERSIEPE-BERGE and BACKHAUS (chloral hydrate and Eunarcon) and by ROTH (chloral hydrate and Cito-Eunarcon) have not yielded absolutely satisfactory results. Expecially Cito-Eunarcon must be injected repeatedly at about 11 minutes' interval; up to 300 ml. are consumed for an operation lasting two hours. Recently BLENDINGER and TEUSCHER and others have tried the long-lasting nitrogen-methyl-barbiturate (Narcoren) (Bacteriologic Institute, Dr. Rentschler & Co., Warthausen) in the horse with good results. TEUSCHER gives 3.0 g. chloral hydrate per 50 Kg. body weight and after having waited for 3 minutes he injects 2.5 to 4.0 ml. Narcoren per 50 Kg. Thus a two to two and a half hours' anaesthesia can be obtained with a 30 to 50 minutes' stage of tolerance. The narcosis is deep and reflexless, it is simple and cheap and therefore is said to be very suitable for practitioners.

(6) *Muscle-relaxants in the horse*

As mentioned above the preparations of the Succinyl-choline and Dekamethonium group have been of value in the horse. They have a relaxing or completely paralysing effect on the voluntary muscles (HANSON, TRATNIG-FRANKL, AMMANN, ÜBERREITER and others); they are used for the so-called medicinal casting, as an adjuvant to narcosis, and also therapeutically (for example tetanus). Their use as an adjuvant to narcosis is permissible only if an inhalation apparatus for narcosis is available as a stand-by for artificial respiration as well in case of need. The antidote for the preparations of the succinylcholine group is pseudocholinesterase. These preparations are not well suited as adjuvants to narcosis. Their effect is very short, as their degradation begins immediately after intravenous intoduction.

A preparation of the myanesin group, namely guaiacol—glycerine-ether (GGE) presently studied in the Berlin Clinic (VEB Chemical Works, Grünau) has shown better results. The preparations of the myanesin group act centrally on the brain and spinal cord and/or peripherally. The relaxation of voluntary musculature ensues on paresis of the spinal and bulbar centres. The respiratory musculature including that of the diaphragm is not influenced. Therefore the use of an inhalation apparatus is not necessary. If combined with chloral hydrate the effect of guaiacol-glycerine-ether (GGE) (120 to 150 ml. in 20 per cent. solution) lasts 15 to 20 minutes in the horse. During this time complete suppression of reflexes is obtained. The usefulness of this preparation for the horse is now being subjected to further study.

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II. The Different Methods of Narcosis in Cattle

Upon surveying the recent literature on veterinary medicine, one is surprised that there are only a few publications on general anaesthesia in cattle, in spite of their growing economic importance. Consequently general anaesthesia in cattle must not be considered a solved problem—quite the contrary. Two sets of circumstances prevent the veterinary surgeon, working without assistance, from using general anaesthesia in cattle. Firstly the complicated anatomical and physiological arrangement of the fore-stomachs may cause insufficient eructation leading to tympany and consequent respiratory distress during reflexless narcosis of long duration. This opinion, recently advocated by Stöber, is contrary to experiences in practice. In more than 1,000 general anaesthesias in cattle conducted in the Berlin and Leipzig clinics within the last 10 years we have not observed a single case of tympany of the rumen. It has been our experience that flowing out of the contents of the rumen can be prevented almost completely by twelve hours' starving and thirsting of the animals. Hitherto not a single case of aspiration-pneumonia by regurgitation of ruminal contents has occurred but could be prevented by adequate lowering of the head.

The other circumstance restricting the use of general anaesthesia in cattle is the tolerance to surgical intervention peculiar to this species. Abdominal surgery, especially, can be executed under infiltration, conduction or sacral anaesthesia. The use of tranquillizers nearly always allows the use of local anaesthesia for the most important surgical procedures even in animals that are particularly intractable and vicious. However, there are operations in cattle where general anaesthesia is preferred, for example, extirpation of actinomycomata, operations for herniae, extirpation of larger haematomata, amputations of claws with advanced phlegmosis, etc. The older general anaesthetics in cattle are not satisfactory for these purposes.

(1) *Alcohol*

The oral administration of 1–3 litres of 30–50% ethyl alcohol only dulls the senses. One must reckon with possible accidents in anaesthesia, as the therapeutic index of alcohol is very low for cattle.

(2) *Ether*

Ether and ether-oxygen inhalation has not been successful in cattle. Special equipment is necessary. In case of emergency slaughter during or immediately after operation the smell of ether clinging to the meat is disadvantageous and may lead to condemnation of the carcass.

(3) *Chloroform*

Chloroform anaesthesia for cattle is still recommended by Westhues and Bauer (Stöber). Only low concentrations of chloroform are necessary for general anaesthesia. The danger of tympany is insignificant. However, the duration of narcosis may not exceed 20 minutes, so that extensive operative intervention cannot be undertaken.

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(4) *Chloral hydrate*

Chloral hydrate is still the most common narcotic for cattle. It is applied intravenously in 10 per cent. solution at a dosage rate of 4.0 to 5.0 g. per 50 Kg. body weight as for the horse. In cattle chloral hydrate narcosis leads to crepuscular sleep: complete suppression of reflexes cannot be obtained.

(5) *Chlorpromazine and potentiated narcosis*

The use of chlorpromazines and their derivatives have led to improved methods of narcosis in cattle. The sole administration of these preparations, for example Megaphen, Propaphenin and Decentan, causes a sort of sleep; vicious animals can be calmed and most of the operations can be performed under local anaesthesia.

Tranquillizers are employed for purely therapeutical purposes, for example to remove obstruction from the pharynx. The penis of the tranquillized bull can be masaged out of the prepuce without difficulty for diagnostic and therapeutic purposes.

The combination of chlorpromazines—especially of Megaphen (10 to 20 ml. of a 0.5 per cent. solution) and of Propaphenin (5–10 ml. 2.5 per cent solution) with chloral hydrate (4.0 g. per 50 Kg. body weight)—gives a potentiated narcosis in cattle with almost complete suppression of reflexes and a state of operative tolerance for 60 minutes. Post-operative sleep lasts 1 to 3 hours. The animal must be laid on its right side in order to prevent tympany of the rumen with all its consequences.

(6) *Barbiturates*

Both the short- and long-acting barbiturates used in Germany up to now (STÖBER), namely the N(nitrogen)-methylated barbiturates—have proved to be inefficient for cattle. Investigations on the use of the fairly recently introduced thiobarbiturates have been more successful. Thiogenal (Merck), at 18 mg. per Kg. body weight, is suitable as a short-term narcotic for removing the horns of calves (DIETZ, KIRSCH, KIESSLING). KLAUS and RUNTER first reported on Thiogenal narcosis in adult cattle. STÖBER and WEAVER narcotized 60 cattle with Thiogenal (12 to 16 mg. per Kg. body weight). In 42 cases Decentan (Merck) was given as premedication. The narcosis obtained is safe and lasts about 10 minutes. It makes short surgical operations possible.

According to our own experiments a better and longer lasting effect can be obtained by the combination of chloral hydrate and Thiogenal: 20.0 g. chloral hydrate in 10 per cent. solution and 15 mg. Thiogenal per Kg. body weight intravenously give a narcosis nearly without reflex, lasting 30 to 45 minutes. This narcosis is especially good for operations on the head, for example, extirpation of larger actinomycomata.

(7) *Muscle-relaxants in cattle*

Muscle-relaxants of the Dekamethonium and Succinyl choline groups are contra-indicated in cattle. According to our own investigations only one tenth of the dose usual for horses is tolerated by cattle

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without lengthy paralysis of respiration. On the other hand, preparations of the myanesin group, especially guaiacol-glycerine-ether are suitable for cattle, for casting as well as an adjuvant to narcosis (DIETZ, KRAUSE, SATTLER, WESTHUES). Twelve to sixteen ml. per 50 Kg of a 20 per cent solution injected or infused intravenously, cause a complete relaxation of the skeletal musculature so that lateral decubitus is obtained. Chloral hydrate (40 g. per 50 Kg.) and 20 per cent guaiacol-glycerine-ether, at the rate of 15 ml. per 50 Kg. body weight intravenously, give a narcosis without reflexes lasting 30 to 45 minutes. There is no danger of over-dosage by GGE.

III. NARCOSIS IN SWINE

Until the last decades, as already mentioned in the introduction, operative interventions in swine were restricted, with a few exceptions, to castration of male animals. As result of the increased economic importance of swine breeding, swine surgery won attention. Veterinary research had to look for methods of narcosis suitable for practical, large scale application to swine.

At first narcosis with chloral hydrate, chloroform and ether were tested. Later on, about 25 years ago, barbituric acid derivatives were introduced for narcotizing swine. The perfection and improvement of these preparations is still continuing. For research purposes swine were recently narcotized by laughing gas, propane and steroids.

A. VARIABLE DOSAGE NARCOTICS

Chloroform, ether, and recently laughing gas and cyclopropane, belong to the group of narcotics of which the dosage can be accurately controlled. These narcotics have the advantage that their administration can be broken off at any moment; thus an immediate reduction of concentration of the narcotic in the organism can be effected. In practice the application of these narcotics is not without danger and technically too troublesome.

(1) Chloroform, chemically trichlormethane, was the main anaesthetic up to 25 years ago. In swine surgery it is no longer used as it seriously injures the heart, and its narcotic margin is very narrow.

(2) On the other hand ether is a much safer narcotic for swine. The effect on heart and circulation is less than that of chloroform. Ether produces a deep narcosis and the dosage may be regulated according to course and duration of operation. Easily handled masks and equipment can be employed. An economic disadvantage in case of sudden need for emergency slaughter is the tainting of the flesh by the smell and flavour of ether. The use of ether is recommended, therefore, for swine weighing up to 25 Kg. as the objection mentioned virtually plays no part in such cases. The onset of narcosis follows 3 to 5 minutes after administration and lasts as long as ether is supplied. The animal awakens some minutes after stopping the administration of ether.

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(3) Laughing gas and cyclopropane, recently introduced into veterinary surgery, require special equipment, at present only used in clinics. The gases are mixed with oxygen and fed under pressure, with absorption of carbonic acid, in a closed system with provision for artificial respiration. Chlorpromazine (Megaphen or Largactil—10 ml. of a 5 per cent solution, or Propaphen—5 ml. of a 2.5 per cent solution) may be used intramuscularly as general sedative.

For the induction of narcosis, Nembutal or Scopolamine-Eukodal-Ephedrin (SEE) are given, followed by inhalation. In order to reduce the salivation and the mucous flow, 0.2 mg. atropine sulphate per Kg. body weight is injected subcutaneously.

The advantages of laughing gas narcosis are the quick induction of anaesthesia and the insignificant influence on heart and circulation. In the slaughtered animal there is no abnormal smell or taste (HILL and PERRY).

B. PRE-DETERMINED DOSAGE NARCOTICS

Usually the non-controllable narcotica are administered parenterally. One of the ear veins, and recently the vena cava cranialis, are used for the intravenous injection. Likewise the peritoneum is suited for parenteral administration of narcotics, as absorption is very satisfactory. For castrations one may use the narcotic intratesticularly, too. It is absorbed quickly by the lymphatic vessels and thus the desired concentration in the blood is obtained.

(1) *Chloral hydrate* still plays an important part as an anaesthetic for swine. Pharmacologically it is a hypnotic, possesses a sufficient therapeutic margin of safety, does not cause pre- or post-narcotic excitement and is cheap, hence its continuing popularity. The dose is 5.0 g. per 50 Kg. body weight for intravenous injection. In case of intraperitoneal administration of chloral hydrate a concentration of 4 per cent should not be exceeded because of severe damage to the tissues with danger of adhesions forming. The dose is 0.25 g. per Kg. body weight.

(2) *Some of the numerous derivatives of barbituric acid* have been introduced successfully into veterinary surgery; they are very well suited to surgery of swine. The effect of the drug introduced into the blood stream is prompt, thus facilitating the preparations for operation.

The animals lie in a deep narcosis; only few assistants are necessary for their fixation and the operation can proceed without any disturbance. The adequacy of the dosage of barbiturates cannot be controlled accurately when injected. This is a disadvantage, which endangers life in case of over-dosage and may delay matters for the operating surgeon. The effect of barbiturates depends on the constitution and reactivity of the patient. These factors often are not sufficiently obvious and are, therefore, rarely given due consideration. Moreover, the barbiturates must be injected in the prescribed concentration and only slowly. If injected quickly and if the concentration of the solution is too high, the barbiturate reaches undesirably high levels in

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the blood-stream and the drug concentrates too quickly in the central nervous system. As unconsciousness sets in, the vagotonic system becomes hyperexcitable and sudden disturbances of respiration and circulation may ensue. During the last 25 years numerous barbiturates were developed, some of which will be mentioned here:

(a) *Evipan (Sodium).*—This preparation is obtainable in the trade only as a dry substance, as the solution is not stable. Evipan causes extreme excitement and often its effect is too short. In swine surgery it was, therefore, displaced by other barbiturates.

(*b*) *Eunarcon* was the commonly used narcotic in swine surgery for a long time in Germany. With this barbiturate an exact determination of weight and a slow and equable injection are necessary for good results. In many instances these requirements can be met only with difficulty. A great disadvantage of Eunarcon is the early falling down of animals with all its disagreeable effects, for example, the canula slipping out of the ear vein, excitement and so on. A degree of narcosis suitable for operation is obtained for 20 to 30 minutes. The dose is given intravenously at the rate of 0.15 to 0.25 ml. in 10 per cent solution per Kg. body weight.

(*c*) *Narcovet*—consisting of five parts of Evipan (Sodium) and one part of Luminal Sodium—is a narcotic that is not dangerous even in slight overdosage. The presence of Luminal produces a two hours' sleep after the operation. The narcosis is deep and the narcotic must not be repeated. Narcovet is used extensively and with safety in all operations on swine, such as castration, cryptorchidectomy, caesarean section and operative treatment for prolapsus uteri and actinomycosis. The dose is 1.2 g. per 50 Kg. body weight.

(*d*) *Narcoren* belongs to the recent barbituric acid derivatives. Experiments and application in practice have shown that the necessary 45 minutes' narcosis is obtained by an intravenous dose of 0.15 ml. per Kg. up to 20 Kg. body weight, by a dose of 0.10 ml. per Kg. up to 50 Kg. body weight, and by a dose of 0.075 ml. per Kg. for more that 50 Kg. body weight. The animals sleep up to 5 hours after operation—too long for practitioners when they want to wait for awakening.

Evipan (Sodium), Eunarcon, Narcovet and Narcoren belong to the N-(nitrogen)-methylated barbiturates which did good service some years ago. However, they fell into disuse when the chemical industry produced the O-(oxygen)-methylated thiobarbiturates. The thiobarbiturates have especially a short stage of induction, a short time of recovery and a wide safety margin. They produce narcosis free or nearly free from excitation and are quickly removed from the blood circulation because of their special affinity to body fat. Thiogenal (Merck) and Baytinal (Bayer) belong amongst others to the thiobarbiturates used in Germany.

(*e*) *Thiogenal* (Merck) is chemically Na-(sodium)-methyl-thioethyl-phenyl-thiobarbituric acid. The solution must be prepared before use. Thiogenal has only a minor effect on respiration and circulation and does not bring about pre- or post-narcotic excitement if administered correctly. The dose is 1.2 to 1.5 g. per 50 Kg. body

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weight in a 10 per cent solution for swine up to 100 Kg.; for more than 100 Kg. body weight the dose is 1.0 g. per 50 Kg. in a 20 per cent solution. Thiogenal administration may be repeated without fear of untoward effects after abating of the narcosis. It is especially recommended for caeserean section in swine because of its low tocicity.

(*f*) *Baytinal* (Bayer), chemically a sodium salt of the 5, 5-allyl-(2-methylpropyl–) thiobarbituric acid. The preparation is sold as a powder and it must be made up into a 10 or 20 per cent solution prior to use: 0.025 g. per Kg. is administered to swine up to 30 Kg., 0.03 g. per Kg. for swine between 30 and 100 Kg., 0.02 g. per Kg. for swine of more than 100 Kg.

Compared to Thiogenal, Baytinal causes the animals to lie down very slowly and quietly and excitement is rare. Anaesthesia lasts about 30 minutes and may be extended by slight overdosage. The narcosis is without reflexes. The animals have a postnarcotic sleep of only 10 to 15 minutes, but must be rested after awakening, as they stagger and fall down.

(3) *Chlorpromazines and their derivatives*

The phenothiazines have a wide range of activity. Amongst other effects they depress the cerebrum and evoke a general sedation without decreasing apperception and reflex activities. This feature makes it suitable for the treatment of boars and sows which are often intractable and vicious.

One of the phenothiazine derivatives most frequently used is chlorpromazine known under the name of Megaphen (Bayer) (or Propaphenin).

Chlorpromazine is suitable for intravenous and intramuscular administration. The intramuscular injection is preferred as a rule, because it is easier. The intravenous dose begins to take effect after 5 to 10 minutes, the intramuscular dose after 20 to 30 minutes. The animals go quietly out of the stable after having received the dose, remain quiet under painful treatment and allow exact and clean and safe operating. The experiences with the phenothiazine derivative perphenozin known under the name of Decentan (Merck) are similar. Particularly good results are said to be obtained with a dose of 0.2–0.3 mg. per Kg. in sows eating their piglets.

(4) *Potentiated Narcosis*

Potentiated narcosis is used in swine with very good results. Schebitz and Dietz recomend premedication by Megaphen or Propaphenin (Largactil) with a Narcoren-narcosis. Thiogenal and Baytinal anaesthesias are suitably potentiated by Megaphen. Potentiated narcosis is recommended for caeserean section. Sedation and relaxation are obtained by premedication with Megaphen; the required degree of anaesthesia is reached more quickly and lasts longer after application of the narcotic, thus allowing the operation to be performed slowly—thus giving the inexperienced operator some leeway without overtaxing the patient.

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The phenothiazines evoke hypothermia because they depress the metabolism. This drawback often leads to the death of piglets when caeserean section is performed. Especially for swine with their rather large body surface, one must take care that the animals are laid on a warm thick litter and covered, if possible. The postnarcotic period lasts longer in a potentiated narcosis; this fact is regarded favourably by numerous authors, because of the postoperative immobilization. Megaphen or Propaphenin can be used intramuscularly about 20 to 25 minutes before the narcosis or intravenously directly before the injection of the narcotic. Intravenously the patient receives 5.0 to 10.0 ml. of 5 per cent solution of Megaphen, thereupon the complete dose of Narcoran, Thiogenal or Baytinal is injected.

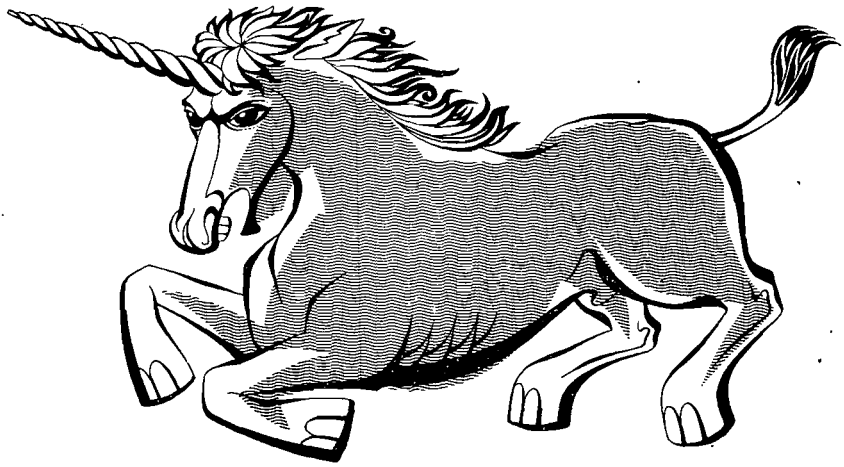
Summary

During the past 10 years the methods of general anaesthesia for horses, as well as for cattle, and swine have been improved fundamentally. The introduction of new inhalation methods, potentiation of anaesthetics and the application of muscle-relaxants essentially contributed to this fact. The risk of narcosis has been diminished considerably by these methods. In the clinic as well as in private practice the surgeon is able so select a method according to the circumstances of the case, a method that does not tax the patient unduly and yet fulfils desired requirements. Of course, all problems of narcosis have not yet been solved. The production of analgesia is a general medical requisite which should receive due attention in the case of our domestic animals, now and in the future.

References

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"THE MONOCEROS

is a monster with a horrible howl, with a horse-like body, with feet like an elephant, and with a tail like a stag's. A horn sticks out from the middle of its forehead with astonishing splendour to a distance of four feet, so sharp that whatever it charges is easily perforated by it. Not a single one has ever come alive into the hands of man, and, although it is possible to kill them, it is no possible to capture them'

T. H. WHITE "THE BOOK OF BEASTS"

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THE 1960 EPIZOOTIC OF AFRICAN HORSESICKNESS IN THE MIDDLE EAST AND S.W. ASIA

P. G. Howell*

By way of introduction to this short report on the epizootic of African Horsesickness in the Near East and S.W. Asia, I wish to express my appreciation of the great privilege of representing this institute, and providing the channel whereby the results and experience of many years of research work, could be made known to the veterinarians in the field, and in the laboratories of this region. My assignment with the Food and Agricultural Organization of the United Nations was a most enlightening experience, and in my brief association with this international organization, I was deeply impressed by the many and varied programmes which are being sponsored in the underdeveloped areas of the world. As much as I would like to describe in detail my impressions and observations, my associations with people of vastly different creeds and social habits, and the many acts of kindness and hospitality so frequently extended to me throughout this tour, I am afraid that in a formal meeting of this nature I must confine my remarks to the more technical aspects of this epizootic.

Before proceeding to describe the origin and development of the disease, it may be well to refresh your memories with some of the more important geographical features of the affected region and the salient climatological conditions, in so far as they have influenced and will influence the course of the disease.

In the extreme east, India lies astride the Tropic of Cancer, with a great northern mountainous wall separating it from the rest of Asia. A central lowland plain is formed by the basin of two great rivers, and is flanked to the south by the plateau of peninsular India. Between this plateau and the west coast, there is a narrow coastal plain, where up to 80 inches of rain may be recorded during the rainy season of June to October. West Pakistan is essentially an extension of the more severe climatic and physical features of India. The great Indus valley regularly experiences temperatures of over 90°F during July, while in the delta area with its extensive swamps, a high humidity prevails. To the west, Pakistan is separated from Afghanistan by the Sulaiman range of mountains, where its numerous valleys and temperate summer climate encourage large groups of nomads to seek grazing for their flocks. On the banks of the small streams, villages are settled and a limited amount of cultivation practised. Afghanistan forms the most easterly extension of the Iranian plateau, bordered in the north by high mountains, and to the south by dry sandy deserts. Rainfall is sparse and great extremes of temperature are recorded.

The greater part of Iran is formed by a dry plateau with an elevation of three to five thousand feet, crossed by numerous hilly ranges. On

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this plateau the winters are extremely cold, while in the summer the heat is dry and scorching. The vegetation is sparse with the exception of the southern coastal belt, where a more temperate climate improves its density. The greater part of Iraq is flat open country, which continues unchanged into Syria. Cultivation is confined to the irrigable banks of the wide Tigris and Euphrates rivers. In the north the affected eastern regions of Turkey present a complex pattern of low mountainous ranges with a typical Mediterranean climate.

From the description it will be observed that the affected region is characterised by great extremes—dry arid deserts to hot humid coastal belts—severe winters in the higher altitudes, but more mild and accommodating temperatures near the sea. It is only natural therefore, that these great variations in climate will have a marked influence on the struggle for existence of both man and his animals in these areas.

In order to appreciate the reasons why such a very large area is now involved, and the great distances over which the virus has been transported, it is essential to say a few words about the social habits and agricultural practices of the populace; particularly in Iran. While the greatest fears have been expressed for the safety of horses used by the military and police, certainly the greatest losses have been experienced by the nomadic tribes and farming communities.

The greatest nomadic activity originates in Iran, where it is estimated that more than two million people are involved. These nomads may undertake short migratory movements a few miles outside the perimeter of their villages, or they may move over a period of two to three months, in a northerly and north-easterly direction, covering six to seven hundred miles. The prime objective is the escape from severe climatic conditions and the search for fresh grazing for their herds of goats and sheep. Between Afghanistan and Pakistan large groups of nomadic workers cross the borders for short periods of employment. Without exception, all these people are entirely dependent upon donkeys, horses and camels for the transportation of their personal possessions.

The cultivation of cotton, wheat, vegetables and a little rice, is the essential feature of agriculture amongst the villages, where groups of about 100 inhabitants are accommodated in houses built invariably alongside rivers or wells. The animals of a village, and in particular the horses and donkeys, are used for the transportation of produce, the drawing of water and a limited amount of ploughing. These equines are confined to the villages, although young animals may be purchased or sold far afield. Once again we are confronted by a contrast between small static equine populations coming into contact with the droves of pack animals of the migrating nomads, under conditions which will favour transmission of the disease by an active localised and dense insect population. In passing it should be mentioned that the epidemiology of malaria in these areas is also influenced by similar environmental factors, and many similar features can be found in the present epizootic of horsesickness.

Numerous difficulties make it almost impossible to accurately trace the origin and date of the first introduction of the disease into

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S.W. Asia. It is almost certain, however, that this epizootic had its origin with the introduction of an infected animal by sea through one of the southern ports of Iran. It is acknowledged that a considerable traffic by small boats takes place between the shore of the Persian Gulf, the adjacent islands, Saudi Arabia, and even as far afield as the West Coast of Africa. The first outbreaks to attract attention in the summer of 1959 took place in the neighbourhood of Baft, some two hundred miles fom the coast. This is a summer grazing area of the Rudbari tribe and it is therefore conceivable that the disease was brought inland from the coast during the annual migration in April of the same year. By midsummer numerous foci had developed along the coast and many outbreaks followed in the wake of the migrating nomads. Morbidity reached 80 per cent and a conservative estimate puts the losses at 10,000 animals. There is little doubt that the disease entered Afghanistan by an extension of these nomadic movements, where extremely high mortality was reported from the southern districts.

In September 1959 a highly fatal disease of equines was reported from Quetta and Fort Sandeman, on the Afghani-Pakistan border. A second wave of infection during November, spread rapidly eastwards and northwards to the banks of the Indus River. Only at this stage was the diagnosis eventually confirmed, by the examination of specimens forwarded to Onderstepoort. The implementation of control measures limited mortality to 4,000 animals, and the disease was not seen during the remainder of summer. With the return of warmer weather, however, horses in 10 villages in the Tharpaker desert became infected and again later in July further cases were reported in the extreme north in Azad Kashmir.

Despite every effort on the part of the Indian Government to prevent the introduction of horsesickness, the disease was diagnosed on 22nd April amongst horses and mules in two isolated towns, Jaipur and Udaipur, situated some 300 miles from the Pakistan border. The entry of the disease into India was traced to the encampment of a gypsy caravan, in the immediate neighbourhood of a military camp. Deaths continued for two weeks, when all trace of the infection disappeared. It came as a great disappointment therefore, when in spite of the control measures sucessfully employed during the first outbreak, that a month later 1,000 horses should become infected, 400 miles to the south, near Bombay.

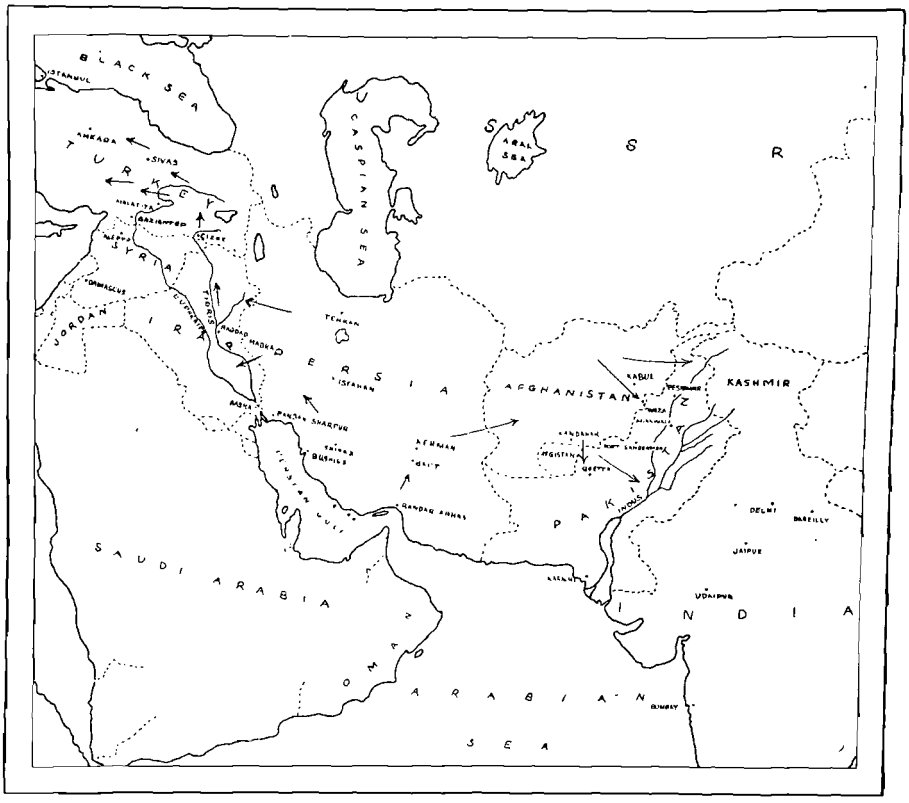
The north-westerly movement of nomads resulted in the spread of the disease to Iraq, where on the 28th April a number of police horses patrolling the border in the neighbourhood of Badra, died of the disease. Within a few days further outbreaks were reported along the border, from Abul-Khaseck in the south, to Peabaz in the north. Under the prevailing conditions, the dense insect population along the river banks assisted in an alarming extension of the disease in a northerly and easterly direction. At times more than 200 deaths were recorded in a day and it was not long before the pollution of rivers by the decaying carcases, gave rise to a serious public health problem. By the end of May 14,000 horses had died, and it was estimated that thirty per cent of the nomads' horses had been lost, en route to the summer grazing

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areas. At the earliest possible opportunity an extensive campaign of mass immunization was introduced, using a polyvalent mouse attenuated vaccine. It soon appeared that this policy would pay great dividends, for during the last two weeks of July only 10 cases were reported throughout the country.

The northerly progress of the disease along the banks of the Euphrates and Tigris rivers, soon reached the border of the United Arab Republic (Syrian Region), but only appeared in this country as sproadic outbreaks, possibly as a result of the control measures adopted. Unimpeded, the epizootic extended further northwards to the watershed of these two great rivers in eastern Turkey, crossing the border at the small town of Cizre on the 23rd of May. Within two months 10,000 horses died and 4,000 were destroyed, in the twenty-four infected eastern provinces of this country.

In endemic areas of Africa, the clinical symptoms shown by affected horses have been described in two distinct forms. In recent years natural cases of the pulmonary form are only very rarely seen. Throughout the Middle East the disease has been recognised by the marked supra-orbital oedema, extending to the ventral aspect of the neck and the subcutaneous tissue of the sternum, which is typical of



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the cardiac form. In convalescing animals an unusual feature has been the very severe involvement of the eyelids, where the enlargement has given rise to erosion and rupture of the mucous membrane, followed by secondary infection and blindness. A small percentage of mules have succumbed, the majority showing only mild symptoms, while the mortality amongst donkeys has been negligible.

At present the disease remains active in the extreme East and West, where the sudden reappearance in Alleppo and Bombay gave reason for concern. Potentially, the most serious threat exists in Turkey, where further uncontrolled spread to the North West will endanger the Mediterranean countries, and the mainland of Europe.

The control of this disease is based upon established and well founded principles, although emphasis on only certain aspects in each country has met with varied success. In Iran mass vaccination was vigorously enforced, as soon as it was realized that any attempts to restrict the movements of equines was well nigh impossible. In this country both formalized and attenuated vaccine has been administered to 60,000 animals. In Iraq mass vaccination has been followed by what appears to be very good results. On the other hand, Syria and Turkey have followed a policy of strategic vaccination by immunizing animals on the perimeter of infected foci. This policy has been adopted partly as a result of the short supply of vaccine due to the high cost of importation, and only at a later date will it be possible to assess the effectiveness of these measures. India and Pakistan in contrast have avoided the use of vaccine as far as possible. They have emphasised a slaughter out policy in addition to quarantine measures and strict control over the movement of equines. In a certain country the payment of over two million pounds in compensation for animals destroyed has given rise to an embarrassing economic situation.

From the outset many difficulties have been encountered in the programmes of control. In some countries field veterinary services are grossly understaffed, and their difficulties were accentuated by inadequate communications, poor or insufficient transport, and impassable terrain in the remote areas. Great difficulty was experienced with the animals of the migrating nomads, whose uncontrolled movements recognise no political boundaries. Before the full impact of the consequences of the epizootic was realised, ignorance and a lack of co-operation between the local populations and field personnel, made efficient mass vaccination very difficult. In the early stages the lack of co-ordination and exchange of information relating to the diagnosis and progress of the epizootic and its consequences, provided a pertinent reminder of the importance of regional or international control over diseases of this nature. Certainly one of the greatest problems encountered has been the serious lack of laboratories and trained personnel. In addition, the small animal colonies in the laboratories, were found to be hopelessly inadequate to meet the pressing demands of vaccine production. At present only the Razi Institute in Teheran is producing vaccine at the rate of 15,000 doses a week, while other laboratories in India, Turkey and the United Arab Republic will

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shortly go into production when these shortcomings have been overcome.

The effect of this disease on the economy of the countries within this region may not be immediately realised. The reduction in agricultural products, and the interference with the nomadic system through the death of over 50,000 horses, will add to the present financial burden of the immediate control measures, in countries where foreign currency is invariably scarce.

I will not attempt to forecast the future course of this epizootic. You will have noticed that favourable climatic factors exist which are closely associated with the biological cycle in endemic areas, and how, when the virus has once been introduced, mechanical transmission by dense insect populations, has followed the course of the great river basins. Our present knowledge of this cycle is incomplete, and we can therefore only hope that a severe winter will break the present epizootic, and that those links in the biological cycle do not exist within the region. While extensive vaccination will bring about immediate relief, the development of additional endemic foci outside the African continent, will constitute one of the most severe threats to the equine population in other parts of the world.

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DISCUSSIONS ON PAPERS DELIVERED BEFORE THE 55th ANNUAL CONGRESS

HOWELL—THE 1960 EPIZOOTIC OF AFRICAN HORSESICKNESS IN THE MIDDLE EAST AND S.W. ASIA. *Jnl. S.A.V.M.A. XXXI, 3,329.*

Dr. Alexander initiated discussion by an explanation of the amount of work that had to be carried out at Onderstepoort simultaneously with Dr. Howell's visit. The relationship between the Middle East virus and South African types had to be elucidated.

Dr. Weiss then informed the meeting that, according to isolates made from material from Pakistan, Iran and India, he had come to the conclusion that the outbreaks were apparently caused by one type of virus which was unrelated to any of the known seven South African types. Where the polyvalent South African vaccine was used, some 90% protection was obtained, which could be due to basic immunity, as their strain was not incorporated in the vaccine. Apparently the S.A. Type 6 gave a certain degree of protection through a slight overlap. Serum samples from India again, gave full protection against the Asian and four of the S. African types. In his opinion new virus types could still be found in Africa as the degree of protection given by the polyvalent vaccine was not always satisfactory.

Dr. Alexander continued by saying that 15 different isolates from the affected countries had been made by complement fixation. Some 4,000 mice were used in the process during a period of two months of very intensive work. The isolates proved to be either identical or closely related to one another.

One of the first essentials was to determine the immunity produced by the South African vaccine. According to the first report, which had just been received, some 48,500 horses in Iran had been immunized. Of these 187 had died subsequent to vaccination. Closer examination of the figures showed that 167 had died within 14 days, i.e. before immunity could develop. The South African vaccine, therefore, had proved to be very effective.

WEYLAND—"THE CONTINUOUS FLOW CULTURE OF MICRO-ORGANISMS AS A RESEARCH TOOL IN BIOLOGY AND BIO-CHEMISTRY." *Jnl. S.A.V.M.A., XXXI, 1, 7.*

In the discussion that followed this lucid review of the continuous flow culture as a useful tool in basic and applied research the possible advantage as a method of vaccine production was mentioned. Strain 19 Brucella vaccine was suggested as a suitable example of a product likely to benefit in stabilized quality and reliability. Disadvantages to

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be overcome included the high technical ability required of laboratory personnel and the increased risk of infection by cultures dangerous to attendants.

The adaptations necessary to employ the method for the purpose of toxin production did not seem insurmountable on theoretical grounds but practical considerations including staff requirements did present an obstacle to immediate application.

In Dr. Mason's experience the method had many uses.

Dr. van Drimmelen felt that the time had come for increased expansion of work of this kind at Onderstepoort both in basic research and in the field of vaccine production.

JANSEN—"THE DIAGNOSIS OF PULPY KIDNEY DISEASE."
Jnl. S.A.V.M.A. XXXI, 1, 15.

Q: How long are lambs born from vaccinated ewes protected?
A: The passive colostral immunity has been reported to last for about 5 weeks. At the same time lambs can be vaccinated when about one week old and given the secondary stimulus about seven weeks later. This applies when trypsin-activated, alum-precipitated vaccine is used and a detectable titre only develops after the secondary stimulus in passively protected lambs.

Q: What is the value of a length of gut, tied at both ends, and preserved in 50% glycerine, for the detection of epsilon toxin in its contents?
A: Epsilon toxin increases the permeability of the gut wall. When, therefore, a length of closed gut containing toxin is immersed in 50% glycerine, the hypertonic medium withdraws the toxin from the gut contents by osmosis through the gut wall. When the toxin is diluted in the glycerine it cannot be detected by any conventional method.

Q: What is the possibility of types of *Cl. welchii*, other than type D, being responsible for losses in sheep in South Africa?
A: In spite of continued search over the past years no other member of the *Cl. welchii* group could be associated with any outbreak of mortality in sheep in South Africa.

Q: Does chronic pulpy kidney occur?
A: Although pulpy kidney is an acute disease in the majority of cases, I have reason to believe that some cases take a few days to die. These exceptional cases are prostrate, comatose and may show a slight diarrhoea. I have not seen any such cases recover.

Q: Does *Cl. welchii* type D cause deaths in bovines in South Africa?
A: Although *Cl. welchii* type D enterotoxaemia has been described in calves, the condition has not been encountered in South Africa.

Q: How does the Onderstepoort Pulpy Kidney Vaccine compare with imported vaccines.
A: The Onderstepoort product has consistently shown a higher Lf value than imported pulpy kidney vaccines. This does not mean,

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however, that the imported vaccines do not fulfil their purpose. Any pulpy kidney vaccine which can produce a circulating epsilon antibody titre higher than 0.15 I.U./ml. is satisfactory. In terms of Act 36 of 1957 all vaccines imported into and produced in this country must be registered. Before registration all steps are taken by the technical advisers concerned to ensure that the remedies conform to any claims made in their favour.

A functional immunity can be produced against enterotoxaemia with relative ease. In this connection I may mention that at times farmers have reported continued losses from enterotoxaemia in spite of repeated vaccination against this disease. In several instances imported as well as Onderstepoort vaccine had been used. I had no hesitation in ruling that the continued deaths could not have been due to enterotoxaemia, especially in the cases where the Onderstepoort product had been used. The immunogenicity and lasting properties of the latter have been worked out in detail and convince me of the fact that mortality should stop about one week after its administration as a secondary or booster stimulus. In every instance where the continued mortality after repeated vaccination has been investigated, a cause other than enterotoxaemia has been established.

SCHULZ—"A REPORT ON NATURALLY-ACQUIRED BESNOITIOSIS, WITH SPECIAL REFERENCE TO ITS PATHOLOGY."

Jnl. S.A.V.M.A., XXXI, 1, 21.

Dr. Bigalke.—Professor Schulz mentioned in his introduction of the paper that he had found protozoal organisms in sections of the gut of a tabanid fly caught on a farm on which besnoitiosis occurs. I think than I can assist him in identifying these organisms. Although morphological details are indistinct, they are probably crithidia of a *Trypanosoma* sp. judging by their pallisade attachment to the gut. Tabanid flies are the intermediate hosts of *Trypanosoma theileri*, a parasite commonly found in cattle in these regions. I am convinced that Professor Schulz is dealing with crithidia of *Trypanosoma theileri*.

In his paper Professor Schulz suggests that transmission of besnoitiosis takes place biologically, and discredits the possibility of mechanical transmission on the grounds that all cases, produced artificially at Onderstepoort, have been mild. I must point out that Cuillé and Chelle, working in France, produced typical cases artificially, and that Professor Schulz and I observed the typical symptomatology of natural besnoitiosis in cattle infected artificially in the Rustenburg district. I feel, therefore, that there is no reason to discredit the possibility of mechanical transmission of besnoitiosis at present.

Professor Schulz.—I did not state categorically that the organisms in the gut of the tabanid fly were *Besnoitia besnoiti*. I mentioned the possibility and had the colour slide projected merely to indicate that it was a possible method of biological transmission. Histological sections were made from numerous ticks found on farms on which the

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infection occurred but I was unable to find parasites in them in this manner. This does not of course, mean that they do not play a rôle in the transmission of the disease.

The reason why I am more in favour of a biological method of transmission as against a mechanical method is that only a light reaction can be produced experimentally by mechanical transmission. I have read of other protozoal diseases that in order to cause a severe reaction must pass through an intermediate host. I speak under correction but I believe malaria to be one of these. If the method of transmission were entirely mechanical, then the distribution of the disease would be greater and one would also find more cases on infected farms.

REINECKE—"THE ROLE OF INFESTED DUNG IN THE TRANSMISSION OF NEMATODE PARASITES OF CATTLE."

Jnl. S.A.V.M.A. XXXI, 1, 45.

1. Dr. Thomas pointed out that animals with a diarrhoea or animals passing watery dung were rather dangerous in infesting pastures, because faeces in the semiliquid state sink easily into the soil surface and at roots of plants, thus creating favourable conditions for larval development. A wet season and lush pasture help considerably towards creating these conditions and thus aid the dissemination of infestation.
2. Attention was drawn to the dangerous and undesirable method or practice of spreading fresh dung over lands and pastures.
3. It was argued that it may be dangerous or undesirable to keep young stock too clean. Early calfhood infestation could help to build up an immunity.
4. Urine as a moisture factor does not aid larval development—on the contrary it is detrimental.

GROENEWALD—"NOTES ON TRACE ELEMENT REQUIREMENTS ACCORDING TO A RAPID SURVEY OF THE UNION."

Jnl. S.A.V.M.A. XXXI, 1, 55.

1. According to the discussion held and the photographs shown, it would appear that trace element deficiencies occur more generally in South Africa than previously suspected.
2. Cobalt is not required for plant growth and the higher the pH of the soil, the lesser is the extent to which cobalt, copper and manganese are taken up by the plants.
3. Certain deficiencies cause symptoms which are opposite to those of others, for example, a phosphorus deficiency causes sickle hocks, whilst a manganese deficiency causes straight hocks. In other words, where both phosphorus and manganese are deficient in the pasture, such deficiencies may have a mutually inhibiting influence on the symptomatology of each other.
4. Mutual interactions must not be lost sight of, as excess of molybdenum fertilisation increases the copper requirements of sheep

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grazing on such pasture and they may suffer from copper deficiency. An excess of fluorine intake may increase the iodine requirement and thus cause goitre in man, whilst the elevation of calcium and phosphorus contents in poultry rations may increase the manganese requirements.

5. As calves become older, some of the deficiency symptoms disappear, probably due to the slower rate of growth of the older animals, which thus require less of a particular trace element.
6. Reproductive disturbances do not necessarily precede trace element deficiencies, such as straight hocks, sway-back, etc., because in areas where sway-back does occur amongst lambs, the lambing percentage is not abnormally low. In cases of serious cobalt deficiency, abortions occur, while general reproductive ability is lowered in cases of copper and manganese deficiencies.
7. It would appear that indigenous cattle breeds, e.g. the Afrikaner, are less susceptible to trace element deficiencies than exotic breeds, e.g. Fries cattle, in other words herditary susceptibility to trace element deficiencies does appear to play a rôle.
8. The recommendations regarding the amounts of trace elements as given in the paper appear unnecessarily high. Copper sulphate, cobalt sulphate, etc., are poisonous in excess, and may cause abortions. Copper carbonate is less toxic than copper sulphate: it is more compatible with life and function of the ruminal bacteria.
9. The whole problem is most complicated and demands further and more intensive research. In such research not only pasture analyses but analyses of certain tissues and organs such as hair, blood, liver, ovaries, etc., must be undertaken for trace elements. Correlations between such values and growth, health, wool and milk production, and reproduction must be established.

TOMS, LOVEDAY, FREAN, HENNING—"SYMPOSIUM ON THE REARING AND CARE OF NEWBORN ANIMALS."

Jnl. S.A.V.M.A., XXXI, 1, 61–96.

1. Inoculation of lucerne hay, supplied early in the calf's life, with ruminal flora had proved not to be necessary.
2. In the procedure of calf rearing current at Onderstepoort—a modification of Preston's system (National Agricultural Advisory Services, Quarterly Review, Volume 35, page 18)—the calf is fed whole or skimmilk for the first 30 to 35 days of its life, when milk feeding is stopped abruptly. From the second week it also receives a good quality lucerne hay *ad libitum* and a calf starter, which is a dry meal mixture containing 20% skimmilk powder. Such a formula is excellent, but was considered much more expensive than a formula which used lucerne as a source of protein from an early age. By contrast, experiments would be conducted at Onderstepoort to determine how soon after weaning the calf starter can be replaced by an ordinary dairy meal.
3. The question of taking necessary steps to keep down the degree of

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worm infestation was dealt with in one of the papers cited by the author.

4. Dr. Boswell expressed his preference for brick as against concrete floors, but the author was against any type of floor on which calves could get soiled, and favoured wooden slatted duckboards, plain in summer and with some bedding in winter.
5. No practical method had yet been devised for the use of indigestible proteins, carbohydrates and fats fed with appropriate enzymes added beforehand.

HORAK, LE ROUX, BELONJE, DORE and VAN DRIMMELEN—"SYMPOSIUM ON REPRODUCTION."

Jnl. S.A.V.M.A. XXXI, 1.

1. Observations at Onderstepoort on over 500 oestrous cycles of over 200 cows of both dairy and beef breeds revealed ovulatory failure in 26 per cent.

2. The influence of age showed a remarkable contrast between the above two classes. In the dairy breeds (Frieslands and Jerseys) according to another speaker, the incidence of ovulatory failure increased rapidly after the fifth year, while in beef animals (Afrikaners) this type of physiological infertility was most prevalent in three year olds, and decreased rapidly to reach its lowest point in animals 10 years old and over. This incidence of delayed ovulation could be correlated with milk yield in the dairy breeds, the extra strain imposed by high milk production on the hormonal system probably gradually exhausting that system with advancing age.

3. As regards the interval elapsing between parturition and breeding, both dairy and beef breeds showed the lowest incidence of defective ovulation 60 to 90 days *post partum*, which suggests this as the optimum time for serving cows again.

4. Other factors which appear to have a profound influence on ovulation are nutrition and heredity.

5. Conception very rarely results if ovulation does not occur within 24 hours after insemination.

6. Inseminations at 12, 24 and 36 hours after onset of oestrus, indicate that the highest conception rate is attained at 24 hours. In practice, delaying insemination by 12 hours in the case of repeat breeders, increases the conception rate by 20%.

7. Delayed ovulation in about 80% of animals bred artificially, was mentioned by one participant in the discussions.

8. Dr. Horak admitted that it was possible that genital infections in the cows might have influenced the results of the observations recorded in his paper. However, shy breeders were culled from the herd in question after three barren seasons.

9. Dr. Horak stated that in his experience the different methods of semen collection did not materially affect conception rate, although this experience was not based on critical experimental evaluation. He thought electrical stimulation for semen collection not quite as good as using the artificial vagina, all else being equal. Time was, however,

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very limited during the breeding season, hence his preference for the electrical method.

10. Cervical insemination was preferred in cases of delayed ovulation, as the cervix was the most favourable environment for the survival of spermatozoa. Under conditions of natural service the bull ejaculates into the vagina, but a large percentage of sperms dies off. Nevertheless, a larger quantity of spermatozoa may survive than was deposited in the case of artificial insemination, accounting for the possible advantage of natural service over A.I. as far as conception rate was concerned.

11. A conception rate of 70–75% is difficult to exceed with A.I. Foetal reabsorption, which may be found to occur in all farm animals up to 3 months of pregnancy, probably played an important rôle, especially where nutrition was poor or in disbalance.

12. Retro-flexion of the penis may be a more desirable method of creating teasers than vasectomy.

13. Dilution of semen had no attenuating effect on the spermatozoa.

14. After manual examination of the genital tracts of cows per rectum, about 20% of them will come into heat within 3–4 days, probably as result of the palpation. The question of electrical stimulation of cows was raised.

15. Cows exhibiting a regular oestrous cycle had a higher conception rate.

16. The value of Vitamin A supplementation in ensuring a regular breeding cycle was repudiated by a number of participants in the discussions.

17. When the possible deleterious effect, of herding cows into a small camp for insemination, upon their condition as well as fertility was considered by the meeting, Dr. Horak pointed out that cows were collected in the morning and held for only about half an hour. On the other hand, herding of animals leads to trampling of the veld. Once enough bulls were obtained the idea was to do away with A.I.

18. Doubts were raised regarding the reliability of the culling of calves solely on a weight basis at a certain age. It was thought that the milk production of the cows should also be considred. Dr. Horak pointed out that calves not doing well, had not improved by the time they had attained 18 months of age.

19. It was generally agreed that heifers were under-fed as a rule. Doubt was cast on the adverse effects this might have on fertility.

20. Expression of the corpus luteum on the 17th day of the cycle, instead of the 10th to 12th day, to ensure ovulation at the ensuing oestrum, was contrary to generally accepted opinion and practice. With the use of progesterone and PMS many cows will come into heat, yet fertility is optimal only at the subsequent heat period. Nevertheless, Dr. Le Roux maintained that good results followed expression of the corpus luteum on the 17th day and serving at the first heat period.

21. With regard to salpingitis, oöphoritis and ovarian adhesions as late complications of vibriosis described in Dr. Le Roux's paper, one speaker found similar lesions in the absence of vibriosis. The

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cause was unknown, but the introduction of A.I. controlled the incidence thereof. The question arose whether infectious sterility had been considered adequately as a possible cause in such cases.

22. The opinion was expressed that a single irrigation with streptomycin-penicillin solution did not cure vibriosis, although it was more effective than using oxy-tetracyclines. Two treatments within a 24 hour period and the combined use of all three types of antibiotics seemed very promising.

23. The sugar test for detecting oestrus in equines and bitches held little promise, although work done by Dr. S. J. van Rensburg on the assay of glucose in cervical mucus during oestrus indicated that the test worked well in pigs, and was of use in bovines. In equines no glucose could be detected until after ovulation, and in bitches the reaction on the test paper was obscured by the colour of the cervical mucus during heat. In bitches a vaginal smear stained according to Schorr's method was of much greater value in determining the optimum mating period.

24. The opinion was expressed that exercise and the administration of Vitamin E appeared to improve fertility in dogs.

WEISS—"LUMPY SKIN DISEASE."

The discussion flowing from the film and commentary was directed at two aspects of the disease in particular, firstly the rôle that direct contact plays in the transmission of the virus, and secondly, the phenomenon of allergy, as encountered in lumpy skin disease.

It was made clear that direct contact transmission between animals, as also transmission through the handling of animals, does not play a major rôle. Transmission via the saliva, however, can occur as the virus may be present in the saliva of the reacting animal for as long as eleven days.

On several occasions so-called breakdowns of natural or vaccinal immunity to lumpy skin disease have been reported. In every instance the symptoms have been very mild and superficial. Two explanations of this phenomenon have been offered; firstly, that the animal was in fact suffering not from the Neethling type of Lumpy Skin Disease, but from the immunologically distinct Allerton virus infection, or secondly, that the animal, being immune to the Neethling virus, showed an allergic reaction on reinfection. This type of reaction has also been encountered when the Lumpy Skin Disease vaccine is administered to immune animals. All attempts at isolating Neethling virus from reactors of this type have ended in failure, thus lending strong support to the theory that the nodules present constitute in fact an allergic reaction.

Further discussions on the subject of lumpy skin disease revolved around the general symptomatology of the condition. The conclusion, drawn from the various opinions voiced, was that a very variable clinical picture could be expected in this disease.

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The excellent quality of this film, and the clear and concise way in which the subject is presented therein, was greatly appreciated by the members present.

DIETZ—"MODERN NARCOSIS IN HORSES, CATTLE AND SWINE."
Jnl. S.A.V.M.A. XXXI, 3,315.

In reply to three questions put by Dr. Reinecke the speaker answered: (i) he had used magnesium sulphate with chloral hydrate in horses but not in cattle; (ii) that he had not recognised a case of hypostatic atelectasis following anaesthesia of bovines, and (iii) he had no experience of cocaine administered intravenously as an antidote to chloral hydrate in horses.

Dr. Irwin: In a moment of aberration I once injected 5 ml. of chloral hydrate (1 gm/ml.) bilaterally instead of 3% procaine hydrochloride for cornual nerve block in a 350 lb. Jersey bull calf. In 15 minutes the animal was under light general anaesthesia, the horns were removed and fortunately no untoward sequelae were noticed in a three week's observation period. In reply to a question by Dr. Irwin, the speaker said he had not much experience of intravenous use of ether in the horse.

Dr. Muller: Large animal surgery is largely straightforward if good anaesthesia is obtained. I have only used morphine sulphate once in equines—intravenously in a mule—since the experiences of Prof. R. Clark in this regard were not promising. Because some drugs mentioned by Prof. Dietz are possibly unfamiliar to members, I will give some synonyms and pharmacological data concerning them. Methadone ("Polamivet", Diadone, Polamidone) was synthesized in Germany in 1941, and has since been manufactured widely in the U.S.A. It is used by physicians in the Union and has all the actions of morphia, but is less habit forming. As used by Prof. Dietz by intravenous injection, it potentiates chloral hydrate about three times. "Lethidone" (Nalorphine) reverses the action of Methadone as it does the natural morphine drugs.

G.G.E. refers to guaiacol (belonging to the cresol group), glycerine and ether. It is a muscle relaxant administered intravenously. In my experience Largactil (chlorpromazine) administered i.v. in horses causes excitement. I have used Pethedine i.v. followed by Largactil i.v. (both 1 mg./Kg. administered quickly) in trials, seeking an equine tranquillizer. The heart rate was increased, a jugular pulse was produced and one case of a group of eleven died, so the method appears unpromising. Chloral hydrate administered intravenously in doses around ½ oz. to horses remains an outstanding narcotic. The horse remains standing, and can stand on three legs, as in shoeing fractious individuals. Lastly, "potentiated narcosis" is the equivalent of "balanced narcosis."

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Prof. Dietz: Methadone is used in conjunction with Largactil in dogs, whilst barbiturates are used less commonly.

Dr. J. Doré congratulated the speaker, and proposed that his paper be published in the Journal, since the many facts could not be remembered readily.

Prof. Hofmeyr: I conducted trials with succynil choline chloride on 33 horses. On seven cases the E.C.G. was used and an alarming picture was produced; of the 33 cases two died. Thus my experiences are somewhat similar to those of British workers. On the other hand, Hanson has had only one death in about 1,000 horses and cattle: perhaps the barbiturate he used concurrently protected the heart in some way. My trials with "Neraval" in dogs (cf. Thiogenal in the paper red today) have not been encouraging. Evipan, first used about 20 years ago, is comparable to "Intraval".

Dr. Azzie: Is there a drop in blood pressure in using G.G.E. or succinyl choline chloride?

Prof. Dietz: There is an increase in blood pressure in 10–15 minutes.

SYMPOSIUM ON ANAESTHESIA AND SURGICAL SHOCK

Jnl. S.A.V.M.A. XXXI, 1, 143–157.

Dr. Doré showed his film on small animal anaesthesia with Fluothane, and its use in thoracic surgery. The President congratulated hom on his presentation, particularly as he worked only with non-professional assistants.

Dr. P. H. le Roux briefly emphasized a few points from his paper, and demonstrated the anaesthetic apparatus he uses.

Dr. D. Irwin: To facilitate positive pressure closed circuit anaesthesia either of the following points may be found useful:

(i) Replacement of the corrugated (expandable) tubing with polythene tubing. Incidently this is more readily sterilized, and fluid condensation in the system is observed.

(ii) Addition of unidirectional valves at the two T-pieces.

By either of these means the expansion of the lungs can be controlled more exactly, and any "coughing" (indicating too light anaesthesia) can be appreciated through change in bag pressure. The corrugated tubing cushions bag-lung contact.

Professor Hofmeyr in presenting his paper, drew attention to the salient points only, so as to leave time for discussion.

Dr. Muller: If neither blood nor plasma are available, intravenous injection of hypertonic solution may briefly delay fatal shock. Since nor-adrenalin ("Laevophed") is destroyed in the body in about four minutes, it is employed in a drip. The sodium lactate of Ringer's solution counteracts acidosis. In cases of hypoglycaemia, glucose should be added.

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Prof. Dietz: In shock, particularly when due to burns, cortisone is useful.

Dr. J. M. M. Brown: With reference to some points in Professor Hofmeyr's paper, I would like to add a few remarks. There are some easy and quick methods one can use to arrive at an estimate of the plasma volume, without the use of special apparatus or involved techniques, for instance the haematocrit and haemoglobin determinations. Given the weight of the animal, the normal figures for the haemotocrit and haemoglobin for that species and assuming the blood volume to be about 1/10 to 1/12th of the body weight, a rough assessment can be made of the plasma volume at any particular time. Similarly there are determinations which are quick and easy to perform which may be helpful in establishing a diagnosis of acidosis. I refer here to the bicarbonate and chloride of the plasma.

With reference to Dr. Muller's remarks, I must say I cannot see why he advocates the use of hypertonic solutions in shock or dehydration states. To my mind this is dangerous and should be avoided.

Prof. Hofmeyr: In a case of bovine *hydrops amnii* relieved by surgery, 35 gallons of fluid was drained, which amounted to half the weight of the patient. Intravenous fluid containing Laevophed was employed to overcome the paracentetic shock, and kept the pulse rate down.

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PROCEEDINGS OF THE 55th ANNUAL CONGRESS OF THE SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

The 55th Annual Congress of the South African Veterinary Medical Association was held in the Faculty Building, Onderstepoort, on August 29, to September 2nd, 1960.

The Welcome to guests, delegates and members by Prof. R. A. Alexander, Director of Veterinary Services, the Opening Address by Dr. S. M. Naude, President of the Council for Scientific and Industrial Research, the Reply thereto by the Vice-President of the S.A.V.M.A. on behalf of the Association, and the Presidential Address by Dr. H. P. Steyn follow immediately upon this report.

As Dr. P. G. Howell had just returned from a tour of investigation on African Horse Sickness in the Near and Middle East, undertaken at the request of F.A.O., his report was given in place of Dr. Lambrechts's paper on "International Animal Disease Control" and Prof. Alexander's report on "Foot and Mouth Disease in Southern Africa" by kind consent of the authors concerned. Dr. Howell's paper and the subsequent discussion appear in this issue (p. 329), as well as the paper read on invitation by Prof. O. Dietz on "Modern Narcosis in Horses, Cattle and Swine," (see p. 315).

Miss Rous's paper on "The Study of Foam Formation in the Rumen in relation to Forthy Bloat" took the form of a progress report and was not offered for publication.

The contents of papers by Dr. J. M. M. Brown on "Icterus and Photo-sensitivity: Modern Conceps regarding Aetiology" and by Drs. T. F. Adelaar and R. C. Tustin on "Bacterial Icterus in Sheep", and information emanating from the discussions, have been and will be dealt with in papers appearing currently in this Journal; consequently as part of the Congress Proceedings this aspect will not be published. There were no discussions on the last-named three papers, nor on that presented by Dr. W. D. Malherbe; probably because they were of a highly tecnhical nature.

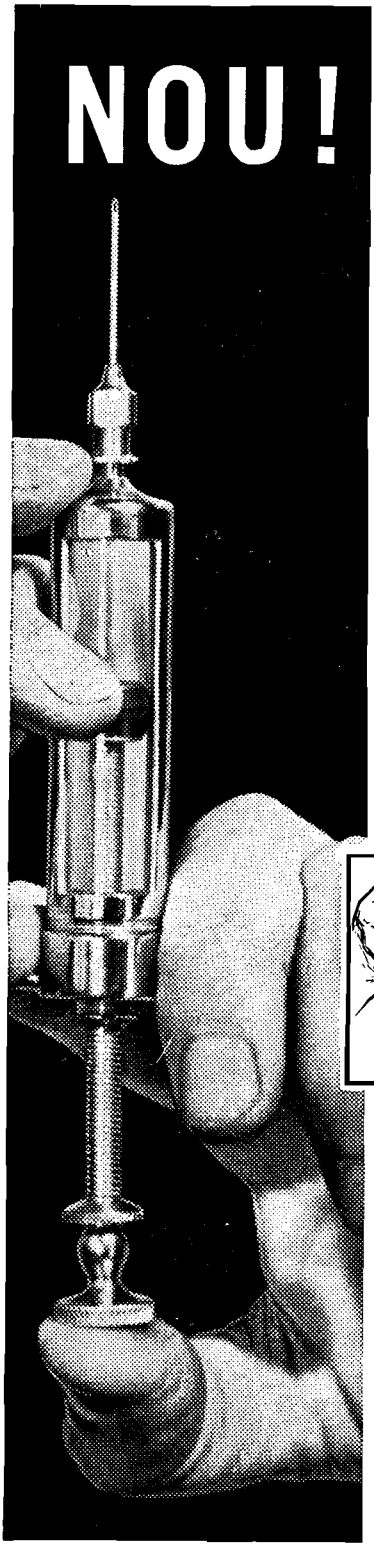
Dr. Weiss's report on "Lumpy Skin Disease" took the form of a film and is commented upon under "Discussion of Papers".

All the other papers delivered have been published in the Pre-Congress Issue of this Journal (Volume XXXI, No. 1). Edited versions of the discussions, if of sufficient merit, appear in this issue (pp. 335 to 345) under the heading "Discussions of Papers Delivered before the 55th Annual Congress."

A brief account of the Trade Exhibition will be found on page 419.

A Review of the proceedings of the Annual General Meeting of the Association with an abridged version of Minutes of Council Meetings appear at the end of this issue.

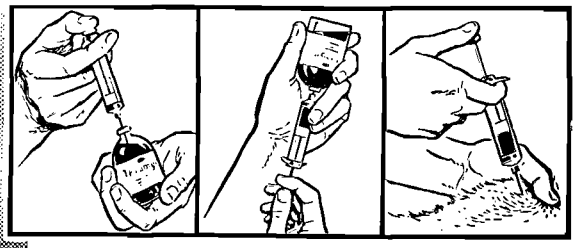
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WELCOME BY PROF. R. A. ALEXANDER, DIRECTOR OF VETERINARY SERVICES, TO GUESTS, DELEGATES AND MEMBERS ATTENDING THE 55th ANNUAL CONGRESS OF THE S.A.V.M.A.

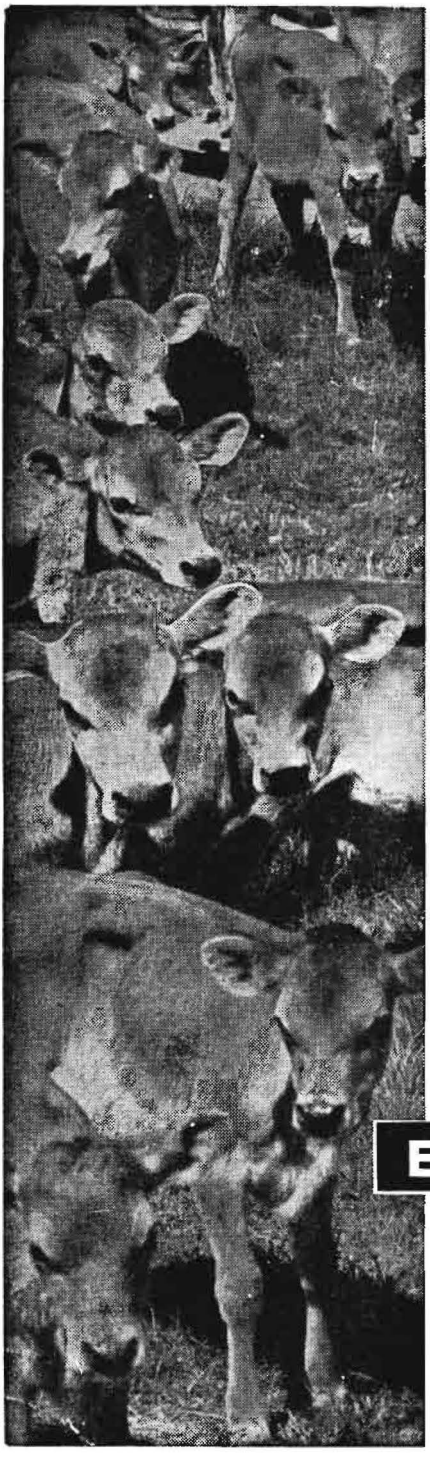
It is again my very great pleasure and honour to welcome the guests and delegates to this Congress of ours, the 55th Annual Congress of the Association.

It may seem strange but before I welcome these guests I want to mention in particular three of our prominent invitees who are unable to be present. Firstly, the Hon. Minister of Agricultural Technical Services, who extends his best wishes for a most successful congress and regrets that owing to pressure of work he is unable to be present personally. Similarly, Dr. M. S. du Toit, Secretary for Agriculture, regrets that he is unable to come and Prof. Rautenbach, Rector of the University of Pretoria, was unable to find time.

Amongst us as our guest is: Dr. Louis van den Berghe, Director-General of IRSAC—the Institute for Scientific Research in Central Africa. He is on his way to Cape Town to attend a meeting of the Scientific Council for Africa; he broke his journey to be with us and we hope he will be able to take part in the proceedings. Then we have as representatives Dr. R. J. Thomas for the British Veterinary Medical Association, Dr. G. K. Shone for the Federation. We have other visitors: Prof. Dietz of Berlin, who is visiting Onderstepoort at this time, and Dr. Dennig, a guest worker from Hamburg. Furthermore, we have Dr. B. M. Clark, Acting Secretary for Health, Dr. P. R. Viljoen. Dr. Chase, Dr. Tony Martinaglia, who has found time to come from Durban, and Dr. D'Abreu, from Moçambique. We extend to them all a very sincere welcome.

I apologize if I have omitted any names; I do so on behalf of my President, as it is his fault.

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ADDRESS DELIVERED BY DR. S. M. NAUDÉ, PRESIDENT, S.A. COUNCIL FOR SCIENTIFIC AND INDUSTRIAL RESEARCH, ON THE OCCASION OF THE OPENING OF THE 55th ANNUAL CONGRESS OF THE S.A. VETERINARY MEDICAL ASSOCIATION ON MONDAY, 29th AUGUST, 1960.

Mr. President, Ladies and Gentlemen,

It is to me a great honour that I, as physicist, should have been invited to open this, the 55th Annual Congress of the South African Veterinary Medical Association. There was a time, not very long ago, when a physicist was completely out of place at a Congress such as this. However, times have changed and the physicist, the chemist and the mathematician are no longer *persona non grata* at meetings of the biological sciences. It is realized in ever growing circles that the physicist, the chemist and the mathematician are able to make real contributions to the advancement of the biological sciences. Such physical subjects as electrical responses, radio-isotopes, low temperatures as well as electron microscopy are used to an increasing extent in the research on biological problems. In the field of chemistry the importance of trace elements and the work of the organic chemist are gaining in importance every year.

I trust that the invitation extended to me to open this Congress has been prompted by two considerations. Firstly, I hope that you feel that the contributions made by the C.S.I.R. to the advancement of the biological sciences in South Africa is proving to be of value. In the field of medical research, which the C.S.I.R. was called upon to explore shortly after its establishment, the Council has avoided the creation of laboratories. Instead, medical research units, groups and projects have been developed around individuals in existing institutions. On the whole, the results have been most gratifying. This scheme provides for maximum flexibility and individual initiative and excellent co-operation has been achieved between the different organizations which are concerned with these projects, for example the medical schools, the provincial hospitals, the Department of Health and the C.S.I.R's National Research Laboratories.

I sincerely hope that the second reason why I was invited to attend your Congress today is the realization that co-operation between the exact sciences and biological sciences can be very fruitful indeed. Co-operation between the exact sciences and the biological sciences has led to the creation of the so-called mixed field subjects like biophysics and bio-chemistry. These are comparatively new subjects and subjects in which some of the most dramatic and important advances in science nowadays are being made. In these subjects, experts from the different fields are brought together forming a team in order to utilize the specialized techniques and knowledge of every particular field in the study of a particular problem.

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In South Africa, co-operation of this type has been limited in extent up till now, but where it has been tried, the results have been most rewarding. For instance the National Chemical Research Laboratory has established several very close links with Onderstepoort. The programmes on rumen metabolism, "geeldikkop" and "vermeersiekte" have yielded fruitful results. Organic chemists in the C.S.I.R. have been very happy to participate in this work in conjunction with Onderstepoort and although neither problem has yet been solved, the mutual interaction of chemists and physiologists has led to results of greater significance than would otherwise have been obtained, had either worked in isolation.

It is significant that in this instance the chemical and physiological teams work in close collaboration but in separate environments, neither being subject to the other. It is a fairly well established fact that, to achieve the best results in any field of research, a scientist must work in a place where several other similar kinds of scientists are working. In other words, for success in a collaborative effort, teams of experts must be brought together, but an expert can only remain an expert in any particular field, if he is in constant and close touch with his expert colleagues in that field.

To quote one more example of the collaborative effort in South Africa: the water demineralization programme of the C.S.I.R. has, as one of its aims, a simple unit for farmers. This has led to the establishment of another bond with Onderstepoort in the form of a joint field experiment near Upington. The demineralization unit of the C.S.I.R. will supply drinking water to cattle from brack water supplies in this area and its quality and effects will be tested in cattle under controlled conditions by Onderstepoort.

I doubt that it is necessary to plead the cause of co-operation in science on an occasion such as this or before such an audience. I think we are all agreed on the necessity of full co-operation between the different fields of science. The question that remains is how to obtain the conditions under which this co-operation can best be achieved. Such co-operation must be on a research level, between the research scientists and the laboratories; it must be on the administrative level and finally it must also be on governmental level. A large percentage of money devoted to research in this country accrues to the different laboratories and institutes either directly or indirectly from the Treasury, and it is, therefore, only reasonable that the Central Government should have a say in how this public money is spent. We have, therefore, three different groups of people to satisfy. There are, firstly, the research scientist, secondly the administrator of scientific research and thirdly the guardians of public money. A scheme must be devised under which all three of these groups can be satisfied as far as possible.

The research scientist wants to work in an inspiring atmosphere, an atmosphere that can only be created by fellow scientists in his own or a closely related subject. He would also like to have reasonable access to modern equipment and assistance when required, and not six months or a year later. The research scientist would like to be

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able to draw, when necessary, upon the specialized experience, techniques and equipment of fellow scientists of other disciplines. The wishes of the research scientist can usually only be met in relatively large research institutes where the central facilities and services can be created under optimum conditions. These specialist services include such items as electronic workshops, instrument design and manufacture, mechanical workshops, good library facilities, translation facilities and information services.

The cardinal fact to remember in the administration of scientific research is that the administration is there to serve the research laboratories and not vice versa. On the basis of this fact alone it would seem possible for co-operation on the research level, the important level, to be organized fairly easily, irrespective of the administrative framework of the co-operating organizations. In practice, however, we find that co-operation between organizations, having vastly different frameworks of administration, becomes very cumbersome and sometimes well-nigh impossible. This has a most deleterious effect on the research climate. It is frustrating to scientists when they have to, and want to co-operate, on the research level and they find that around each corner they are being obstructed by purely administrative procedures of secondary importance. The organization of co-operative efforts between different laboratories and different organizations becomes much easier, much more simple on the administrative level if these organizations have the same or related frameworks of administration. It is then much easier, for instance for staff to be seconded from one institute to another, for instruments and supplies to be loaned or for special facilities to be created on a co-operative basis when necessary and with the necessary speed.

The demands of the Treasury, I think, can be summarized by saying that the Treasury, as the guardian of public money, is primarily concerned with ensuring that any expenditure is properly authorized and channelled according to parliamentary procedures. At present it must be very difficult indeed for Parliament and the Treasury to assess the amount of money that is spent annually on any particular field of research. If we take, for instance, the hydrological cycle in South Africa, starting with the rainfall and ending with the amount of water that flows into the sea from our river systems, we find that at least four different Government Departments are engaged on research in this one cycle. Firstly, there is the Weather Bureau under the Department of Transport responsible for climatology, weather forecasting and rainfall records; secondly, we have the Department of Mines, through the Geological Survey doing research on underground water; thirdly, there is the C.S.I.R's Institute for Water Research, mainly concerned with problems of water treatment and the treatment of municipal and industrial effluents and finally we have the Department of Water Affairs, responsible, amongst other things, for surface storage and irrigation Another example of this state of affairs we find much nearer home today, and that is money spent on medical and veterinary research in South Africa. Medical research is, to a large extent, financed through the C.S.I.R. and therefore through the Department of Commerce and

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Industries, whereas veterinary research, a closely allied subject, is financed and controlled by the Department of Agricultural Technical Services.

In the interest of research generally, and in the national interest, I think the time is now ripe for a thorough and complete overhaul of the whole organizational framework of State-supported scientific research in South Africa. A number of very prominent scientists have called for this reorganization and here I may mention the names of two of your most esteemed mebers, viz., Dr. P. J. du Toit and Prof. H. O. Mönnig.

A fundamental reorganization of the State-supported research framework in South Africa will necessitate some radical changes in the whole organizational structure as we know it today. It would, therefore, be wise to approach any reorganization with the utmost caution and to make sure of each step that we take. In considering any reorganization of the research framework in South Africa, there are a few guiding principles which should be borne in mind.

The first one is that the organization must be such that every research scientist is given the best opportunity for successful work, in other words, the right climate for research must be created and fostered throughout the whole scientific community in South Africa.

A second principle is that the barriers between the different subjects in science, especially in applied science, should be broken down. We are again approaching the stage where the physicist and the botanist, the biologist and the chemist, and the engineer and the agriculturalist will work more closely together in teams and utilize fully the specialized facilities, techniques, and knowledge which we still tend to divide into watertight compartments.

The third guiding principle which must, I think, be taken into account in any reorganization in our industrial and technological age, is that science and applied science are taking on an encompassing role in the welfare, the economy and future planning of countries. Today there is hardly a facet of everyday life that is not decisively influenced by scientific research, by the scientific and technological results that are constantly forthcoming from the research laboratories. For this reason the organizational framework of scientific research must be moulded to cater for this encompassing role that science is playing in modern society.

Finally, there are two more principles which cannot be overemphasized. The first one is that the administration of any particular scientific discipline should be entrusted to scientists thoroughly schooled in that particular discipline. There is nothing so frustrating to the research scientist than to be administered by somebody who has not a thorough background of the subject that he is administering—somebody for whom the t's must constantly be crossed and the i's dotted. I think that we must realize now that the time when somebody could say that any good organization must have a scientist on tap but not on top, are gone forever. This means that one cannot, for instance, take a physicist and expect him to administer a research institute specializing

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in a biological science, or conversely to take a biologist and ask him to administer a mechanical engineering laboratory.

The final guiding principle which I would like to mention is that the different research institutes should not be too small. The cross-fertilization of scientific research cannot be over-emphasized. As I said earlier, an expert can only remain an expert if he is constantly in touch with experts in his own or closely related fields. An institute must, therefore, be large enough to accommodate a number of experts in any one particular field for this cross-fertilization to take place. I do not thereby mean that research institutes should be allowed to grow to the extent where they become unwieldy, but there is a very real danger that our rather meagre scientific manpower may be so thinly dispersed over the country that we cannot foster institutes where the real research atmosphere is prevalent. Just as in the atomic pile, there is also a critical size under which a research institute cannot flourish.

Apart from this necessary interaction between scientists, there is also an economic reason why institutes should not be too small. An institute in any field of science requires certain specialist services and these services must be efficiently supplied for the institute to function effectively. These specialist services, associated with any particular field of research, also have a minimum size, which is, to a certain extent independent of the size of the institute that is being served.

The principles which I have just enumerated for the successful operation of scientific research, are, to an alarming extent, absent from the organizational framework of scientific research in South Africa at the moment. These principles do not only affect scientific research laboratories as such but also scientific services to the general public.

The Chairman of the Advisory Council on Scientific Policy, Prof. H. O. Mönnig, analyzed the scientific research framework in South Africa in his address to the Chemical Institute in Pretoria last year. He came to the conclusion that this framework needed a thorough overhaul and I most heartily agree with him. Prof. Mönnig made the sound proposal that four autonomous research councils be created to administer scientific research in South Africa. The four councils proposed are:—

(i) The C.S.I.R. which must be responsible for the physical and engineering sciences;

(ii) A research council for agriculture which will be responsible for biological research, including fishing research, forestry, botanical gardens, museums, etc.;

(iii) A medical research council responsible for medical, veterinary and dental research;

(iv) A fourth council responsible for geology, geomagnetism, hydrology, meteorology, and astronomy.

To these four councils a fifth must be added now, viz., the field of nuclear energy for which I think a separate council should be responsible.

We would, therefore, have the position that, under this proposed scheme, scientific services and scientific research would be adminis-

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tered by five independent and autonomous councils. This would make it possible for these councils, if they possessed a related or similar structure, to co-operate closely and smoothly on any research problem which required to be attacked jointly by two or more councils. The encompassing role that science is now playing in everyday affairs makes it imperative that these five independent councils should be co-ordinated at Governmental level in one department. The obvious way to co-ordinate the policies and activities of these five councils is through the office of the Prime Minister, because from this office access to the different Government departments and to all the different facets of the modern state can easily be arranged. This could most easily be achieved by the appointment of a scientific adviser to the Prime Minister in the same way as the Economic Adviser was recently appointed to the Prime Minister's Office. The Advisory Council of Scientific Policy, which is at present responsible to the Minister of Economic Affairs, should also be transferred to the Prime Minister's Office and the scientific adviser should preferably be the chairman of this council.

The Veterinary Medical Society of South Africa, one of the country's oldest and most respected Societies, can and I hope will play a decisive role in the creation of the new deal for science in South Africa. I hope that the Veterinary Medical Society will give its full support to a reorganization of science along the five principles which I mentioned and which more or less cover the organizational requirements of modern science.

Mr. President, in conclusion, allow me to wish you a very fruitful and happy Congress. I hope that your deliberations will be to the advancement of science in South Africa and to the advantage of every research worker engaged in the different aspects of veterinary medical work.

I now declare this Congress formally opened.

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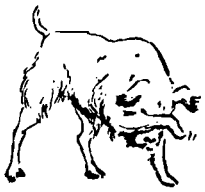
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REPLY TO DR. S. M. NAUDÉ BY PROFESSOR R. M. DU TOIT, VICE-PRESIDENT, ON BEHALF OF THE ASSOCIATION.

Mr. President, Dr. Naudé, Ladies and Gentlemen,

To me has fallen the privilege of replying on your behalf to the opening address of our distinguished guest, Dr. Naudé, President of the Council of Scientific and Industrial Research.

At the onset let me say at once that seldom have we had the privilege of listening to a more inspiring demonstration of a well reasoned and clearly thought out analysis of the organisational difficulties besetting scientific research in this country. This is a subject to which all of us have given a considerable amount of thought and one on which, we must confess, we have all been groping, if not in the dark, at any rate in a murky atmosphere. It is, therefore, indeed stimulating to hear the views of someone else on this subject, one who, by virtue of his past experience and the position he now holds, is eminently qualified to speak upon it.

In the last few years particularly, we have come to realize more and more the inter-dependence which exists between the various scientific disciplines and which is so essential for progress in practically every research undertaking. It is quite unnecessary for me to repeat what Dr. Naudé has already emphasized in this respect, in so far as it relates to our own particular field, namely, the close links which already exist between the physicist, the chemist, the bio-chemist and ourselves, links which are becoming stronger and more essential as our knowledge increases and our science advances.

One has listened with a great degree of admiration, and I might also say with envy, to the explanation as to how the C.S.I.R. operates in its research activities. I refer particularly to the efforts that are made to create the correct research climate and to allow the individual research worker the greatest possible amount of freedom to get on with his job and to free him so far as possible from routine chores which are inevitably a part of any civil service organisation.

A term used by Dr. Naudé, and one which is in general usage but which may need modification, related to the co-operation between what he called the exact sciences and the biological sciences. Biologists today may well afford to be touchy about subjects such as mathematics, including statistics, and physics being referred to as *exact* sciences when one sees what has been happening at Cape Canaveral. However, I do not intend enlarging on this, except to state that the further we go into an investigation of biological processes, the more we realize that we are in fact dealing with processes which operate according to cause and effect along clearly defined paths and according to immutable laws and little, if anything, is left to chance.

We will all, I feel sure, welcome the suggestions put forward by the Chairman of the Advisory Council on Scientific Policy and which are endorsed by our speaker, on the advisability of the creation of various autonomous research councils for the furtherance and coördination

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of all research activities in the Union. Five such bodies are envisaged, but Dr. Naudé stressed the necessity of thoroughly examining, step by step, all the implications connected with the creation of such councils and I cannot but strongly support the note of caution he sounds.

In so far as it affects ourselves, the Division of Veterinary Services is confronted with a multitude of tasks connected with the livestock industry, by no means the least of which is research, upon which our future progress depends. By its very nature research in the veterinary field cannot be confined to the laboratory bench, and field research—the detection and preliminary investigation of outbreaks of disease, and particularly of something new which always seems to crop up in this Africa of ours—is essential. Here we must not forget our field service who, apart from the watching brief they hold for us here in the research laboratories also have many regulatory duties to perform in carrying out the provisions of the Stock Diseases Act, duties which are difficult if not impossible to divorce from government service. I make no proposals but would like to add my word of warning to allowing ourselves to be involved in schemes without very carefully examining all the implications.

In conclusion, Mr. President and members I would like to express to Dr. Naudé on your behalf our appreciation and thanks for the most stimulating address he has given us and would like to assure him that we will watch the future development he has outlined to us with the greatest interest and, I might say, with a prayer towards their fulfilment.

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PRESIDENTSREDE GELEWER DEUR DR. H. P. STEYN TYDENS DIE OPENING VAN 55ste JAARKONGRES VAN DIE S.A.V.M.V., 1960

Soos gebruiklik wens ek alle lede van ons Vereniging ook hier te verwelkom. U teenwoordigheid is 'n treffende bewys van die belangstelling wat u het by die bedrywighede van u professie. Ek vertrou dat hierdie 55ste Kongres baie suksesvol en leersaam gaan wees.

As ek vanjaar in hierdie Presidentsrede nie sou verwys na die opspraakwekkende verklarings wat ek verlede jaar in my Presidentsrede gemaak het nie, sou dit beskou kon word as 'n erkenning dat daardie, verklarings foutief en ongegrond was, veral aangesien die waarheid van daardie verklarings ontken is deur sekere baie invloedryke persone. Om daardie rede ag ek dit my plig teenoor die veeboere, die veenywerheid en my professie om wat ek verlede jaar gesê het in meer besonderheid te ontleed en so ver moontlik met syfers en feite te staaf.

U sal onthou dat ek veral daarvan beskuldig was, dat, wat ek gesê het, aan kwaadwilligheid teenoor my professie grens, dat ek nie oor die nodige kennis beskik om 'n oordeel te kan vel nie, ens. Dit is seer sekerlik nie my bedoeling om hier te velde te gaan teen diegene wat 'n persoonlike aanval teen my gerig het nie.

Die beginsels, waarna ek, op my moontlik baie onbekwame en onervare manier, probeer strewe, gaan persoonlikhede te bowe. Dit is beginsels wat, volgens my mening, uiteindelik die welsyn van my professie ernstig beïnvloed, en as gevolg daarvan die welsyn van die veenywerheid in die land ewe ernstig gaan raak.

Ek mag terloops, en met alle nederigheid, daarop wys dat my kollegas klaarblyklik nie die persoonlike kritiek, wat teen my geloods is, onderskryf nie. Dit blyk uit die feit dat hulle my vanjaar onbestrede herkies het tot President van ons Vereniging. Ek waardeer die vertroue wat u sodoende betuig het en ek onderneem om u tot die beste van my vermoë te dien.

Na my toespraak van verlede jaar, en tot nou, het daar nog niks gebeur wat enigsins my verklarings as ongegrond bewys het nie. Om daardie rede en ten spyte van die aanvalle wat teen my geloods is, het ek geen gevoel van spyt oor my sogenaamde „uitlatings" nie. Ek het egter heelwat leed deurgestaan omdat dit geblyk het dat my bedoelings tot 'n mate deur sommige van my kollegas verkeerd vertolk is. Om 'n herhaling van so 'n wanvertolking te vermy, wil ek graag dadelik beklemtoon dat daar hoegenaamd geen bedoeling was om 'n enkele lid van my professie te na te kom nie. Inteendeel, ek wil nou daarop wys dat die bekwaamheid, die ywer, die pligsgevoel en getrouheid aan diens van my kollegas nooit ter sprake was nie. Verder wil ek daarop wys, dat die gehalte van die werk wat my kollegas binne die Staatsdiens, sowel as daarbuite, daagliks lewer, dikwels onder besonder moeilike omstandighede, alleen bewondering kan afdwing. Maar dit sê nog hoegenaamd nie dat al die probleme in navorsing, voorligting aan boere,

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laboratoriumsdiagnostiek en opleiding van toekomstige veeartse bevrediging afdwing nie. Die rede vir die ontevredenheid is nie enige gebrek aan bekwaamheid van die manne wat hierdie werk probeer onderneem nie, maar wel gebrek aan voldoende personeel. En hierdie gebrek is veral te vinde in staatsveterinêre dienste.

Dit sou as kortsigtigheid van my beskou kon word, indien my woorde die indruk wek dat hierdie tekort alleen bepaal is tot veeartsenykunde. Dit is seer sekerlik nie die geval nie. Maar as ons ander afdelings in die Departement Landbou in aanmerking neem, dan skyn dit asof die tekort veel ernstiger in die Afdeling Veeartsenykunde is dan in enige ander. Mnr. Uys, Minister van Landbou-Ekonomie en Marke, het verlede jaar te Bloemfontein, tydens sy opening van die S.A. Landbou-Unie-Kongres gesê dat die aantal landbouwetenskaplikes en tegnici vermeerder het van 1529 na 2580 gedurende die voorafgaande tien jaar. Die veeartsenypersoneel het, inteendeel, verminder van 99 na 93 in die afgelope 15 jaar, terwyl die vooroorlogse (d.w.s. 1938–1939) veeartsenypersoneel ongeveer 108 was.

In 1945 was die aantal beskikbare veeartsposte in die Staatsdiens 120. Daarvan het gedurende 1950 twintig poste verval as gevolg van 'n personeeltekort, en selfs die verminderde getal beskikbare poste is blykbaar nooit gevul nie.

Die Adams-Komitee het gesê dat daar in 1945 27 vakante veterinêre poste was en aanbeveel dat daar onmiddellik 'n verdere 34 poste geskep word om te voldoen aan die *minimum vereistes* van die veeartseny-afdeling. Van die 34 nuwe poste wat deur die Komitee aanbeveel is, sou 25 na navorsing moes gaan. Vandag is die totale aantal veeartse op Onderstepoort 42, waarvan 6 tydelike aanstellings is. Daar is dus slegs 36 permanente veeartsposte op Onderstepoort, almal gevul, en hulle moet doseer- en navorsingswerk, toesig oor entstofproduksie, diagnostiek, korrespondensie ens., ens., behartig. Die veldpersoneel het 62 poste, waarvan ses vakant is.

Aangesien hierdie syfers, so ver bepaal kan word, 'n werklike en akkurate weergawe is van die professionele personeel-posisie in die Veeartseny-Afdeling, vra 'n mens jouself af waar die Adjunk-Direkteur verlede jaar daaraan gekom het om in 'n persberig te sê dat die personeel posisie bevredigend is en dat verwag word dat alle vakante poste teen die end van 1959 gevul sou wees. Dit was duidelik 'n wanvoorstelling.

Ek het nie verlede jaar kritiek uitgespreek oor die bekwaamheid van my kollegas op Onderstepoort nie, of omtrent die gehalte van die werk wat deur hulle gelewer word. Dit is egter my plig om die haglike toestand, waarin veeartsenydienste verkeer, te openbaar, sodat georganiseerde Landbou gewaarsku kan wees omtrent die werklike toestand. Meer as dit kan ek nie doen nie.

Om 'n poging aan te wend om nuwe graduandi in die Staatsdiens te lok is Regeringsbeurse aan studente toegeken. Die poging het ook misluk omdat graduandi liewer die beurse na voltooiing van hulle studies terugbetaal, selfs teen 'n rentekoers van 10%.

My woorde van verlede jaar, dat Onderstepoort teer op sy roem van die verlede, het 'n storm van kritiek teen my persoon uitgelok.

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Laat my toe om aan u voor te lees wat sekere ander persone gesê het:

„Onderstepoort was originally intended as a research station. Its most important function is still research, but results of past research have brought new and additional duties to the Institution and because of the latter and the absence of adequate facilities and staff, research became over the past few years, almost a secondary function, to such an extent that lately there have been outspoken fears that the good name and reputation of Onderstepoort as a veterinary research institute will suffer gravely. It will be fatal to allow the present position to continue . . ."

„The outside world will judge Onderstepoort as a research institute by the amount and quality of its scientific publications."

Dit is gesê, here, deur die Adams-Komitee *vyftien jaar* gelede.

Ek erken rondborstig dat daar sekere belangrike verbeterings aangebring is sedertdien, veral wat betref 'n hele aantal pragtige geboue. Maar wat is geboue werd sonder die nodige personeel?

Ook is daar 'n baie groot verbetering in die Fakulteit aangebring. Die vernaamste verbetering wat in die Fakulteit aangebring is vir die daarstelling van 'n hele aantal voltydse doseerposte met salarisskale wat ooreenstem met die van ander soortgelyke poste in universiteite deur die land, is egter nie 'n gevolg van die Adamsverslag nie, maar is 'n direkte gevolg van die beleid en optrede van die Raad van u Vereniging. Terloops mag daarvan melding gemaak word dat die Fakulteit ongeveer drie jaar gelede voor die ernstige dreigement te staan gekom het dat daar 'n totale onderbreking van die opleiding in sekere belangrike vakke sou plaasvind. As gevolg hiervan is u Raad in die geleentheid gestel om 'n vasberade en besliste standpunt in te neem.

Daar was destyds 'n verskeidenheid van voorstelle aan u Raad gedoen, waarvan die doel was om 'n sogenaamde tydelike oplossing vir die kritieke toestand, waarin die Fakulteit verkeer het, te vind. Dit strek u Raad tot eer, dat hy absoluut eenparig aan sy besluit gekleef het dat tydelike oplossings meestal op 'n langtermyn tydelikheid uitloop, en dat hy met niks minder tevrede sou wees as 'n oplossing wat sou vermy dat daar weer na 'n verdere twintig jaar 'n herhaling sou wees van die destydse noodtoestand nie.

'n Belangrike Fakulteit soos die van Veeartsenykunde, en veral waar dit die enigste Fakulteit van sy soort in die land is, kan net nie bekostig om op tydelike oplossings voort te bestaan nie. Dit wek 'n ongesonde toestand van onsekerheid en ontevredenheid onder die personeel wat onwillekeurig verhoed dat werk van die hoogste gehalte gelewer word.

Die Adams-Komitee het ook verklaar dat daar gedurende die volgende vyftien jaar na die verskyning van sy verslag 'n beraamde vermeerdering van die veeartspersoneel in die Staatsdiens tot 200 behoort te wees, as daar selfs aan die minimumvereistes, wat aan veeartsenydienste gestel word, moet voldoen word.

As ons ons eie staatsveterinêre dienste vergelyk met dié van 'n hele aantal, indien nie die meerderheid, van westerse oorsese lande, is dit tragies om te sien hoe ver agter ons geraak het. In die meeste westerse lande blyk dit dat staatsveterinêre personeel verdubbel tot

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verviervoud is gedurende die afgelope 15 jaar. Daar benewens word private praktisyns op groot skaal gebruik om staatsdienste aan te vul. By ons het die getal verminder en word private praktisyns glad nie gebruik nie. Die onvermydelike gevolg van die haglike tekort aan professionele personeel is dat buitenavorsingsinrigtings gesluit moes word. Voor die wêreldoorlog was daar vier veterinêre navorsingsinrigtings in die Unie, vandag is daar slegs twee. In ons uitgebreide land is daar 'n oneindige verskeidenheid van probleme wat ontstaan as gevolg van heersende plaaslike omstandighede. Daar word maar karige pogings aangewend om plaaslike probleme te ondersoek en op te los. Dié probleme kan nie verplaas word na een of twee sentrale laboratoria vir grondige ondersoek en bevredigende oplossing nie.

Gedurende die afgelope jaar is daar 'n poging aangewend om op een of twee plekke streekslaboratoria op te rig. Maar weer vind die Direkteur van Veeartsenydiens hom voor die probleem: „Waar gaan die nodige personeel gevind word?" Weereens sê ek: „Pragtige geboue bied geen oplossing aan veeartsenyprobleme nie!"

Daar was tot onlangs 'n streekslaboratorium in die Noordelike Transvaal, maar ek verstaan dat die laboratorium ook nou gesluit is as gevolg van die bedanking van die veearts wat toesig daaaroor gehou het. 'n Streekslaboratorium sonder 'n veearts is natuurlik so goed soos 'n ouderwetse voertuig sonder trekdiere.

Die Direkteur van Veeartsenydiens het verlede jaar tydens die Wolkwekers-Kongres te Bloemfontein erken dat die werksaamhede op Onderstepoort gestrem word deur 'n ernstige personeeltekort. Hy het beweer dat ons minstens tien ervare navorsers uit die buiteland moet invoer. Daarteen kan ek baie besware opper. Om maar een te noem, blyk dit onwaarskynlik dat ons beter navorsers oorsee sal vind as wat hier opgelei kan word, en aangesien die diensvoorwaardes in die Staatsdiens nie aantreklik genoeg is om ons eie graduandi te lok en te behou nie, kan ons seker nie verwag om ervare navorsers van elders te trek nie.

Met die huidige personeeltekort is dit onmoontlik om fundamentele navorsing aan te pak. Die geldelike waarde van fundamentele navorsing word oor die wêreld erken, byna afgesien van hoe groot die onkoste daaraan mag wees.

Dit is nie nodig om baie diep in ons veeteeltprobleme te delf om te kan sien hoe noodsaaklik fundamentele navorsing mag wees nie. Die S.A. Landbou-Unie het onlangs sy verontrusting uitgespreek oor die onbevredigende aanwas in die veestapel van die Unie. 'n Paar belangwekkende syfers sou nie hier onvanpas wees nie:

Volgens Henning (1956) en Harwin (1957) word daar beswaarlik een kalf van drie volwasse koeie per jaar in die Unie grootgemaak. Lambrechts (1950) beweer dat die vrektesyfer onder kalwers in Noordelike Natal 22 persent is. Die voorsitter van die Natalse Landbou-Vereniging het verlede jaar beweer dat slegs sowat 30–40% van die kalweroes in party dele van Natal grootgemaak word. Thomas (1956) beweer dat die sterftesyfers onder kalwers in die Noordelike Transvaal 40% beloop. Ons sien dus dat die vrugbaarheid van ons beeste besonder laag is en dat daar buitengewoon groot verliese is onder die

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paar kalwers wat aankom. Dit is nie 'n veeteeltprobleem nie. Dit is 'n egte veeartsenykundige probleem.

Ten spyte van hierdie tragiese toestand is daar, sover my kennis strek, nog geen bevredigende ondersoek ingestel na die werklike oorsake van die toestand nie. Om te beweer dat ons volgens algemene kennis van heersende toestande in die land weet wat die oorsake is, bring maar skrale troos aan die lydende beesboere. Dit sou ook nie die waarheid wees nie, want voor grondige navorsing uitgevoer is, gaan alles na gissing.

Die verhouding van beeste wat jaarliks geslag word tot die getalle vee in die land is seker die swakste in die westerse wêreld, d.w.s. 10%. Die syfers tot my beskikking dek nie alle westerse lande nie, Amerika is nie ingesluit nie, maar vir die lande waarvoor ek syfers gesien het is die verskil treffend. Die jaarlikse vrektesyfer by ons is seker een van die hoogste, indien nie die hoogste in die beskaafde westerse wêreld.

Die getal onaangepakte probleme is legio. Dit is onnodig om hulle hier op te noem, maar die onvermydelike gevolg is dat veeboerdery nie naby so lonend is as wat hy behoort te wees nie. Dit is ongetwyfeld een van die oorsake dat daar 'n voortdurende trek van die platteland na die stede is, wat soveel besorgdheid van regeringsweë opwek. Om die neiging te probeer teëwerk word geweldige bedrae aan boerebystand ensomeer gestem, terwyl sommige belangrike fundementele oorsake agterweë gelaat word.

Moet asseblief nie van laasgenoemde opmerking aflei dat ek in die minste teen boerebystand gekant is nie; ek noem dit slegs om te beklemtoon hoedat die aanvaarding van tydelike noodshulp dikwels nie in die minste bydra tot 'n permanente oplossing van 'n vraagstuk nie, en ek wil so ver gaan om te beweer dat 'n deegliker oplossing van ons veesiekteprobleme met groter sukses die red van veeboere sal bewerkstellig as sommige van die talle boerebystandskemas wat oor die jare aan boere verleen is.

Om die saak anders te stel, mag gesê word dat die gevraagde uitbreiding aan veeartsenydienste en navorsing regmatig beskou mag word as 'n kerngesonde vorm van boerebystand, met die een groot voordeel dat die hulp aldus verleen van 'n baie meer blywende aard sal wees as enige geldelike hulp wat natuurliker wys ook van tyd tot tyd verleen moet word. Daarby sou ek sê dat hulp van dié aard mettertyd tot gevolg sal hê dat veeboere al hoe minder geldelike hulp sou nodig hê, namate hul veeboerderybedryf meer lonend word.

Ons hoor dikwels van die geweldige vooruitgang wat veeboerdery gemaak het gedurende die na-oorlogse jare, en veral gedurende die afgelope 10 jaar. As mens jou beperk tot die vermeerdering aan waarde van ons veestapel is die bewering juis, maar is die vermeerdering aan waarde nie slegs die gevolg van die vermindering van die koopkrag van geld nie? Die onlangse verskyning van „Uniestatistieke oor Vyftig Jaar" onderstreep die juistheid van laasgenoemde bewering. Daarin sal gevind word dat daar in werklikheid, wat getalle beeste en skape betref, geen noemenswaardige vermeerdering oor die twintig jaar eindigend 1957 plaasgevind het nie. Dit is die laaste jaar waarvoor al die syfers, wat ek moes gebruik, aangegee word. In 1937 is die totale

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getal beeste aangegee as 11,188,000; in 1957, 12,042,000; 'n vermeerdering dus van 854,000. Die respektiewelike getalle skape was 41,034,000 en 38,241,000: 'n vermindering van 2,793,000. Die getal beeste in besit van blankes in 1937 was slegs 6,197,000 en in 1957, 6,976,000: 'n vermeerdering van ongeveer 'n driekwart miljoen.

As ons die syfers vir wol nagaan, vind ons dat daar gedurende 1937 255,148,000 lb. en in 1957 302,310,000 lb. verkoop is. Die waarde van die wol in die ooreenstemmende jare was £13,834,000 en £72,265,000. Dit wil sê, alhoewel die wolopbrengs oor 'n twintigjaartydperk slegs gestyg het met 47,162,000 lb., die totale waarde in 1957 gestyg het tot vyf keer dié van 1937. Vir die jare 1958 en 1959 is daar 'n klein daling in die hoeveelheid geproduseerde wol en 'n aansienlike daling in waarde. Laasgenoemde is egter toe te skryf aan prysdalings.

Wat vleisproduksie en suiwelprodukte betref, vind ons dat die waarde van beeste geslag gestyg het van £4,941,000 in 1937 tot £35,721,000 gedurende 1957, d.w.s. 'n vermeerdering van ongeveer sewe keer; bokke en skape geslag het vermeerder van £3,344,000 tot £21,125,000, 'n vermeerdering van ongeveer ses keer; en suiwelprodukte van £5,266,000 tot £42,174,000, 'n vermeerdering in waarde van ongeveer ag keer. Die vermeerdering in fisiese volumeindeks vir beeste geslag was slegs van 93 tot 158; vir bokke en skape van 97 tot 108 en vir suiwelprodukte van 98 tot 121. Dit is dus duidelik dat die vermeerdering aan waarde enige vermeerdering aan opbrengs ver oortref. Van die voorafgaande is dit duidelik dat die vermeerdering aan waarde van ons veeteeltbedryf hoofsaaklik 'n kunsmatige toestand is.

As ons verder die syfers vir veeverliese oor dieselfde periode ondersoek, dan vind ons dat daar geen vooruitgang gemaak is met die inkorting aan die geweldige jaarlikse veeverliese nie. Per toeval was die verliese aan beeste gedurende 1957 byna twee keer die van 1937, terwyl die skaapverliese ietwat minder was. Aan dié syfers kan geen besondere waarde geheg word nie, behalwe om aan te dui dat die verliese van jaar tot jaar nie verminder het nie en gewoonlik tussen 657,000 tot 1,257,000 beeste en 1,891,000 tot 2,719,000 skape beloop, oor die periode wat ons in oorsig neem. Dit is die totale verliese, insluitend die verliese van vee van nie-blankes. Die verliese aan beeste deur blanke eienare gely gedurende 1957, die laaste jaar waarvoor syfers tot my beskikking is, het 7.2% van die beeste in hulle besit beloop. Bantoes het gedurende dieselfde jaar 8.9% van hul beeste verloor. Die verskil tussen die verliese van blanke en Bantoe beeseienare is gewoonlik groter. Gedurende 1937 was die onderskeidelike syfers 4.7% en 7.3%. Dit sou moontlik gevaarlik wees om enige poging aan te wend om tot enige gevolgtrekkings te raak net van dié syfers, maar dit is treffend dat daar nie 'n groter verskil tussen die verliese van blanke en Bantoes is nie.

Die totale jaarlikse vermindering van beeste in die Unie as gevolg van slagting en vrekte, beloop dus ongeveer 15% tot 18%. 'n Syfer wat aansienlik laer is as die persentasie beeste wat in baie oorsese lande jaarliks vir gebruik geslag word, en tenspyte daarvan is daar feitlik geen vermeerdering van die beesbevolking van die Unie nie.

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Van die voorafgaande kan ons met reg aflei dat ons vee-boerdery, wat getalle betref, in werklikheid min of geen vooruitgang gemaak het nie. In werklikheid was die getal skape in die Unie een tyd baie groter as ooit daarna, nl. in 1930, toe die totale getal skape op 48,358,000 beraam is. Droogtes en beperkte drakrag van ons weiding het blykbaar veroorsaak dat skaapboere noodgedwonge minder skape aanhou.

Daar is egter 'n klein verbetering aan opbrengs te bespeur. Ons produseer 47,162,000 lb. meer wol van 1,473,000 minder wolskape, en die fisiese volume beeste geslag het vermeerder van 93 na 158, en skape en bokke van 97 na 108. Werklike vermeerderings aan gewig van vleis geproduseer, is nie tot my beskikking nie.

Ek is daarvan oortuig, en ek spreek die opinie uit nie alleen as veearts nie maar as boer met boerderybelange in twee provinsies van die Unie, dat baie bygedra kan word tot 'n betekenisvolle verbetering aan ons veebedryf deur 'n grootskaalse uitbreiding van navorsings- en voorligtingsdienste, waaronder veeartsenykunde ongetwyfeld die toonaangewende rol behoort te speel.

Die verbeterings wat ek beoog en nastreef sou ingrypend wees, maar voor ons enige merkwaardige vooruitgang kan verwag, sal hulle noodgedwonge aangebring moet word. Die verbeterings en reorganisasie gaan nie net die Veeartseny-Afdeling van die Departement Landbou aan nie, maar ook veeteelt, wat seer sekerlik 'n byvoegsel tot veeartsenydienste behoort te wees. Die samewerking en eenwording van die twee naverwante groepe is 'n klaarblyklike vereiste, as die welvaart van ons veenywerheid ons vernaamste besorgdheid is.

Daar behoort nie in so 'n organisasie ruimte te wees vir persoonlike of vak-afguns nie, en dié ideaal is nie te vinde onder die huidige organisasie nie.

Here, ek is verlede jaar deur 'n Staatsamptenaar daarvan beskuldig dat ek 'n „buitestaander" is. Dit is natuurlik 'n baie juiste beskuldiging sover dit die Staatsdiens aangaan, maar as daarvan afgelei word dat ek 'n „buitestaander" is aangaande veeartseny- en veeteeltprobleme, moet ek die bewering beslis ontken. Ook wil ek u graag daaraan herinner dat ek twaalf-en-'n-half jaar in die Staatsdiens werksaam was en gedurende dié tyd genoeg ondervinding opgedoen het om 'n opinie te kan uitspreek.

Ek het egter nie verlede jaar 'n aanval teen die Staatsdiens as sodanig gedoen nie, en is nie van plan om dit vanjaar te doen nie. Ek herhaal egter, dat die werksaamhede en pligte van 'n staatsdiens en dié van 'n groep professionele mense nie vereenselwig kan word nie.

Sover het ek my hoofsaaklik daarby bepaal om te probeer bewys lewer dat die navorsingsdienste van die Veeartseny-Afdeling onbevredigend en ontoereikend is om die doel wat ons graag wil bereik, nl. vooruitgang van die veenywerheid, te bevorder. Ek herhaal, dat dit nie toe te skryf is aan enige gebrek aan die gehalte van werk wat wel onderneem en verrig kan word nie, maar aan gebrek aan personeel en fasiliteite.

Een van die vernaamste oorsake van die gebrek aan voldoende personeel is ongetwyfeld die onbevredigende en karige beloning wat deur die Staatsdiens aan nuwe graduandi aangebied word. Daar word

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beweer dat groot verbeterings aangebring is in die salarisskaal wat aan veeartse aangebied word. Ek ontken dat dit die werklikheid is, en wel om die volgende vername redes.

Vroeër het ek gesê dat die vermeerdering aan waarde van ons veeteeltbedryf en sy produkte hoofsaaklik te danke was aan die verminderde koopkrag van die pond. As u ,,Uniestatistieke" wil naslaan, sal u binne 'n half-uur uself kan tevrede stel dat die £1.0.0 van 20 jaar gelede maar ongeveer 8/– vandag werd is; m.a.w., as 'n beginsalaris gedurende 1939 £400 was, moet dit vandag ongeveer £1,000 wees om gelyk te staan aan die 1939-salaris. Voor daar dus in werklikheid van enige verbetering van salaris gepraat kan word, moet daardie salaris meer as £1,000 per jaar bedra. Dit blyk dus dat die huidige aanvangsalaris van £750 wat aan veeartse aangebied word in werklikheid 'n kleiner salaris is as die van 1939.

Hierdie knellende klagte omtrent onbevredigende salarisskale, verlies van voorregte ensomeer, is 'n toestand wat ek kan onthou vir solank as ek veearts is, d.w.s. byna 29 jaar. 'n Sprekende bewys dat die klagtes goed gegrond was, is te vinde in die feit dat toetredings tot die staatsveterinêre poste nie kan tred hou met bedankings en normale aftredings nie. Ons moet dus aanvaar dat dit 'n permanente en vaste beleid van die Staatsdiens is om 'n toestand te handhaaf wat nie veeartse in die Staatsdiens sal lok nie.

Daar is natuurlik goeie en gegronde redes hiervoor, en daarom sê ek dat 'n permanente oplossing van die haglike toestand van ons veeartsenydienste nie te vinde is binne die raamwerk van Staatsdiens nie. Ek herhaal wat ek verlede jaar gesê het,–dat dit gebiedend noodsaaklik is dat veeartsenydienste gereorganiseer moet word buite die Staatsdiens. En ek wil graag vanjaar 'n stap verder gaan en aanbeveel dat dit moontlik, na behoorlike ondersoek, sal geld vir die hele Landbou-Departement, maar ook veral vir die Afdeling Veeteelt. Ek kom terug op my aanbeveling van verlede jaar dat die skepping van 'n landboukundige navorsingsraad 'n moontlike oplossing gaan wees.

Tot dusver het ek my bepaal by veeartsenykundige navorsing. 'n Ander besonder belangrike funksie sou 'n doeltreffende voorligtingsdiens wees.

Ons veeboere het oor die jare byna 'n soort van vrees vir Staatsveeartse ontwikkel, omdat hulle hoofsaaklik die Staatsveearts in verband met die toepassing van die Veesiektewet sien. As gevolg hiervan is daar 'n gebrek aan die gewenste goedgesindheid wat teenoor veeartse onder boere te vinde behoort te wees.

Dit is ook heel duidelik dat goeie navorsing hand in hand met goeie voorligting moet gaan, om sodoende die waarde van akademiese kennis op praktiese wyse beskikbaar te stel aan boere. Dit sou natuurlik 'n verdere vermeerdering van personeel vereis.

Daar is 'n bykomstige probleem wat ons in die nie baie verre toekoms afwag en dit is, dat Bantoes opgelei sal moet word om in hulle eie gebiede veeartsenydienste en voorligting aan hulle eie mense te verskaf. Hierby bedoel ek nie elementêre opleiding nie.

Daar is gedurende die afgelope jaar geweldige publisiteit verleen aan die ontdekking en beskikbaarstelling van 'n nuwe entstof teen

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knopvelsiekte. Die nuwe entstof is 'n besliste en merkwaardige vooruitgang en ek wil graag van die geleentheid gebruik maak om die Direkteur van Veeartsenydiens en Dr. Weiss, en Onderstepoort, van harte geluk te wens met 'n nuwe prestasie.

Voor ek sluit moet ek nogmaals terugverwys na wat ek verlede jaar gesê het, toe ek verklaar het dat dit tot die nadeel van ons professie strek dat ons Fakulteit nie volle akademiese status geniet nie. Daarop is geantwoord dat my sogenaamde bewering onjuis is en dat die lede van die Fakulteit wel akademiese status geniet.

Laat my aan u voorlees wat die Adams-Komitee daarop te sê gehad het:

> „As full-time civil servants the teaching staff are subjected to many handicaps and disadvantages in comparison with professors and lecturers in other faculties of the universities. Thus they have only thirty-eight days vacation leave per annum compared with some four months university recess; they work the full service hours; they are debarred from engaging in private practice or undertaking other remunerative work; . . . they do not enjoy the 'academic freedom' of speech."

Baie ander nadele wat die professie as geheel tref kon genoem word, maar dit is onnodig om verder uit te wy.

Ek is verlede jaar gevra om bewys te lewer van die stellings deur my gemaak. Ek het vanjaar probeer om sover moontlik op 'n onpersoonlike en geheel objektiewe wyse sekere bewerings van my met feite te staaf. My pogings mag misluk het: ek laat dit aan die gesonde oordeel van my kollegas en die veeboere van die Unie oor om tot hulle eie besluit te raak. Wat ek 'n jaar gelede gesê het, en vandag probeer staaf het, is gemotiveer deur 'n opregte besorgdheid oor die welsyn van my professie en dié van die veenywerheid.

Ons het vanjaar Sy Edele mnr. le Roux, Minister van Landbou-Tegniese Dienste, genooi om hierdie Kongres te open. Dit spyt my dat hy weens ander belangrike pligte nie hier teenwoordig kon wees nie. Veral sou ek hom graag persoonlik wou ontmoet het, en sou ek graag wou hê dat hy persoonlik moes hoor wat vandag hier gesê is.

In conclusion, ladies and gentlemen, it must be very obvious that in an address of this nature it would be impossible to deal with all aspects of the problem which I have endeavoured to cover. It is only possible to discuss the salient features and emphasize those points which may stimulate sufficient interest to cause a more thorough investigation to be brought about. I know everybody is not satisfied with what I have said, and some are probably going to be extremely annoyed, but I have endeavoured to present the facts to the best of my ability and for what they may be worth. I make no apologies for having done so. To me and to the majority of my colleagues there is very good cause for grave concern about the future of our profession. I trust that many people outside the profession, especially stock farmers, will develop the same concern, although possibly for somewhat different reasons, and that as a result they will assist us to bring about the long overdue and much desired improvements.

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There are many improvements concerning my profession which I would like to see brought about, and although I assume full personal responsibility for what I have said today and have certainly not spoken on behalf of Council, I believe I can say without fear of contradiction that Council and the Profession wish to see many improvements, e.g. we would like to see the Veterinary Act amended and brought up to date, we would like fuller and more adequate representation on all bodies dealing with matters appertaining to animal health, animal industry, education of future veterinarians as well as the veterinary education of people in closely related subjects. And finally and above all we would like to receive full acknowledgment of our professional status.

PRESENTATION TO PROF. E. M. ROBINSON

During the course of the proceedings of the 55th Annual Congress the President made a presentation to Prof. E. M. Robinson and thanked him most sincerely on behalf of the Association for his valued services as honorary editor of the Journal for the long period of twelve years. In reply Prof. Robinson expressed surprise at and thanks for this unexpected tribute. He went on to say that he had enjoyed the work, but had found proofreading a laborious task. He also suggested that the Journal might need a full-time editor and that a monthly issue might be considered.

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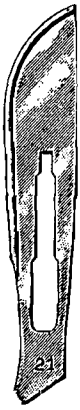


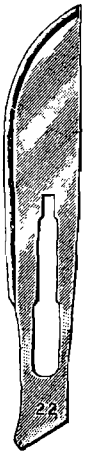
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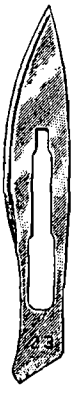
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CLINICAL INVESTIGATION INTO THE VALUE OF METHITURAL SODIUM (NERAVAL SODIUM—SCHERAG) AS A SHORT ACTING ANAESTHETIC

C. F. B. Hofmeyr*

Methitural sodium (Neraval, Scherag) is a short acting thiobarbiturate. It is irritant and should not be deposited outside a vein. A five per cent solution is the most convenient.

INTRODUCTION

Reifferscheid and Dietmann[5] tested a preparation (AM 109—identical to methitural sodium) on ten students. Compared with other thiobarbiturates the period of anaesthesia was somewhat shortened and post-anaesthetic depression was absent.

Dietmann[2] advocated rapid injection, i.e. 2–3 ml. of a 10 per cent solution intravenously, followed by 1–2 ml. repeated until anaesthesia supervened. After the first injection respiratory depression was observed. In spite of the fact that some authors maintain that morphine or its derivatives should not be used in premedication before barbiturate anaesthesia, for fear of summation of respiratory depression, morphine and atropine were used before Thiogenal† with good results. In 500 observations in man 5–10 per cent. of cases evinced vagotonic manifestations.

Wille[8] reported on the use of Thiogenal on pigs. He stated that, in general, respiratory depression was slight. He quoted von Zima, v. Werder and Hotovy as giving duration of sleep 52.3 minutes $\pm$ 4 and of anaesthesia 28.7 minutes $\pm$ 1.7. A 10–20 per cent solution was used depending on the size of the pig.

A paravenous injection of 10 per cent solution led to local inflammation and of 20 per cent. solution to limited necrosis. Rapid injection appeared desirable. Smooth induction was the rule with a wide margin of safety and rapid recovery. The dose varied between 25–30 mg./Kg. for young pigs and about 20 mg./Kg. for older pigs. No deaths occurred in 203 cases.

Ullrich[7] used 75 mg./Kg. Thiogenal intraperitoneally as an aneasthetic in cats.

Tolle[6] found 40 mg./Kg. to be the anaesthetic dose in the dog. He advised giving half the calculated dose at once, with further injections according to the first response. He mentioned parasympathetic stimulation as evidenced by salivation. In 228 cases in dogs one death occurred. In 31 cases in pigs the observations of Wille were confirmed.

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† Thiogenal is identical to methitural.

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Tolle also agreed with Ullrich that Thiogenal is excellent for intraperitoneal anaesthesia in cats. His experience was based on 17 cases.

Irwin *et al.*[3] made a comparative study of methitural and thiopental in the cat, dog and monkey. In the dog cumulative action could only be demonstrated with methitural after nine hourly doses, whereas it was shown after three doses with thiopental. Body tremors and twitches were observed occasionally with methitural in the cat and dog, but not in the monkey. Atropine was found compatible with this drug, and 0.1 mg./Kg. abolished salivation. Depth and duration of surgical anaesthesia were enhanced. The anaesthetic action of methitural was significantly shorter than that of thiopental.

Blake and Perlman[1] conducted studies on the metabolism of methitural and thiopental. Both were absorbed rapidly into the body fat depots. Methitural was degraded by the liver and eliminated more rapidly than was thiopental.

Lieberman[4] gave seven representative case histories of methitural anaesthesia in dogs and cats, most of which had some form of premedication, including atropine. No mention was made of side effects.

The Effect of Repeated Anaesthesia

Five young cross-bred fox-terrier bitches were anaesthetized with 5 per cent methitural sodium intravenously, often at daily intervals. Half the computed dose (50 mg./Kg.) was injected fairly rapidly. Exactly one minute later, one quarter the computed dose was given. At the end of the second minute this latter dose was repeated, unless a smaller dose was indicated. If, at the end of the third minute, anaesthesia was not complete as shown by the absence of the pedal reflex, a further quantity was injected as required.

Duration of anaesthesia was taken as the time elapsing between injection and ability to raise the head and was, therefore, longer than surgical anaesthesia.

The results are detailed in Table I.

The time taken for induction of anaesthesia, and the dose, remained very constant throughout the trials. There is therefore no indication of either increase in resistance or in susceptibility to the anaesthetic. There is some variation in the duration of anaesthesia but this cannot be related to repetition of treatment. This conclusion was confirmed by clinical observation.

No changes were observed in total blood protein, albumin, globulin, uric acid, urea nitrogen, glucose, total cholesterol, cholesterol ester, serum iron, erythrocyte sedimentation rate, red and white cell counts, total red cell and mean corpuscular volume, and liver function (as judged by zinc turbidity, thymol turbidity, thymol flocculation, colloidal gold, serum alkaline phosphatase and serum glutamic oxalacetic and pyruvic transaminase tests) in analyses performed before and after the full course of anaesthesias in each of the five cases.

The E.C.G. of each case, taken at the beginning of the experiments as well as at the end, did not reveal any damage to the heart. The only figure differing significantly after the anaesthesias from that

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TABLE I

Date	Period of Induction	Dose of 5% Neraval mg./Kg.	Period of Amaesthesia	Remarks
Case I: Weight 7 Kg.				
9–7–59	3 min.	50	38 min.	Slight clonic spasms of body.
10–7–59	3 min.	50	31 min.	Quiet induction.
15–7–59	3 min.	50	33 min.	Masticatory movements. Respiration slow and irregular.
16–7–59	2 min.	50	22 min.	Fed before anaesthesia. Violent excitement at induction. Struggling and crying. Respiration very slow and shallow.
17–7–59	2 min.	50	44 min.	Quiet induction. Respiration slow and shallow.
Case II: Weight 6 Kg.				
9–7–59	3 min.	58.3	35 min.	Masticatory movements. Clonic spasms of lips and of body.
10–7–59	3 min.	58.3	30 min.	Clonic spasms of lips and of body.
15–7–59	3 min.	58.3	15 min.	Excitement. Clonic spasms. After brief anaesthesia spasms resumed.
16–7–59	3 min.	58.3	31 min.	Fed before anaesthesia. Masticatory movements as if case had a fit. Clonic spasms. Excessive salivation. Slight spasms of odd muscle groups continued throughout anaesthesia.
17–7–59	4 min.	62.5	32 min.	Fed before anaesthesia. Masticatory movements as if case had a fit. Clonic spasms. Excessive salivation. Slight spasms of odd muscle groups continued throughout anaesthesia.
20–7–59	4 min.	62.5	36 min.	Fed before anaesthesia. Masticatory movements as if case had a fit. Clonic spasms. Excessive salivation. Slight spasms of odd muscle groups continued throughout anaesthesia.
Case III: Weight 8 Kg.				
7–7–59	2 min.	50	18 min.	Small quantity peri-venous. Slight excitement.
8–7–59	2 min.	50	24 min.	Quiet induction.
9–7–59	2 min.	50	38 min.	Quiet induction.
10–7–59	2 min.	50	37 min.	Fed before anaesthesia. Fair excitement, jerking lips, salivation.
15–7–59	2 min.	50	27 min.	Fed before anaesthesia. Quiet induction.
16–7–59	4 min.	62.5	41 min.	Clonic spasms and masticatory movements. Salivation.
Case IV: Weight 7 Kg.				
9–7–57	2 min.	46.4	38 min.	Quiet induction.
10–7–59	2 min.	46.4	60 min.	Slight spasms right foreleg.
15–7–59	2 min.	46.4	44 min.	Fed before anaesthesia. Slight clonic spasms of limbs.
16–7–59	1 min.	39.3	28 min.	Fed before anaesthesia. Slight clonic spasms of limbs.
17–7–59	2 min.	46.4	31 min.	Slight spasm of forelegs.
20–7–59	2 min.	46.4	36 min.	Slight spasm of forelegs.
Case V: Weight 9 Kg.				
8–7–59	4 min.	61	20 min.	Clonic spasms of muscle groups.
9–7–59	4 min.	61	43 min.	Clonic spasms of muscle groups.
10–7–59	4 min.	61	27 min.	Clonic spasms of muscle groups.
15–7–59	6½ min.	77.7	47 min.	Very excited.
16–7–59	3 min.	55.5	42 min.	Masticatory movements. Clonic spasms.
17–7–59	4 min.	58.3	45 min.	Masticatory movements. Clonic spasms.
20–7–59	2 min.	50	52 min.	Masticatory movements. Clonic spasms.

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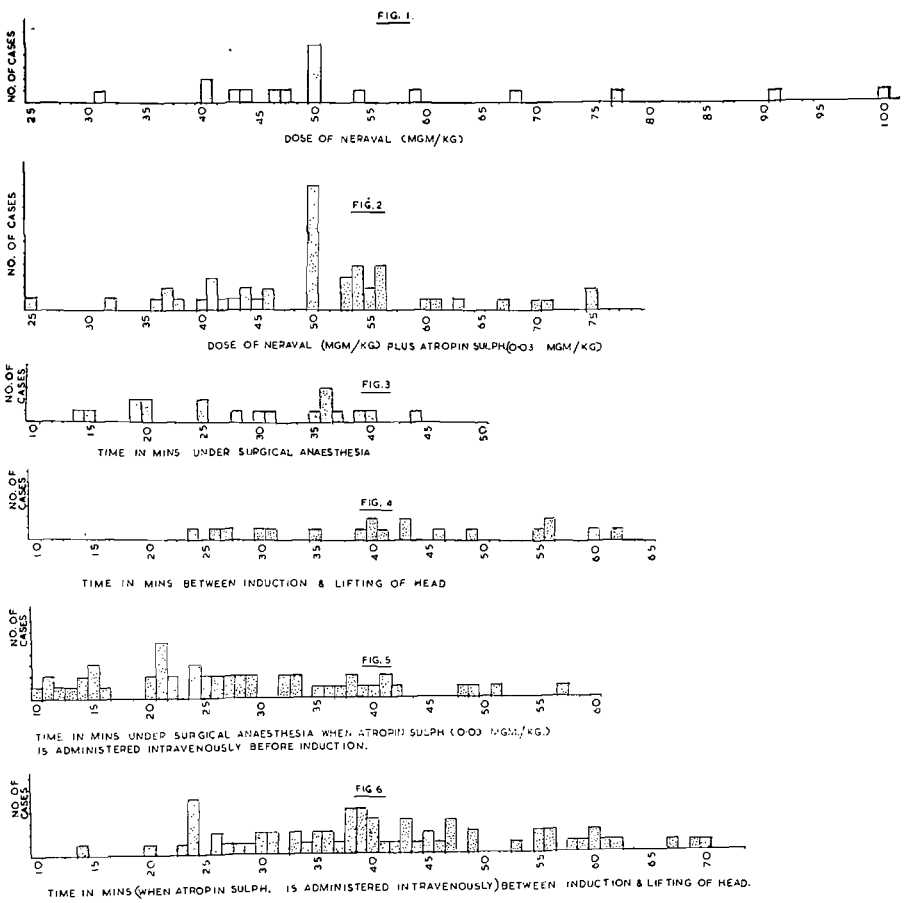
obtained beforehand, is that of the erythrocyte sedimentation rate in case 1. This apparently indicated an intercurrent infection unassociated with the experiment.

Findings in the Surgical Clinic

The following cases were all routine oöphorectomies and were divided into two groups. In Group I (18 cases) methitural sodium only was employed. In Group II (49 cases) methitural sodium was given intravenously preceded by the intravenous injection of atropine sulphate (0.03 mg./Kg.) to counteract certain parasympathetic effects. The dose of anaesthetic without atropine (Fig. 1) was 55.56 (± 15.72) mg./Kg. and with atropine premedication (Fig. 2) it was 50.59 (±5.05) mg./Kg.

The duration of surgical anaesthesia without atropine (Fig. 3) was 28.89 (± 8.93) minutes and, until the head could be lifted, 42.26 (±5.07) minutes (Fig. 4).

The comparable times with atropine premedication were 26.29 (±12.41) minutes (Fig. 5) and 40.82 (±12.84) minutes (Fig. 6).



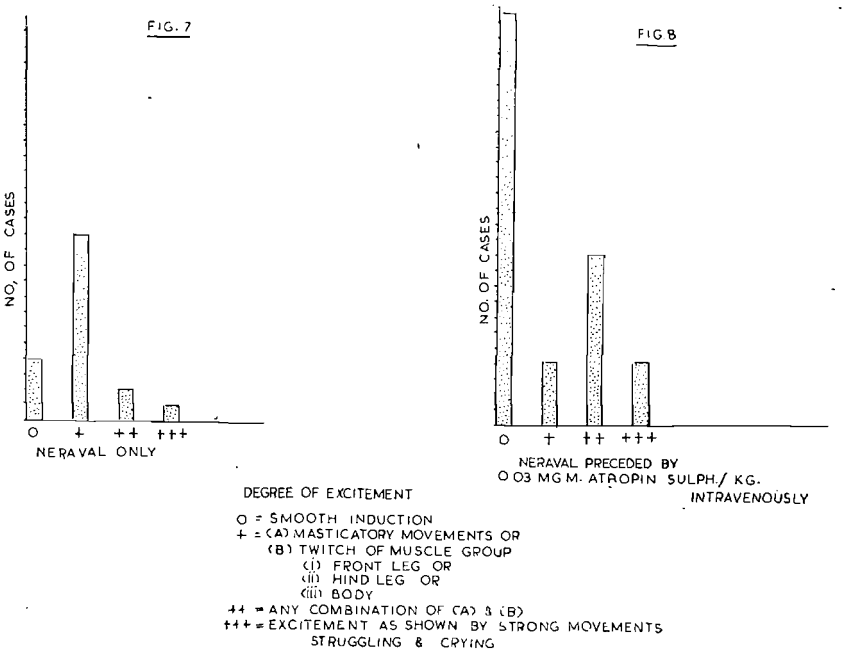
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Side effects

Muscular twitching of one muscle, a group of muscles, a limb or the whole body was frequently seen (cf. Figs. 7 and 8). It was not uncommon for a twitch of a forelimb to persist until the case had entered surgical anaesthesia.

As is the case with other barbiturates, excitement during induction was often shown. Parasympathetic action as shown by salivation was marked in most cases not pretreated with atropine.

The influence of atropine on nervous symptoms may be deduced from the following data (see also Figs. 7 and 8). Atropine had a significant effect in decreasing the percentage of cases showing muscular twitching. Without atropine 20 per cent showed smooth induction and about 18 per cent manifested muscular twitching and masticatory movements, or marked excitement. With atropine 57 per cent of cases were inducted smoothly and 32 per cent showed such signs. This result is significant, as the possibility of obtaining a similar result by chance is between 1 per cent and 0.1 per cent. Atropine thus appeared to suppress the milder forms of nervous reaction.

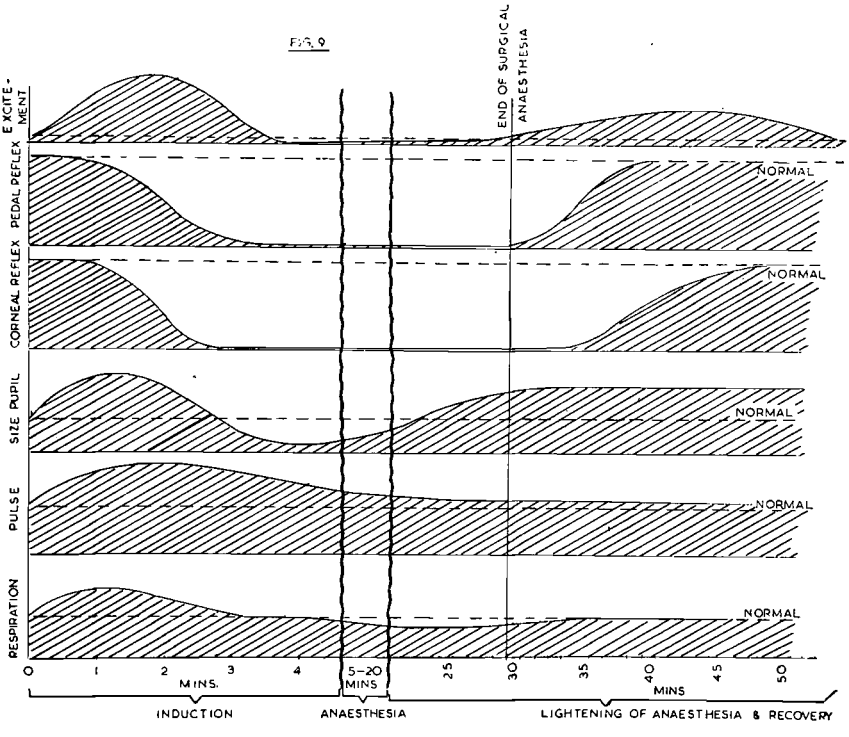


Anaesthetic deaths

Of the 67 cases operated upon by students there were two anaesthetic deaths; both from respiratory failure. The one case had received 25 mg./Kg. and the other 49 mg./Kg. intravenously. No other cause could be found at autopsy.

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In a number of cases the respiration, pulse, size of pupil, corneal reflex, pedal reflex and manifestation of excitement were recorded at intervals of 30 seconds for the first few minutes, and then at longer intervals as indicated. Hence Fig. 9 cannot be taken as correct in minute detail, but it is a fair representation of the general anaesthetic pattern in the average case.



THE GENERAL ANAESTHETIC PATTERN REPRESENTING A TYPICAL CASE OF NERAVAL ANAESTHESIA

Conclusions.

(1) Methitural sodium is comparable to other drugs in this group of short acting barbiturates in that it causes periphlebitis if injected perivenously. There is no choice as to the ease of administration.

(2) In five experimental dogs, anaesthetized a total of 30 times in somewhat more than a week, no increased resistance or susceptibility was demonstrated.

(3) Extensive clinico-pathological examination, before and after the series, failed to indicate damage to the internal organs.

(4) In total of 18 cases, where methitural sodium alone was employed, the mean dose was 55.56 ($\pm$ 15.72) mg./Kg.

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(5) In 49 cases where atropine was used at 0.03 mg./Kg. intravenously immediately before anaesthesia, the dose was 50.59 (± 5.05) mg./Kg. Atropine therefore did not make a material difference to the dose.

(6) The 18 cases remained under surgical anaesthesia for a mean period of 28.89 (± 8.93) minutes and a total of 42.26 (± 5.07) minutes transpired until the head could be raised.

(7) The relevant figures when atropine was used were 26.69 (± 12.41) and 40.83 (± 12.84) minutes—again not materially different from the previous group.

(8) In these two groups the anaesthetic action was not so much shorter than that of other short acting barbiturates as to make a significant difference to the clinician.

In this respect these findings are at variance with those of some authors cited.

(9) There is an unfortunate and high incidence of nervous side effects. These are preventable in a significant number of cases by the use of atropine, yet remain too high. The clinical signs of parasympathetic influence are also much more evident than in cases of other barbiturates in use. This necessitates premedication by atropine.

(10) According to electro-cardiographic investigation, no deleterious effects on the heart could be demonstrated in cases anaesthetized repeatedly.

(11) Two deaths in 67 appears to be too high. As the operators were very inexperienced, no firm conclusions can be drawn in this respect.

Summary

1. Methitural sodium (Neraval sodium Scherag) was tested on five experimental and 67 routine surgical cases.
2. The duration of anaesthesia was found not to be very much shorter than that of other short acting barbiturates.
3. The incidence of nervous manifestations was high at induction.
4. Parasympathetic manifestations (especially salivation) were prominent, but could be suppressed by the use of atropine.
5. No damage to parenchymatous organs could be demonstrated after repeated use. No adverse effect on the heart was detectable by means of electro-cardiographic recording.
6. The duration of surgical anaesthesia was about 30 minutes and the dose about 50 mg./Kg.

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ACKNOWLEDGEMENTS

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To the Director of Veterinary Services for permission to publish this article, to Professor K. van der Walt, of the Department of Medicine of this Faculty for loan of the Electro-cardiograph, to Dr. W. D. Malherbe of the same Department for the clinico-pathological investigation, to Mr. D. de Vos, Chief Technical Officer of the Department of Surgery for customary efficient assistance as well as to other members of the technical staff of this Department. Last, but not least to Messrs. Scherag, (Pty.) Ltd., who very kindly supplied all the methitural sodium (Neraval sodium) used in the trials.

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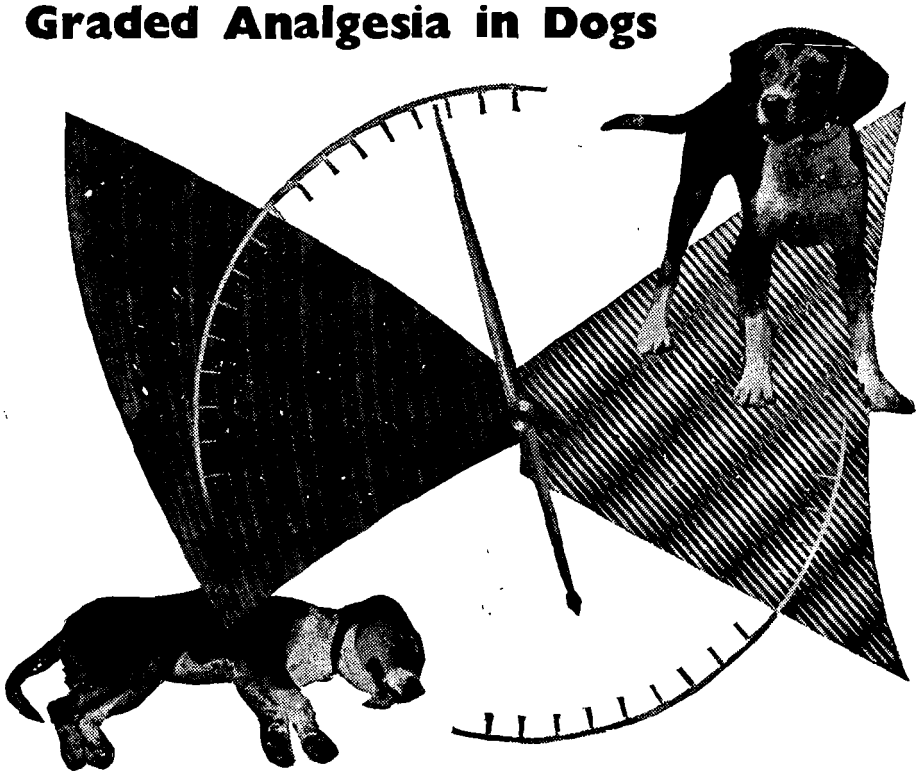
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ANAESTHESIA WITH CONTROLLED RESPIRATION IN THE DOG

K. B. Vetten*

Dr. Vetten explained that for the purpose of experimental heart surgery it was essential that respiration be controlled and consciousness be recovered immediately upon completion of the operative procedure, irrespective of the duration of anaesthesia, which might be from one to six hours.

He then continued:—

Using as a basis J. G. Wright's book on Veterinary Anaesthesia, some dogs were given the recognised veterinary dose, namely, 0.06 g. (1 gr.) Sodium Nembutal per 2¼ Kg. (5 lbs.) body weight, i.e. 1 cc. Nembutal (veterinary) per 5 lbs. body weight. On this it was found possible to divide the levels of anaesthesia into light, medium and deep. (These levels are applicable to Thiopentone Sodium but are passed through so rapidly that they are not seen in the average case.)

Light Anaesthesia (1st level).

Loss of consciousness. The pupil reacts to light, the eyelids remain open and the corneal reflex remains brisk. There is complete relaxation of the jaws and an inability to move the tongue, but a brisk pedal reflex remains. (For this reflex the "web" between the digits is pinched and the limb respons with a definite jerk.) On stroking the malar region a very brisk contraction of the palpebral muscles occurs (palpebral reflex).

Medium Anaesthesia (2nd level).

In this plane the pupil constricts, the lids partially close, the corneal reflex becomes sluggish and the eyeballs start to turn downwards. Both the pedal and palpebral reflexes become sluggish. Respiration is usually slower during this stage often dropping as low as ten per minute.

Deep Anaesthesia (3rd level).

In this plane the corneae have rolled completely out of sight, only the "whites" remaining; on rolling them back the pupils are seen to be the size of a pin's head. Palpebral and pedal reflexes are absent. This depth of anaesthesia is near the danger zone in which the pupils then become dilated and fixed.

In overdose with Nembutal, death is due primarily to respiratory failure, the respirations becoming slower and shallower until they are imperceptible. The heart usually continues to beat for a considerable time (5 minutes).

*Summary of a lecture delivered to the Witwatersrand Branch of the S.A.V.M.A. at the Wanderer's Club on 27th October, 1959, by Dr. K. B. Vetten of the Heart-Lung Unit, Medical School, University of Witwatersrand, Johannesburg.

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The modern tendency in anaesthesia for human subjects is a very light level of anaesthesia combined with a muscle relaxant and control of the respiration. The patient regains consciousness immediately the operation is completed. To apply this to animals, a suitable relaxant had to be found.

The results of our experiments on relaxants have been published in the South African Journal of Medical Science[2], but I would like to stress those which I think you will find most suitable for your own cases. The serum cholinesterase is the important factor in dogs: it ranges between 40–60%[3] (humans 80–95%). Humans develop an apnoea to Scoline when the serum cholinesterase is as low as 60%.

The use of Scoline in dogs was made possible because of its prolonged action; this is not seen in humans as a rule, though this has occurred in persons with low serum cholinesterase.

The reason for this action can be readily seen, when it is considered that muscle paralysis can be produced by drugs causing interference with the normal transmission of nerve impulses at the neuromuscular junction. Acetylcholine released from nerve endings depolarises the motor end plate. This depolarisation excites the muscle fibre and sets off the contraction. Acetylcholine is quickly destroyed by the cholinesterase and the muscle fibre is ready to contract again. Scoline acts by depolarisation and the low cholinesterase content in the blood of dogs probably allows this relaxation to persist.

D-Tubocurarine chloride and Flaxedil produce muscle relaxation by occupying the normal receptor sites of acetylcholine, thus blocking its depolarising action. The effect of these curariform substances is reversed by anticholinesterases, especially Neostigmine (prostigmine).

The experiments showed that the dog seemed relatively sensitive to relaxants in small doses and relatively resistant to barbiturates (Pentothal and Nembutal). The duration of action of the relaxants was longer than after corresponding doses per body weight in humans.

In humans curare is the commonest relaxant used in thoracic surgery, but in dogs respiratory depression lasted several hours, even at a dosage level of $\frac{1}{4}$ mg./Kg., when relaxation occurred after one minute. However, its action can be reversed using atropine/prostigmine. Flaxedil can also be of use (3 mg./Kg.), resulting in apnoea for half an hour, but its onset of action is a little delayed (2–3 minutes.) Its action can also be reversed by means of atropine/prostigmine. The dose of succinylcholine chloride, Scoline (*Anectine*) was found to be 10–15 mg. in the under 20 Kg. weight group (approx. $\frac{1}{2}$ mg. per lb.) and 20 mg. for dogs over this weight, although larger doses could be used with safety. The onset of action was within 30 seconds, and its action lasted 30–45 minutes (average in humans 6–7 minutes). Subsequent doses (smaller than the first) could be given if necessary. There is, however, no antidote to reverse its action and care had to be exercised on dosage given—the larger the dose the longer the apnoea.

Scoline could be used with the lightest level of anaesthesia without deleterious effect on the animal. The dose could be repeated on the first evidence of return of spontaneous respiration before shivering or any palpebral reflex occurred. The fasciculations (small rippling

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movements of the muscles) seen in humans were not so conspicuous in the dog, where movements usually were in the nature of a single jerk. By using Scoline, respiration could be controlled easily and on discontinuing artificial ventilation spontaneous breathing soon became established. There was no drop in blood pressure even after deliberately injecting large doses (150 mg.).

Because of the quick control of respiration, easy restoration of spontaneous respiration and absence of haemodynamic changes, a combination of light Nembutal anaesthesia with Scoline was chosen.

For operations inside the chest, controlled respiration is essential so as to overcome the "mediastinal flap" which occurs with the open pneumothorax. The animal can die rapidly if this is allowed to occur.

There are a few anatomical considerations one should bear in mind. The most important is that the pleura of the mediastinum is extremely thin and frequently shows a small hole connecting the two pleural cavities. Thus an open pneumothorax on one side created by the operation exposes the animal to danger of the development of a tension pneumothorax on the opposite side.

Technique adopted

Premedication

Relatively large doses of atropine (1/50 gr. under 20 Kg.—50 lbs. and 1/100 gr. over 20 Kg.) were required to inhibit excessive salivation. No morphia was used for sedation, owing to respiratory depression caused by it.

The dose of Nembutal was 1 gr. (1 cc.) per 10 lbs. body weight (4½ Kg.). This gave first level anaesthesia when given intravenously. It was followed immediately by Scoline (diluted for accurate dosage, i.e. 10–15 mg. if under 20 Kg. and 20 mg. for over this weight.).

The dog was immediately intubated and connected to Boyle's machine for artificial pulmonary ventilation with O_2 or N_2O and O_2.

This combination of Nembutal and Scoline permitted easy intubation and facilitated control of respiration. The small dose of Nembutal, which sufficed for the operation, allowed the dog to be fully conscious with adequage respiration shortly after completion of the operation.

For very short procedures, Pentothal can be used with Scoline. The dosage of Scoline is as above but the Pentothal must be diluted. For dogs under 50 lbs. use a 1¼% solution and for those over 50 lbs. a 2½% solution; 2–3 cc. is usually sufficient. Give 1 cc. and allow a minute for it to take effect, then add another and, as the animal begins to get drowsy, inject the Scoline. Intubate and connect O_2 or compressed air through a flow-meter.

Intubation in dogs. No laryngoscope is necessary for this procedure. An assistant is required to open the jaws and to pull on the tongue, while the anaesthetist slides his left hand down the throat to feel the epiglottis. By placing a finger over the oesophagus the tube can be directed into the trachea without difficulty. Alternatively, a tongue forceps can be used to pull the epiglottis forward and the tube is directed into the trachea.

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Dogs display a striking absence of laryngospasm and bronchospasm Use no. 10 or larger endotracheal cuffed tubes (flexometallic if possible). Owing to the length and diameter of the dog's trachea the tube has to be placed well down near the carina; if necessary the throat is plugged as well.

Following intubation, the tongue is drawn out to the side for observation of such factors as pallor or cyanosis, an all metal airway is placed alongside the tube and the jaws, etc. are all strapped together.

Pulmonary ventilation with O_2 or N_2O: O_2 was maintained using Boyle's apparatus and Water's cannister. Gas flow was less than 2 litres/min. No additional anaesthetic was required, though extra Scoline (5–10 mg.) was required every $\frac{1}{2}$ to $\frac{3}{4}$ hour. Hyperventilation with apnoea is very easily maintained in dogs.

Flutter valves

These valves, either Stott, Cardiff, etc. are all extremely useful where no carbon dioxide absorber is available or when soda-lime is difficult to obtain. However, when a flutter valve is used, remember the flow rate of the gases required will be in the neighbourhood of nine litres per minute.

This type of anaesthetic can be used for any type of case and is not only applicable to thoracic work. The advantage is complete muscular relaxation for any abdominal procedure or setting of difficult fractures where muscular tension otherwise prevents the fragments easily being set into position. The dog is awake immediately afterwards.

To revert back to thoracic cases, prevention of collapse of the lungs is necessary during and after closure of the chest. As underwater drainage for any length of time is impracticable in animals, special precautions are required to ensure that the *status quo* in the thoracic cavity is restored at the end of the operation.

The drainage tubes, one in each pleural cavity, are graded through the muscles during the closure of the chest wall to act as a valve when the tubes are pulled out. These tubes are connected to a water manometer and the degree of negative pressure measured. They can be attached to underwater drainage bottles placed at a lower level than the animal. Gentle suction can be attached to these tubes if there is considered to be any excess blood in the cavities or an accumulation of air. Only when the negative pressure is static should the tubes be withdrawn and the skin wound closed and sealed off. During withdrawal of the tube, positive pressure is exerted through the endotracheal tube to prevent any leak of air into the pleural cavity. This keeps the lungs inflated.

Post-operative sedation in dogs.

For this purpose Pethidine (2 mg./Kg.), Phenergan (0.75 mg./Kg.) and (optionally) Hydergine (0.02 mg./Kg.) is used. Instead of Hydergine, Largactil can be used.

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Blood Transfusions

It should be remembered that the donor dogs receive an anaesthetic before being bled and that the anaesthetic is therefore present in the blood going into the recipient. Where large amounts of blood are being used, the amount of anaesthetic in it may become an important factor. For this reason the shortacting diluted Pentothal and Scoline technique is extremely useful.

Blood for ordinary transfusions is citrated, whereas that used for the oxygenator pumps is heparinised. No compatibility tests were done and no haemolysis due to this factor was shown, the only cases of haemolysis being due to the pump mechanism.

Post-operative stimulants.

Where a "balanced" anaesthetic has been used, the need for a respiratory stimulant is rarely indicated. Nikethamide (Coramine) may be used, 5–10 mg. at a time.

If the respiratory depression is due to the barbiturate used, Megimide may be given in small doses: large doses will produce convulsions and the animal will require more barbiturate to counteract these.

However, if a curariform relaxant is used and the respiratory depression is due to this, atropine 1/200 gr. followed by small doses of prostigmine (0.25 mg. at a time) should be given. Remember Scoline has no antidote.

During some of the experiments an electro-encephalograph was attached to the dog and a recording of deep anaesthesia was shown following the use of a muscular relaxant in these animals, which may point to some central action. In support of this theory certain experimentalists, both on the continent and elsewhere, have recently shown a tendency not to give the dogs any barbiturate at all but only a muscular relaxant, with immediate intubation followed by nitrous oxide and oxygen anaesthesia. According to these experimentalists, the dogs do not appear to resent this as they are operated on frequently and show no fear of the procedure.

Vervet Monkeys

These usually weigh ± 5 lbs. and a $2\frac{1}{2}\%$ solution of Cyclonal or a $1\frac{1}{4}\%$ solution Pentothal was given intravenously, with the animals still in the cage, 2–3 cc. being sufficient. The monkeys were then removed to the operating theatre and $2\frac{1}{2}$–5 mg. Scoline was given. The action came on in 10–20 seconds and the monkeys could then be bronchoscoped or intubated with ease during the 6–7 minutes apnoea which followed and then inflated with oxygen. The response to Scoline in these monkeys was similar to that seen in human subjects.

Summary

A method of anaesthesia for experimental cardiac research in dogs, which follows the principles of clinical anaesthesia and which could be

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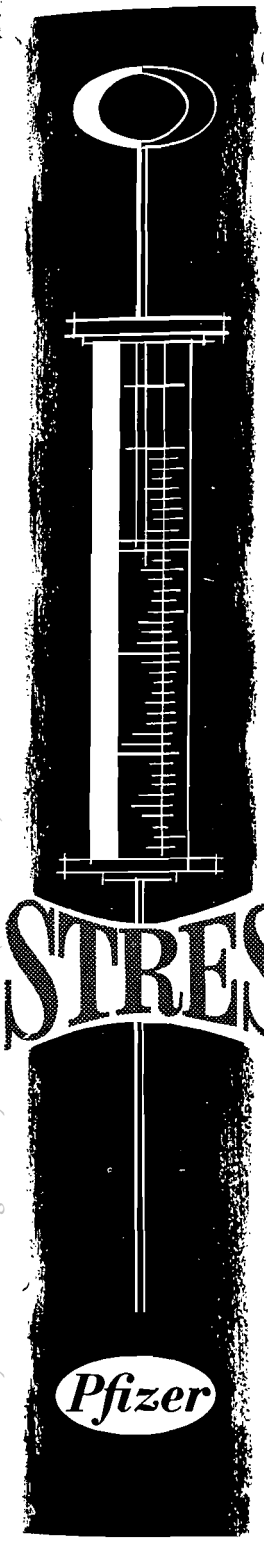
adapted to ordinary veterinary anaesthesia is described. The advantage of the judicious use of relaxants in conjunction with basal anaesthesia is that it avoids excessive port-operative depression.

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CHEMICAL DEPILATION IN SURGICAL PRACTICE

D. H. G. IRWIN†

INTRODUCTION

It is important to be able to remove animal hair quickly and easily. Before most operations, the hair at and around the site must be removed. Sometimes a diagnosis, e.g. in an orthopaedic condition, may depend upon removal of the hair. Sheep shears, curved scissors, open and safety razors and a variety of manual and mechanically operated clippers are of great use in various circumstances. Each has its limitations, however, and it was decided to try chemical depilation as an auxiliary.

THE CHEMICAL DEPILATORS

Barium sulphide has long been used as a depilatory. It may be mixed with starch, zinc oxide and powdered soap. Immediately before use it is mixed to a paste with water and smeared on the part to be depilated. After a few minutes it is scraped off, and the part well washed with warm water[1].

The only other references to chemical depilation found were on the depilation of hog carcasses in the abattoir[2], and the use of intermediary polymers of chlorophene[4]. Neither of these techniques is suitable for clinical work.

A depilation technique employing yellow barium sulphide was recently brought to the writer's attention, by Dr. E. L. French of Melbourne[5]. Yellow barium sulphide is worked to a fine paste in 10% glycerol in water. The hair and skin are thoroughly wetted with water and a detergent. The paste is worked gently over the part to be depilated, and the resulting mass wiped off. The part is next rinsed thoroughly with warm water and then well dried. Finally, a heavy dressing of white vaseline is applied to the bare part. Next day the skin is soft and smooth. If, however, the vaseline dressing is omitted the skin appears dry, cracked and shrivelled. French has used this method of depilation in preparing the sites for skin grafting, the operation being done 48 hours after depilation. His results were highly satisfactory in rats and mice. The method was used routinely by the late Dr. Charles Kellaway in Australia, to depilate dogs, cats and rabbits.

For various reasons it was decided to run trials with a proprietary depilatory agent, having calcium thioglycollate* as the active ingredient, and used for the cosmetic removal of hair in humans. It is presented as a white cream, and is strongly alkaline, having a pH of 11.

TECHNIQUE EMPLOYED IN THESE TRIALS.

Where the hair was thick and long, e.g. on the thorax of an Alsatian dog or around the fetlocks of a wellfeathered horse, much of the

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* "Nair", Carter Products Inc., New York City.

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hair was removed by curved scissors, sheep shears or coarse clippers. This reduces the amount of depilatory cream needed, since the depilation occurs on a quantitative basis. Thereafter, the manufacturers' directions were followed. Where the hair is sparse, as on the ventral abdomen and around the ears of most breeds of dogs and on warm-blooded horses, no primary clipping need be undertaken.

When used on the head, the ears were plugged with cottonwool moistened with a bland oil and the eyes were protected with a few drops of cod liver oil. The directions on the container may be paraphrased briefly as follows: „Shake the bottle. Apply liberally with minimal rubbing. After five minutes remove depilatory from a small area; if the spot bares readily, remove the remainder, if not, wait another two minutes. With a sopping wet cloth wipe the whole area, and rinse with tepid water. Do not rub the area, and avoid soaps. Dry by patting with a towel."

Trials and Observations

(*a*) *The dog.*—Results in the dog have been highly satisfactory. From 1st March to 9th November, 1959, chemical depilation has been used in the majority of cases for operation in the Department of Surgery. No untoward results have been noticed in 526 cases, with the exception of the scrotum. (See contra-indications below).

(*b*) *The cat.*—One individual cat showed skin irritation following chemical depilation when the method was first used. For this reason it was not used again in cats until recently, following an experimental trial on 10 cats selected at random before euthanasia. The cats were anaesthetized and a total of 31 areas were depilated on the ten subjects, i.e. the lateral abdominal wall, ears and scrota. The chemical was allowed to remain on for periods of one to seven minutes. Following the shorter applications, the bristle hairs were not satisfactorily removed, but all applications allowed to remain for four minutes and longer produced satisfactory sites for operation. Irritation was produced on the scrotum only, in two of three of the uncastrated males.

(*c*) *The Horse and Mule.*—In about thirty applications on six Thoroughbreds, three Percherons, seven light crossbred horses and eight mules, irritation was observed in one Thoroughbred only. The period of contact allowed in mules and cold-blooded horses was as long as 15 minutes, but usually it was about six to eight minutes. In Thoroughbreds and light horses chemical contact was kept down to five minutes or less. If the area was not satisfactorily depilated following one application a second was made, leaving the chemical on for a shorter time.

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(*d*) *The Bovine.*—Irritation to the skin followed chemical depilation in three out of four bovines. Within a few hours of removing the hair from sensitive bovines the area became moist as if severe sweating were occurring. Non-pigmented parts were visibly hyperaemic. The exudate dried to form a tough scab, closely attached to the underlying skin, as if the outer layers of the epidermis had become parched and remained firmly attached to the deep epidermis. This film had the brittle feel of old parchment and could be pulled off in strips after 10–20 days. The surface thus exposed bore hairs 1–2 mm in length, and was tender. Pigmented and non-pigmented areas were equally affected as seen macroscopically.

In a trial on a calf, the cream was removed from one area with the usual moist warm swab, and from another with a swab dipped in tepid 5% acetic acid, followed by the usual water sponging. The differently treated areas showed the same degree of exudate and inflammation macroscopically.

To determine the microscopic changes skin biopsy was carried out in a similar manner in all four cases, using local anaesthesia. In the first animal, a castrated Friesian calf, biopsy (Biopsy 1) was done immediately after depilation. As no macroscopic changes were noticed in a three week observation period following depilation, no further samples were collected from this animal. On the second animal, a Friesian heifer, three biopsies were done, one (A) immediately after depilation, another (B) 48 hours later, and a third (C) ten days from depilation.

Professor K. C. A. Schulz, Dept. of Pathology, Faculty of Veterinary Science, Onderstepoort, kindly examined the specimens, and reported as follows:—

Biopsy 1. "Local tendency to form pustules, and disintegretion of epithelial layer. Capillaries in corium markedly engorged, oedema, perivascular lymphocytic cell infiltrations. Swelling and proliferation of endothelial cells and a focal increase of eosinophiles. Some hair sheaths infiltrated with lymphocytes".

Biopsy A. "No tendency to form pustules. Epithelium intact. Corium relatively more oedematous. Perivascular infiltration less marked, but eosinophiles appear to predominate."

Biopsy B. "Similar to A, but oedema apparently more pronounced, capillaries less obvious and necrobiotic changes in infiltrated cells. More eosinophiles present."

Biopsy C. "Similar to B, but changes more advanced. Crust formation. Distinct thickening of epithelial layer (hyperplasia). Increase of fibroblasts in oedematous areas. Perivascular infiltration more pronounced. Some hair follicles are involved. Eosinophiles still fairly frequent."

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It appears, therefore, that even in the absence of undesirable macroscopic changes following chemical depilation, readily recognisable untoward microscopic changes occur almost immediately.

(*e*) *Sheep.*—The use of calcium thioglycollate in preparing the operative site in Merino sheep is highly satisfactory. Areas for doing rumenotomy or caesarian section, 12" × 8", are quickly rendered smooth and hairless. The wool is clipped down to a few mm. length. The depilatory cream is spread over the area in a minute. Within five or six minutes the wool may be sponged off with tepid water and a soft cloth. Should the operator scrape the area, with the thumb nail for example, a red streak will appear. The paper thin skin of Merino sheep is, moreover, sensitive to both energetic rubbing and too warm water, either of which will cause an obvious oedema the following day. The alternative to chemical depilation in preparing the operative site for aseptic surgery in woolled sheep is the use of plastic sheeting adherent to the sheep's body as the laparotomy cloth. Here the sheeting and skin are incised simultaneously, and then one proceeds by applying the remaining drapes in the conventional manner.

The Cost of Chemical Depilation.

The contents of one 4 oz. jar is sufficient to depilate the ventral abdomen of 10 small dogs (± 10 Kg.) or six large ones (± 30 Kg.). Therefore the cost of the agent for depilating this site is about 6d. in small dogs and 10d. in large ones. The term "ventral abdomen" denotes an area bounded by the costal margin cranially, the hind skin flanks laterally, and the pubis caudally.

The time and labour involved are less than that required for clipping and shaving. Replacement or sharpening of clipper blades used on animal hair coats which are often dirty and dusty, is reduced.

Conclusions

(*a*) *Indications.*—This chemical depilatory agent appears to be of use to the veterinary clinician for use on dogs, cats, equines and sheep. It has been found especially useful on the ventral abdomen of the dog with regard to time and labour saved. Parts difficult to shave or clip—around the ears and on the feet and lower legs of small animals, and the lower limbs of restive horses—may be depilated chemically quite conveniently.

(*b*) *Contra-indications.*—For use on humans, the manufacturers advise trying a little "Nair" on a small spot. Should this depilated spot be free from irritation 24 hours later, the cream may be used on larger areas. This procedure should be followed in Thoroughbreds and possibly cats but is

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apparently not necessary in other equines, dogs or sheep. Contact with the scrota of dogs and cats should be avoided. The skin of cattle suffers chemical burn after depilation with the calcium thioglycollate preparation used in these trials. It should not be used on abraded or otherwise damaged skins.

THE ACTION OF THE DEPILATORY AGENT UPON HAIR AND WOOL.

Samples of hair were taken from one horse (mane) one Friesland cow (tail and body hair) one Charollais calf (body hair), one Bulldog, one Alsatian and one Collie (body hair), and of wool from a Merino sheep. These samples were subjected to the action of the calcium thioglycollate depilatory cream, and the chemical process halted by washing in warm water. Mr. Dreyer, of the Onderstepoort Veterinary Research Institute, kindly examined the processed specimens and his report is published separately.

SUMMARY

A depilatory cream containing calcium thioglycollate was found to be useful in dogs, cats, horses and sheep, but contra-indicated in cattle.

ACKNOWLEDGEMENTS

The author is indebted to Prof. K. C. A. Schulz for the histopathology report, and to Mr. de Bruyn for the photography. Thanks are due to Professor C. F. B. Hofmeyr for his interest and suggestions, and to Messrs. D. de Vos and E. Erasmus who collaborated with the trials. Messrs Hill & Murray, Johannesburg, kindly donated the "Nair" for these trials. The Director of Veterinary Services kindly allowed publication of this paper.

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	CONTROL ANIMALS		TREATED ANIMALS	
Months after Treatment	*Average Wt. (lbs)*	*Average Liver Vit. B12 (ug/gm)*	*Average Wt. (lbs)*	*Average Liver Vit. B12 (ug/gm)*
0	283	0.07	283	0.07
2	277	—*	279	—*
4	305	0.29	334	0.53
6	441	0.25	475	0.47

* *Liver Vitamin B12 not estimated.*

REFERENCE + *Skerman, K. D., Sutherland, A. K., O'Halloran, M. W., Bourke, J. M., and Munday, B. L.,* (1959), *Am. J. Vet. Res., XX,* 977.



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THE EFFECT OF A CALCIUM THIOGLYCOLLATE PREPARATION* ON HAIR FIBRE

J. H. Dreyer**

Introduction

In his study on chemical depilation Irwin[1] employed a proprietary preparation ("Nair") containing calcium thioglycollate as active ingredient. This substance attacks the keratin of hair fibre and ultimately breaks it down to amino-acids. As calcium thioglycollate is extremely water soluble, its action can be halted immediately by washing in water at a temperature of about 50°C.

Method

It was decided to study the morphological effect of "Nair" on the different elements constituting the animal hair fibre. The following samples of hair were tested: Bulldog (body), Alsatian (tail), Collie (body), Merino wool, Charollais (body), Friesland (body and tail) and horse (mane). All the samples were divided into eight sub-samples and these were treated separately with "Nair" for periods ranging from one to seven minutes at intervals of one minute. Sample number eight was used as a time-test sample and the immersion time was regulated according to the resistance of the hair to the action of the depilatory. The immersion periods ranged from ten to forty minutes and as soon as the fibres lost their rigidity and became soft and pliable they were rinsed and mounted in a mixture of distilled water and glycerine. A drop of a saturated solution of Metanil Yellow was added as a stain. This method of mounting was preferred as the disadvantage of using ordinary mounting media is that their viscosity is too high to allow the fibres to fall apart into their various elements when pressure is applied to the cover-slip. Of the other samples, fibre bundles were only partially immersed to compare the sound half with the affected half.

Slide preparations were made of the samples treated for the stated periods, including those treated for longer periods, and these were examined under the light microscope. Prior to examination pressure was applied to the cover-slip so that the fibres disintegrated into their various elements. These slides were also ringed with a heated mixture of beeswax and resin to inhibit evaporation of the water and facilitate handling. Samples left in "Nair" overnight were completely destroyed and no trace was left.

*"Nair" Carter Products Inc., New York, N.Y.

**Section of Pathology, Veterinary Research Laboratories, Onderstepoort.

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Discussion and Results

The effect of "Nair" on the different layers of the hair fibre:

The cuticle

This outer layer of the animal fibre consists of layers of cells forming the scales on the outer surface. Each scale is overlapped by its immediate neighbour lower down the fibre. In thick cuticles more than half a scale is overlapped whereas in thin cuticles less than half of each scale is covered. Overlapping also occurs in a lateral direction between neighbouring scales. It appears that a scale consists of more than one layer of cells. The possibility of a thin outer membrane—the "epicuticle" also exists, as a continuous membrane over the entire fibre cuticle, but this has not been demonstrated beyond doubt.[2]

From microscopical observation it appears that the depilatory acts on any defective spot in the cuticle and separates it from the underlying cortex; initially creating the effect of blisters on the cortex. The cuticle then disintegrates into fragments. The ridges of the scales can still be observed on these fragments (Fig. 1). Where the cuticle had not been separated from the cortex, the effect of the depilatory on the cortex could be observed through the cuticle.

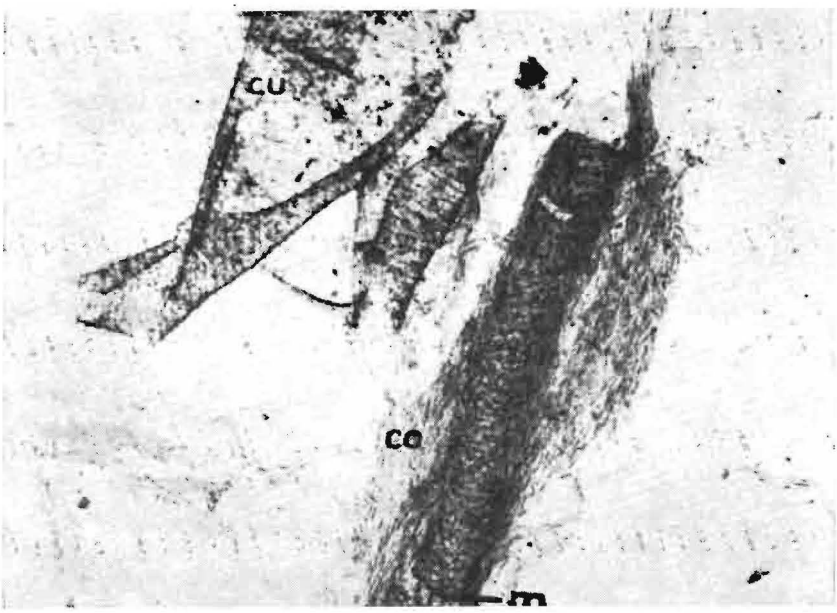


Fig. 1.—Friesland (tail) hair after 30 minutes' immersion. Cuticular sheath with scale boundaries showing separation of cortical cells. Medulla still intact. co = cortical cells. cu = cuticular sheath. m = medulla.

The cortex

The next layer of the hair fibre consists of long narrow cells known as fusiform cells. Fibres treated with trypsin show the boundaries of the cortical cells clearly. The surface of each cell is faceted where it

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abuts on an adjoining cell. These faceted surfaces are not flat but gathered into longitudinal folds which are formed by the fibrils and fit into the folds of the adjoining cell. There are indications that the fibrils of the cortical cells are divided further into what may be termed fibrillae and that still finer divisions may be present.[2].

As already stated it appears that "Nair" attacks the cortex through points of least resistance in the cuticle. The cortical cells are separated and if the process of destruction is halted immediately the cells can be observed in various stages of disintegration (Fig. 2 & 3). The speed with which the cells are broken down depends on the type and thickness of the fibre and also the thickness of the cuticle. Greater resistance to the action of the depilatory is encountered in ornamental hairs (mane and tail) of animals than in wool, or the soft coat of the Collie which is rapidly broken down.

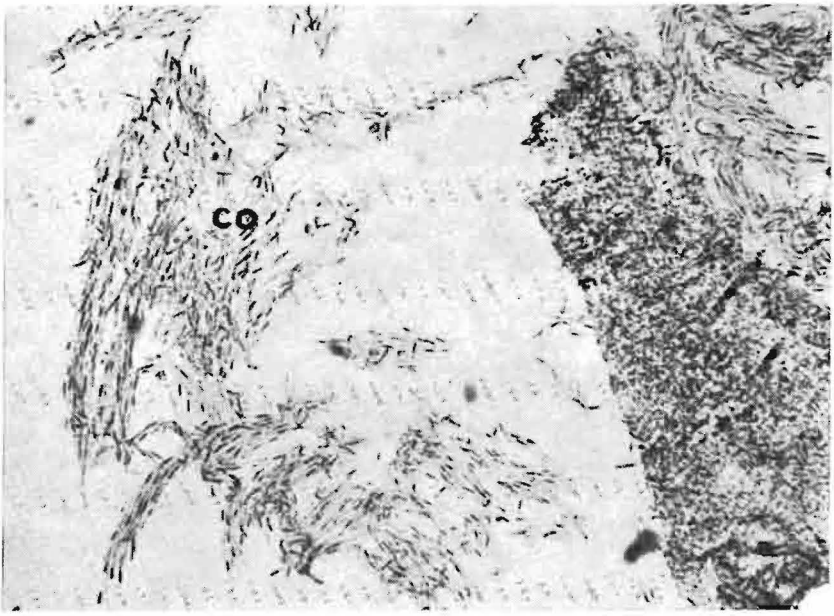


Fig. 2.—Horse hair from mane after 30 minute's immersion. = co cortical cells with pigment granules.

The medulla

The innermost layer of the fibre consists of cells which in some fibres are much larger than the cortical cells and more frail than the other cells of the fibre. They are mostly found in primary fibres and are practically absent in secondary fibres such as Merino wool. Various types of medullae are distinguished e.g. the unbroken medulla, the broken medulla, the fragmental, the lattice and the ladder types.[2]

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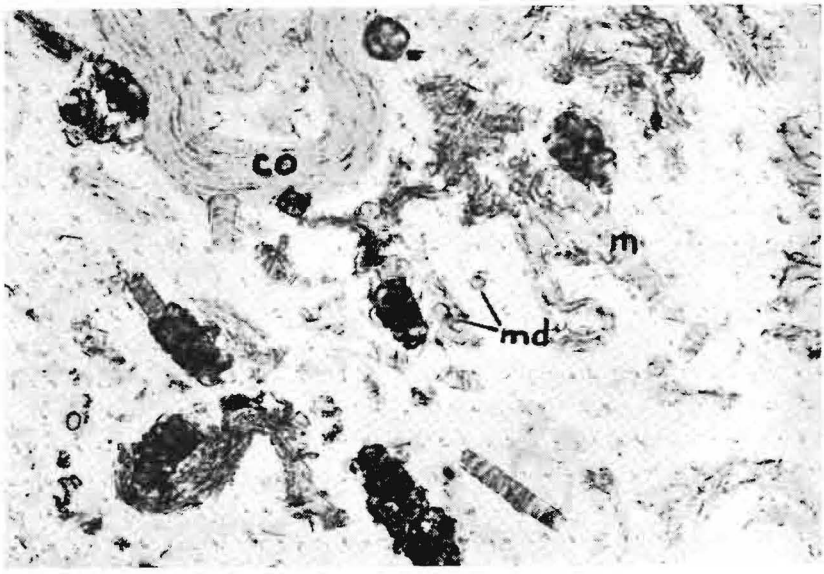


Fig. 3.—Collie (body) hair after 15 minutes' immersion. co = cortical cells. m = medulla. md = medullary discs.

Where fibres are treated with "Nair" and mounted in distilled water the medullary discs can be demonstrated as with treatment in caustic potash (Figs. 3 and 5). In some preparations the medulla was still intact while the cortical cells had been separated from it (Figs. 1 and 4).

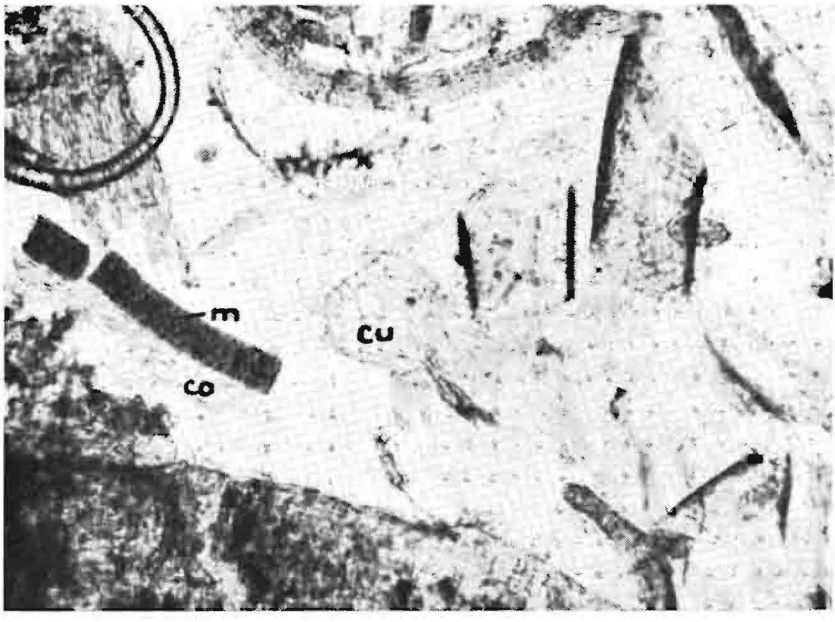


Fig. 4.—Bull-dog (body) hair after 40 minutes' immersion. cu = cuticular sheath. co = cortical cells. m = medulla.

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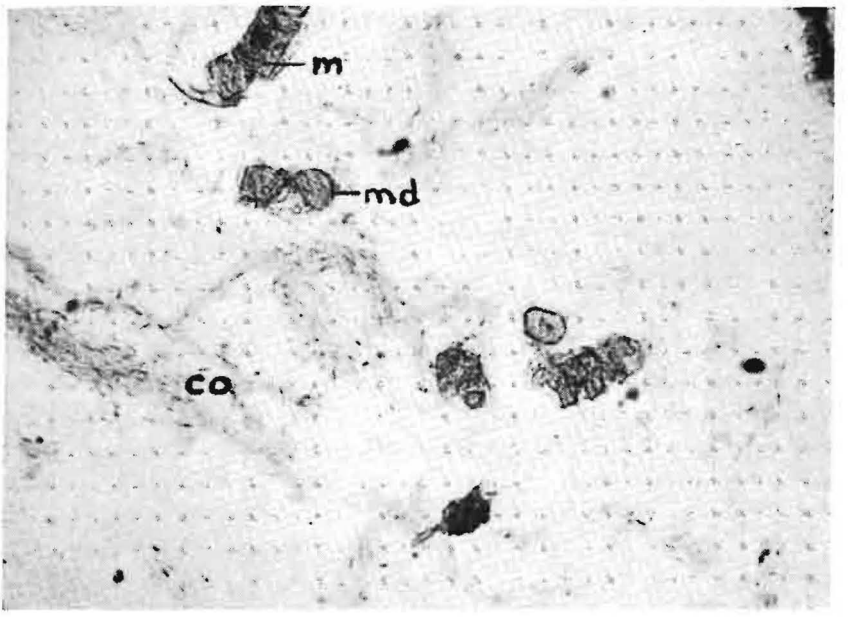


Fig. 5.—Alsatian (tail) hair after 15 minutes immersion. co = cortical cells. m = medulla. md = medullary discs.

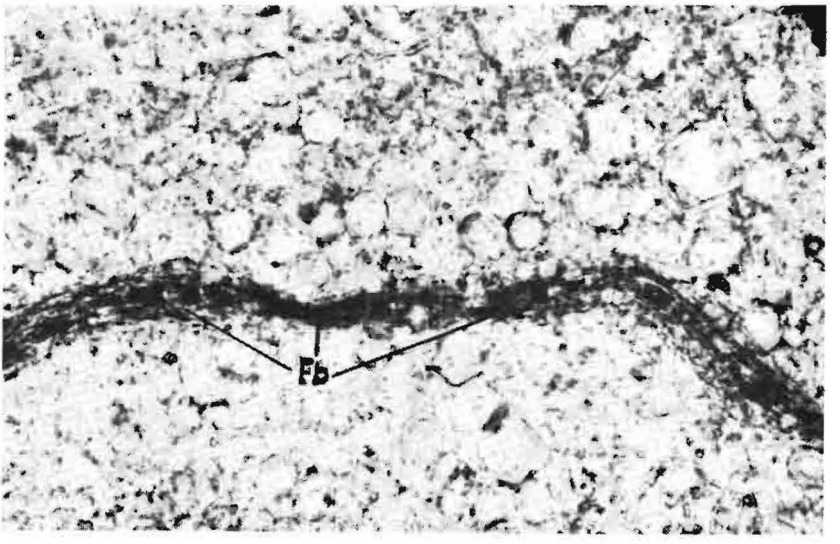


Fig. 6.—Collie (body) hair. Fibre immersed in Calcium thioglycollate for 24 hours. Extreme degeneration of fibre. Photograph taken in *situ*.

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Table I.—The digestion of the cell layers of animal fibre by calcium thioglycollate.

Immersion time in minutes	Bulldog (body)	Alsatian (tail)	Collie (body)	Merino (wool)	Charollais (body)	Friesland (body)	Friesland (tail)	Horse (mane)
1	0	0	++	++	0	0	0	0
2	0	+	+++	++	0	+	0	+
3	0	+++	+++	++*	+	+	0	+
4	+	+++	+++*	++*	+	++	0	+
5	+	+++	+++*	++*	+	+++	0	++
6	++	+++	+++*	++*	+	+++	0	++
7	++	+++	+++*	++*	+	+++	0	++

0 = no appreciable effect; + = cuticle only affected (blistering effect); + + = cuticle and cortex affected—separation of cortical cells; + + + = cuticle, cortex, and medulla affected—medullary discs sometimes apparent; * = fibre completely separated into elements.

The samples used in the time-test experiment were immersed in "Nair" for periods ranging from ten minutes (wool) to 40 minutes (Bulldog body hair). During this interval the fibres were checked occasionally. When it was considered that maximum pliability had been obtained, the fibres were removed from the solution and dissected on a slide in distilled water. In this condition of pliability the fibres are extremely soft and on being removed from the depilatory have a slimy appearance and curl up on the points of the forceps used for handling. When pressure is applied to the cover-slip the fibres readily disintegrate into their various elements. The illustrations (Figs. 1–5) cover these prolonged periods of immersion. Table II shows the effect on the various components of the fibre.

Table II.—The effect of prolonged exposure to calcium thioglycollate.

Immersion time in minutes	Bulldog (body)	Alsatian (tail)	Collie (body)	Merino (wool)	Charollais (body)	Friesland (body)	Friesland (tail)	Horse (mane)
10				++*				
15		++§						
20			+++*		+++*	+++*		
30							++§	+++*
40	+++§							

\+ = Fragmentation of cuticle with boundary markings of scales still visible.
++ = Fragmentation of cuticle and separation of cortical cells.
+++ = Fragmentation of cuticle and medulla, separation of cortical cells.
§ = Medullary discs apparent.
* = Complete separation of fibre into components.

Hair fibres from the Collie (selected on account of the dark pigment) were placed on a slide covered with a film of "Nair" and overlain with a cover-slip. The process was checked at intervals under the microscope. Initially the fibres showed twisting and stretching but retained their form. As time progressed the cells in the cortical layer gave the impression of being scrambled as though the so-called intercellular cement had been dissolved. The illustration (Fig. 6) shows the fibre in a stage of breaking down after it had been immersed in "Nair" for twenty four hours. A normal fibre (Fig. 7) from the same dog is shown.

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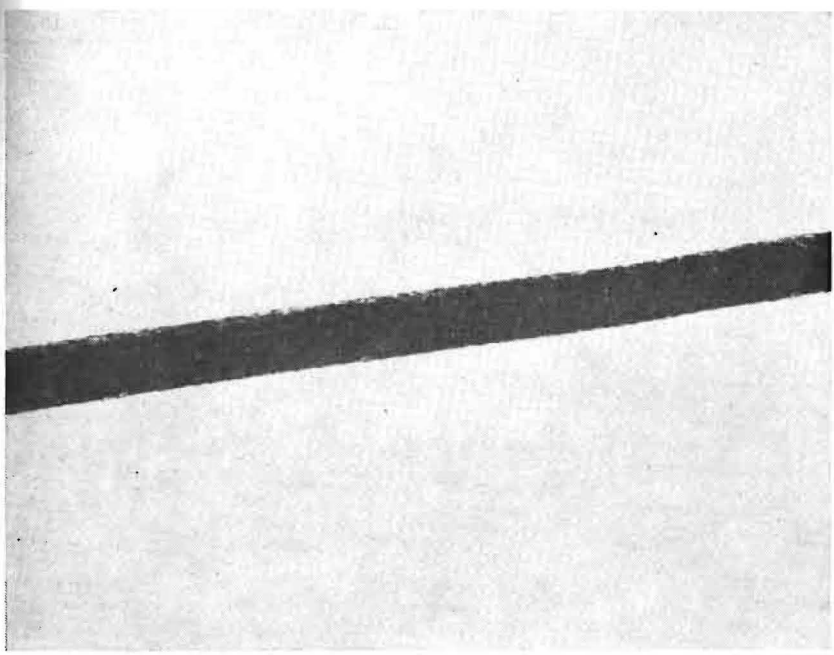


Fig. 7.—A normal hair fibre from a Collie.

Summary

Calcium thioglycollate disintegrates hair and wool fibres by dissolving the sclero-proteins. The so-called intercellular cement is attacked first, with the result that fibres in the initial stages of break-down can be separated into their constituent elements by light pressure. In the early stages the cuticle blisters before separation into fragments, which still show the boundary markings of the scales. Even when the cuticle is still intact the effect of the depilatory on the cortical cells can be observed through it.

When affected hairs mounted in water are subjected to pressure under a cover-slip the cortical cells tend to divide and the medulla disintegrates or breaks into medullary discs. Hair from the body of a bulldog, the tail brush of a bovine and mane of a horse were the most resistant. Wool and the hair fibres from a Collie were broken down very rapidly.

Acknowledgment

The author is indebted to Prof. R. Clark, Prof. H. P. A. de Boom and Dr. R. C. Tustin for helpful criticism and review of the script and to Mr. G. Swartz for technical assistance.

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2. Wildman, A. B. (1954): "The Microscopy of Animal Textile Fibres". Published by Wool Industries Research Association, Torridon, Headingley, Leeds.

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New Books

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BOOK REVIEW

AIDS TO BIOCHEMISTREY

S. P. Datta and J. H. Ottaway

London: Bailliere, Tindall and Cox, 1960, Fifth Edition, pp. 266. Price 15/-

This pocket format volume in the *Students' Aids Series* gives, in condensed form, almost complete coverage of the science of biochemistry at the undergraduate level. The material, entirely rewritten and up to date, is based on the course in biochemistry for pre-clinical medical students at University College, London. After going through 16 of the 18 chapters of the book, the reviewer is satisfied that the authors have succeeded "to make it useful also to more advanced students of medicine . . . who need to refresh their biochemical knowledge. It will also, we believe, be of use to the large number of students of the biological sciences who require a short text in biochemistry to provide background information to their main work."

A description of the chemical properties of the main groups of biological materials, carbohydrates, fats, proteins, nucleic acids and pigments, is given in chapters 2 to 6. The material of chapters 1 and 7 on hydrogen ion concentration and enzymes, respectively, is fundamental to a proper understanding of the processes of metabolism, which are dealt with in chapters 8 to 12. Blood, excreta, biochemical variants and hormones come in for attention in chapters 13 to 16, while the last two chapters are devoted to nutrition.

The treatment of well established facts is concise, without sacrifice of clarity. The important facts are driven home in a nutshell, as it were. Most modern advances in the subject are touched on briefly in a manner which has the effect of whetting the appetite for more.

From the point of view of the veterinarian there is one serious omission, viz. ruminant biochemistry. However, this is to be expected in a book intended for medical students. Nevertheless, veterinarians wishing to "brush up" the biochemistry they learned in their second year of training, without having to wade through the 800 odd pages of a regular text book, are advised to invest in a copy of "Aids to Biochemistry".

There are hardly any misprints or mistakes in the text. One of the few spotted is on page 94 where it is stated that the "synthesis of the porphyrin ring from small-molecule precursors is dealt with in Chapter XII". In reality this is done in Chapter XI, page 159.

J.G.L.

DISEASES OF SWINE

Edited by H. W. Dunne, pp. xiii — 716. Ames, Iowa. Iowa State College Press. $12.50.

The forty-eight contributors to this volume are all recognised authorities in their particular fields. The subject matter is divided

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into the following eight sections: Anatomy and Physiology (three chapters); Viral diseases (eleven chapters); Bacterial and Mycotic infections (12 chapters); Parasitic infections (3 chapters); Toxaemias and Poisonings (six chapters); Miscellaneous Diseases (nine chapters); Surgery (4 chapters); Nutrition, Feeds and Management (five chapters).

The wide range of information provided reflects much of the greatly increased interest in swine management, nutrition and disease control which has become evident during the past decade. Newer information in regard to conditions such as virus pneumonia, parakeratosis, transmissible gastroenteritis, salt poisoning, hypoglycaemia of baby pigs and oedema disease is well presented. There are chapters on feeds and feeding, nutritional deficiencies and the control and elimination of swine diseases through repopulation with disease-free stock. Protozoal diseases are dealt with in the section on parasitic infections. The surgery section includes information about anaesthesia, the handling and restraint of pigs and bleeding from the anterior vena cava.

As minor criticisms, reference may be made to the omission of information regarding spirochaetosis, necrobacillosis, prolapsus ani, and newer concepts regarding pathogenic haemolytic *E. coli* serotypes. The bibliography, although extensive, requires to be brought up to date and the index could be rendered more useful by some amplification. It is hoped that a second edition will not be long delayed.

The book is well printed and lavishly illustrated and is undoubtedly the best text in English on swine disease available today. It may be confidently recommended to practitioners and others who require accurate and up-to-date information concerning swine husbandry and disease.

R.K.L.

PUBLIC HEALTH IN SOUTH AFRICA

by E. H. Cluver

6th Edition, 1960, C.N.A., South Africa, 357 pp. and index—85 illustrations and 32 tables. Price 42/-

This is the long-awaited completely rewritten edition of a book which has become a standard textbook to those engaged in public health work in South Africa, and it remains a "must" for those interested or engaged in any phase of this sphere. It is extremely well illustrated and up-to-date, and is an excellent reference on all public health matters covering as it does the whole field from Atmosphere to Yellow Fever in a most concise and practical manner. Veterinarians will find the chapters dealing with water, food, food hazards, decomposition and poisoning, meat inspection, abattoirs, milk and rabies most useful and informative.

Relevant legislation is freely quoted; unfortunately some of the newer amendments to the Regulations made under the Food and Drugs Act are not included, e.g. the removal of boric acid from the list of approved preservations of sausages.

L.W. v.d. H.

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THE FIFTY-FIFTH ANNUAL GENERAL MEETING AND CONGRESS HELD AT ONDERSTEPOORT BETWEEN AUGUST 29TH AND SEPTEMBER 2ND, 1960.

A REVIEW

The Congress was opened by Dr. S. Meiring Naudé, the very distinguished Chief of the Council for Scientific and Industrial Research. A copy of his address appears in this issue.

Over one hundred and seventy members attended the Congress during the four days it was in session.

The President very aptly described the Congress as "one of the most successful scientific meetings we have yet had". Many members in confirming this view expressed their appreciation to the Editorial Committee for publishing the papers in the Pre-Congress number of the Journal.

Some distinguished persons including Dr. Maule Clark, Acting Secretary for Health, Dr. van den Berghe from I.R.S.A.C., Belgian Congo, attended, as well as a number of the older members, Drs. P. R. Viljoen, W. H. Chase, G. Martinaglia, A. C. Kirkpatrick, P. J. Fourie, J. R. Scheuber, and Dr. Alexander, paid tribute to them when welcoming members and guests to the Congress.

1. *Features of the Congress.*

The amendment of Articles 44, 45 and 46 of the Constitution was discussed at a Special General Meeting, but as a quorum could not be obtained in terms of the New Constitution, it was decided to call another Special Meeting on 30th September, 1960, after the required notice had been given.

The Special General Meeting was duly held on September 30th and authority given for the amendment of these Articles of the Constitution.

Publication of the Constitution.

The Constitution has now been prepared in both languages and Council at its next meeting on 27th January, 1961, will be asked to authorise its publication.

2. *Trade Exhibit.*

The exhibiting firms complained of an apparent lack of business, i.e. an apparent lack of opportunity for Congress members to view the exhibits and discuss details of the items exhibited.

As the result of the activities of the Trade Exhibitors sponsored by Mr. Goddin and Mr. Holmshaw of Maybaker, an amount of nearly £18 was collected for the Benevolent Fund from members and others who exchanged donations for a pair of scissors. This was in addition to the generous donation made to the Benevolent Fund for the stands occupied for exhibition purposes.

A short report on the items exhibited appears as an appendix to this review.

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3. *The 55th Annual General Meeting.*

(i) Draft Agreement Contracts.—A draft Agreement form for use by Veterinarians who wish to enter into contracts with Co-operative Societies was approved and is now available, in both languages, from the Hon. Secretary.

(ii) Finance.—A debit balance was registered during the year, which was largely due to loss of revenue in the publication of the Journal of greater size—the Pre-Congress number.

A joint meeting of the Finance and Editorial Committee was instructed to examine the problem and report to Council.

(iii) Advertisements by Drug Houses, directly to farmers, and stock owners.

This matter was discussed at great length and it was felt that the firms selling proprietary medicines were competing unfairly with Private Practitioners.

A resolution was taken on the subject.

(iv) Formation of an A.I. and Reproduction Group.

The Annual General Meeting approved the formation of this Group.

A resolution was adopted in this regard.

(v) Employment of a Full-time Secretary.

It was agreed to leave this matter in abeyance until the end of the financial year.

RESOLUTIONS—ANNUAL GENERAL MEETING—2ND SEPTEMBER, 1960

1. That this 55th Annual Congress of the South African Veterinary Medical Association instructs Council to hold the 56th Congress of the Association away from Onderstepoort, at a site and time to be decided upon after consideration of representations from the various branches.

   Proposer: I. S. McFarlane.
   Seconder: I. van Schalkwyk.

2. This 55th Congress resolves that Council be instructed to take such steps as are found to be necessary to prevent organisations, companies and other bodies from using the word "veterinary" in the names of their organisations.

   Proposer: S. van Heerden.
   Seconder: K. van der Walt.

3. This Congress approves in principle the formation of a group within the South African Veterinary Medical Association entrusted with coordination of all aspects of reproduction of domestic animals.

   Proposer: R. A. Alexander.
   Seconder: B. C. Jansen.

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4. Hierdie 55ste Jaarkongres van die Suid-Afrikaanse Veterinêr-Mediese Vereniging neem met groot teleurstelling kennis van 'n mededeling dat in stryd met die standpunt van die Universiteit van Stellenbosch, die Professoraat in Veeartsenykunde aan bogenoemde Universiteit na 'n Senior Lektoraat gedegradeer is. Die Kongres is van mening dat die bes-gekwalifiseerde leerkragte nie vir so'n gedegradeerde pos gevind sal word nie, en vereenselwig homself dus heelhartiglik met die standpunt van die Universiteit van Stellenbosch.

Voorgestel: Professor O. T. de Villiers.
Gesekondeer: Dr. P. P. Hugo.

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MINUTES OF THE MEETINGS OF COUNCIL HELD BETWEEN 4th MAY, 1959 AND 30th SEPTEMBER, 1960

A summary of the proceedings of the meetings of Council held during the Congress years ending respectively on 27th August, 1959 (54th Annual General Meeting), 2nd September, 1960 (55th Annual General Meeting) and September, 1961 (56th Annual General Meeting) is given below.

Owing to pressure of work entailed in the production of the Pre-Congress number of the Journal (March, 1960), and through other causes, the publication of extracts of Council Meetings held subsequent to 4th May, 1959, could not be undertaken with the usual regularity.

CONGRESS YEAR ENDING 27th AUGUST, 1959

COUNCIL MEETING ON 27TH JUNE, 1959.

All members were present including Dr. Graf (by invitation). Apologies from Drs. P. J. du Toit, H. H. Curson and M. W. Henning.

The following matters were discussed:

1. *Book Fund.*—It was decided to discontinue this activity as it was no longer a payable proposition. All assets should be consolidated and the project terminated.
2. *Single payment for Life-Membership.*—It was agreed that advice be obtained from an actuary as to the single amount payable for life membership, based on a subscription rate of £5 per annum.
3. *Draft Agreement for Employment of Veterinarians* by Co-operatives and other non-Veterinary Organisations. Legal advice was to be sought on a standardised form of agreement.
4. *Financial Aid by Department of Education, Arts and Science* in the publication of the Journal.—An application to be submitted.
5. *Amendment to Veterinary Act.*—Agreed to proceed with the matter as energetically as possible.
6. *Activities of the Disciplinary Committee.*—Several cases cited by the Chairman of the Committee.
7. *Function to Welcome New Graduates.*—To be investigated and arranged.
8. *Publicity at next Congress.*—Radio and Press to be used as a means of giving publicity to the Congress.
9. *New Members.*—Six new members accepted for recommendation to Annual General Meeting.
10. *Finance:*
    (*a*) President's Badge of Office.—Clasps with names of past presidents to be arranged.
    (*b*) Investments—Treasurer instructed to proceed with the investment of £600 from General Fund in Permanent Building Society shares.

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COUNCIL MEETING ON 27TH JULY, 1959.

All members present except Drs. Alexander, P. J. du Toit, R. du Toit, C. F. B. Hofmeyr and A. F. Tarr.

The following matters were discussed:

(*a*) *Employment of Veterinarians by S.P.C.A., Johannesburg.*—The President and two Veterinarians from the Witwatersrand Branch to negotiate further with the Society.

(*b*) *General:*

1. Veterinary Members of Agricultural Faculties—General Purpose Committee to investigate.
2. Senior Lectureship in Meat and Milk Hygiene.
3. Increase in annual subscriptions to arrange for a full-time, paid Secretary/Treasurer.
4. Antibiotics in Milk.

COUNCIL MEETING ON 24TH AUGUST, 1959.

All members present and in addition Drs. B. M. Horwitz, D. J. Louw, P. W. Thorold, W. Wheeler and L. C. Blomefield.

The following matters were discussed:

1. Amendments to the Veterinary Act.
2. Draft Contract for employment of Veterinarians by non-Veterinarians.
3. Life Membership—The Treasury reported that an actuary had determined that £75 should be a single payment for life membership.
4. Congress arrangements.
5. Antibiotic substances as adulterants in Food of Animal Origin.
6. Reports of sub-committees.
7. Branch resolutions.
8. Membership—Announcement of new Council members elected. New members and life members proposed.
9. Finance—Balance Sheet and Financial Statement discussed.

CONGRESS YEAR ENDING 2ND SEPTEMBER, 1960

Council Meetings were held on 19th October, 1959, 25th January, 1960, 2nd May, 1960, 20th June, 1960, and 27th August, 1960.

All Council Members were in regular attendance.

COUNCIL MEETING ON 19TH OCTOBER, 1959:

1. Veterinary Act.—Amendments to. A Committee comprising Drs. Diesel (Convenor), S. van Heerden, R. Clark, P. R. Viljoen and Col. Jeffrey to proceed in the matter of reviewing the proposed amendments under consideration by the Division of Veterinary Services.
2. S.P.C.A., Johannesburg.—The President reported progress in negotiations with the Society.
3. Press comment on Presidential Address.
4. Reports by Standing Committees.
5. Resolutions, 1959 Annual General Meeting. Attention given to these resolutions.
6. Election of Standing Committees.

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COUNCIL MEETING ON 25TH JANUARY, 1960:

All members present except Drs. A. F. Tarr, P. S. Snyman, M. C. Lambrechts, L. W. v. d. Heever and A. M. Diesel.

The following matters were discussed:

1. Professional Provident Society.—Matter discussed and Dr. J. D. H. Poole asked to represent the Association on the Board of the Society.
2. Reports of Standing Committees.
3. Membership.—The deaths recorded of Drs. E. J. Pullinger and W. Pugh-Jones. Association to express condolence to the bereaved families of these members.

   New Members.
4. Finance—
   (*a*) Man and Wife subscriptions.—Where both were veterinarians, the wife's subscription to be suspended and only one copy of the Journal to each such family.
   (*b*) Subscriptions for members outside the Union.—Agreed that this be left at £3/3/0. This not to apply to South West Africa which was part of the Union in respect of membership of the Association.
   (*c*) Bursaries:
      (i) Book Fund Prize.
      (ii) Dr. Maud Bales Bursary—£110 available for 1960.
   (*d*) Investments.—Council authorised to invest £500 in Building Society shares.

COUNCIL MEETING ON 2ND MAY, 1960.

All members present except Drs. H. P. de Boom, P. S. Snyman and A. F. Tarr.

The following matters were considered:

1. Meat Inspection Regulations.—Dr. L. W. v. d. Heever to pursue the matter of amendments to these regulations.
2. Amendments to the Constitution.—Legal advice to be sought regarding majority voting.
3. Draft Contract for Veterinary Employment.
4. Maud Bales Bursary.—Messrs. A. J. Richardson and C. P. v. d. Merwe to share.
5. Cooper Veterinary Fellowship.
6. Agricura Bursary.
7. Reports of Standing Committees.
8. Salaries of Medical Officers in the Public Service. — Now on scale £1,560–£2,100. President to sign a letter to the Chairman of the Public Service Commission expressing the hope that veterinary scales would also receive attention.
9. Pharmaceutical Society of South Africa.—Progress reported in the discussion with members of the Veterinary Faculty.
10. Presentation to Dr. E. M. Robinson on his retirement as Editor of the Journal.
11. Veterinarians in Commerce.—Matter discussed and legal opinion to be sought.

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12. Price of Journal.—5/– per issue to Cadet Members and £1 per issue to students who are not cadet members.
13. Refresher courses for non State Veterinarians.

COUNCIL MEETING ON 20TH JUNE, 1960.

All members present except Drs. P. J. du Toit, H. H. Curson, E. M. Robinson, P. R. Viljoen, A. F. Tarr and C. J. Muller. Dr. K. van der Walt present by invitation.

The following matters were discussed:

1. Grant-in-aid for Journal.—Dept. of Education, Arts and Science expressed regret at not being able to oblige.
2. Meat Inspection Regulations.—Still being considered by Public Health Group.
3. Amendment to Constitution.—Legal opinion regarding the phrase "by a majority of not less than three-quarters of those present and voting", means "at a meeting where ten persons are present and all vote, eight must be in favour."
4. Cooper Veterinary Fellowship.—Dr. Philip of Salisbury recommended.
5. Agricura Bursary.—Dr. E. W. Verwoerd recommended.
6. Public Service Salaries.—President desires amendment of draft letter to Chairman of the Public Service Commission, before he signs it.
7. Pharmaceutical Society.—Progress reported by Dr. K. van der Walt. More effective control visualised over Poisons, Habit Forming Drugs and Potentially Harmful Drugs. The price structure also being reviewed.
8. Reports of Standing Committees.
9. Veterinarians in Commerce.

COUNCIL MEETING ON 27TH AUGUST, 1960.

All members present except Drs. C. F. B. Hofmeyer, P. J. du Toit, A. C. Kirkpatrick, P. R. Viljoen and R. K. Loveday.

The following matters were considered:—

1. Amendments to draft contract for Employment of Veterinarians by non-Veterinarians.—Proposed amendment agreed to and would be recommended to Annual General Meeting.
2. Presidential Address.—President lays his proposed address before Council and accept Council's views on certain aspects.
3. Public Service Salaries.—President requires further amendment of proposed letter to Chairman of the Public Service Commission.
4. Reports of Standing Committees.
5. Veterinarians in Commerce.
6. Pre-Congress issue of Journal.—Editorial Committee congratulated.
7. Joint Husband-Wife subscription.—Should this be a single (Communal) subscription while both husband and wife are earning their living as veterinarians?
8. Congress arrangements.
9. Branch Resolutions.

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10. Election of Office Bearers.—New Council Members, Drs. J. D. Coles, M. de Lange, M. C. Lambrechts and A. F. Tarr.
11. New Members.—18 New members to be recommended to the Annual General Meeting. Recording the deaths of Drs. Keppel, Graf, O'Dowd. Sympathy to be extended to relatives.
12. van der Wath Committee.—Association to give evidence.

CONGRESS YEAR ENDING SEPTEMBER, 1961

COUNCIL MEETING ON 30TH SEPTEMBER, 1960.

All members present except Drs. P. J. du Toit and A. M. Diesel. The following matters were discussed.

1. Venue of next Congress.—Agreed that it be in Durban during the Transvaal school holidays, between 22nd September and 3rd October, 1961.
2. Resolutions.—Annual General Meeting.
3. Standing Committees.—Members Elected.
4. Reports.—Editorial–Finance Committee reported their recommendations for the improved management of the Journal.

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A SHORT REPORT ON THE ITEMS SHOWN BY THE EXHIBITING FIRMS DURING THE 1960 CONGRESS

Among the items shown by the Exhibiting Firms, the following were of special interest.

(1) *Burroughs Wellcome & Co. (S.A.) Ltd.*
(P.O. Box 10293, Johannesburg—Represented by Mr. L. W. Dey.)

EPIVAX-PLUS.—Combined Canine Distemper Vaccine Egg-Adapted (Living), and Canine Contagious Hepatitis Vaccine, Tissue-Culture Adapted (Living) for the active immunisation of dogs at the age of nine weeks.

FAUNOLEN.—Canine Contagious Hepatitis Vaccine Tissue-Culture adapted (Living) specifically for puppies who are much more likely to be exposed to the Virus of Contagious Hepatitis, Faunolen may be injected from the age of three weeks. An injection of Epivax (which has been considerably reduced in price) or Epivax-Plus six weeks later to immunise them against Distemper and Hard Pad.

The well known combination of THEMALON and LETHIDRONE.

THEMALON—is indicated in all circumstances in which a level of analgesia, hypnosis and narcosis is required in dogs.

LETHIDRONE—is used to terminate the effects of Themalon of for that matter, any Morphine derivative.

LUTORMONE and PROTORMONE.

LUTORMONE—is used in the treatment of infertility and other dysfunctions attributable to deficient natural secretion of the luteinising or interstitial cell-stimulating hormone.

PROTORMONE—is for the maintenance of pregnancy in cases of mild luteal deficiency and for the treatment of cystic ovaries.

(2) *South African Cyanamid (Pty.) Ltd.*
(P.O. Box 7552, Johannesburg—Represented by Mr. Coetzee.)

Verizyme, Targot Mastitis Ointment and Acramycin were on Exhibition.

(3) *Datons.*
(South African National Stock Remedies (Pty.) Ltd. P.O. Box 194, Nigel—Represented by Mr. Gamsu.)

Products manufactured by Hoechst, Farbwerke, Frankfurt, Germany, were displayed—particularly *BERENIL* (treatment for Redwater), *SPIROTRYPAN* (treatment for Anaplasmosis) *HOSTACYCLINE* (Broad spectrum antibiotic).

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(4) *Glaxo-Allenburys (S.A.) (Pty.) Ltd.*,
(P.O. Box 485, Germiston—Represented by Mr. Jamieson).

Main prominence on the Glaxo-Allenburys Stand was given to two new products, I-C-D Granules and Fersamal Granules. The former contains iron (ferrous fumarates), calcium and vitamin D and is intended for use in the small animal practice, i.e. for the prevention and treatment of rickets, during pregnancy and lactation, to promote bone growth, etc. Important features of the product are the ease of administration (just sprinkle over normal food) and the palatability—the granules are so flavoured that even the most fastidious siamese kitten will not refuse them.

Available in 2 oz. tins, public price 6/–.

Fersamal Granules are attractively flavoured granules of ferrous fumarate (one level measure, 3½ grams, provides approximately 0.6 grams of ferrous iron). The granules are used for the prophylaxis and treatment of iron deficiency anaemias found in connection with many common diseases such as

Gallsickness in cattle
Liver fluke in cattle and sheep
Redwater in cattle
Biliary fever in horse

and other diseases leaving aftermath of anaemia.

Among other Glaxo-Allenburys products featured were the vaccines for the prevention of canine distemper and canine virus hepatitis, Canilin D and Canilin H and penicillin preparations, Mylipen and Benapen.

One display that aroused great interest with the veterinary surgeons, was the vast array of surgical instruments, and equipment featured by Glaxo-Allenburys Surgical Division. Everything needed for the surgery if not on display at Onderstepoort, is certainly available from the Surgical Division.

(5) *Imperial Chemical Industries Ltd.*
(P.O. Box 11270, Johannesburg—Represented by Mr. P. Lewis.)

FULCIN (Ringworm) *DICTYCIDE* and Sulphamezathine were particularly displayed.

(6) *Libagric (Pty.) Ltd.*
(P.O. Box 15, Pretoria.)

Displayed a wide range of interesting books on Veterinary subjects.

(7) *Maybaker (S.A.) (Pty.) Ltd.*
(P.O. Box 1130, Port Elizabeth—Represented by Mr. Goddin and Mr. Holmshaw.)

Vallergan.—trimeprazine tartrate, a new phenothiazine derivative which may be regarded pharmacologically as being intermediate between LARGACTIL and PHENERGAN. It has a somewhat greater antihistamine action than PHENERGAN and its

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actions although resembling those of LARGACTIL are less intense on the central Nervous System. VALLERGAN differs from LARGACTIL in having a more marked spasmolytic action and a weaker anti-adrenaline action. VALLERGAN possesses the following advantages over LARGACTIL:—

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VALLERGAN is indicated in small animal practice only for:

1. Sedation;
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3. Control of nausea and vomiting.

VALLERGAN is presented in tablets of 10 mg. and 25 mg. strength and in a 1% solution in ampoules of 1 ml. and 5 ml. A professional user only product.

Strinacin.—a triple Sulphonamide and Streptomycin association and a proven theraputic agent for scours—bacterial enteritis and diarrhoea in all species of domestic and farm animals. Dosed once daily at the rate of 2 grammes per 25 lb. bodyweight for two or three days.

STRINACIN is presented as granules in containers of 1 lb. and 4 oz. and also as tablets of 5G strength.

It is of interest, that numerous Veterinarians have found the 5G tablet to be particularly effective when used in the role of a pessary. The formulation and size making it an ideal uterine medicant. A professional user only product.

Rovamycin.—Spiramycin Intramammary Injection, indicated in bovine mastitis wherever there is reason to believe that the common Gram-positive udder pathogens are implicated or when treatment of such cases with other agents has failed to elicit a satisfactory response. For the treatment of mastitis due to Staphylococcus aureus, S. dysgalactiae, S.uberis, S.agalactiae.

A professional user only intramammary product and thus one which the efficicacy will not be impaired through misuse in lay hands.

Vesadin.—The M&B brand of Sulphadimidine B.Vet.C. An all purpose Sulphonamide for the treatment of bacterial infections in all animals, administered orally, or by subcutaneous, intramuscular, intravenous or intraperitoneal injection at intervals of 24 hours.

Available as the Sodium solution 33⅓% w/v (29.8± w/w) in stock remedy packings of 100 ml. and 350 ml. and as a professional user only packing of 2 litres. Stock remedy packings of 50 ..... 0.5G, 250 ..... 0.5G and 20 ..... 5G tablets are also available.

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(8) *Milborrow & Co. (Pty.) Ltd.*
(P.O. Box 216, Pietermaritzburg—Represented by Mr. G. Milborrow.)

A full range of Veterinary Instruments and other interesting items were offered and discussed.

(9) *Optical Instruments (Pty.) Ltd.*
(P.O. Box 1561, Johannesburg—Represented by Mr. Steeg, Mr. Meier, Mr. Seidler and Mr. Schmidt.)

The Automatic photo-microscope with built-in 35 mm. camera and automatic exposure timing, and the Ultraphot large universal photographic microscope, working on plates 9 x 12 were on display.

In addition the well-known range of Zeiss microscopes and accessories—projection head, attachment camera, etc. and pH meters were shown (Metrohn). A full range of balances (Sartorius) and electrical laboratory equipment (Heraeus)—furnaces, drying ovens and incubators and a range of thermostatic water-baths were on exhibition.

(10) *Parke Davis Laboratories (Pty.) Ltd.*
(P.O. Box 24, Isando, Transvaal—Represented by Mr. Lambert.)

A varied range of these notable preparations were displayed.

(11) *Pfizer Laboratories* (S.A.) *(Pty.) Ltd.*
(P.O. Box 7324, Johannesburg—Represented by Mr. Bouchan.)

Emphasis was placed on Terramycin: the new liquid formula for mastitis. X-rays of animal's udder was exhibited to show how the material soaked in. "Embryostat" for repeat breeder cows and "Delta Cortril" both for the use of veterinarians only—were also shown.

(12) *Reckitt & Colman (Africa) (Ltd.*
(P.O. Box 8247, Johannesburg.)

The well-known products distributed by this firm were exhibited and discussed by the representative.

(13) *A. S. Ruffel (Pty.) Ltd.*
(P.O. Box 8724, Johannesburg—Represented by Mr. Caplan.)

Urea phosphate blocks for winter protein supplementation, Nitropen (mastitis) and other items of veterinary interest were demonstrated.

(14) *Squibb Laboratories (Pty.) Ltd.*
(P.O. Box 9975, Johannesburg.)

The many items marketed by this firm were discussed, a great many being on exhibition.

(15) *Chas. Thackray* (S.A.) *(Pty.) Ltd.*
(P.O. Box 2726, Johannesburg—Represented by Mr. Stabler.)

Exhibited a full range of surgical instruments including their "Pocket Sterilizer" Suture material, Surgeons Plastic Aprons.

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The Bulk Autoclav now being supplied to Onderstepoort was catalogued—No. 5762B.

(16) *Wild of South Africa (Pty.) Ltd.*
(Keig House, Johannesburg—Represented by Mr. Daisley.)

The M.4 Stereomicroscope was exhibited for the first time. In addition the M.5, M.11 and M.20 Stereos were on exhibition.

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OUTBREAKS OF NOTIFIABLE DISEASES—QUARTER ENDING 30th SEPTEMBER, 1960.

1. Anthrax	Johannesburg T Koppies O Barberton T Estcourt N Boshof O Nelspruit Belfast T Wynberg C Bloemfontein O
2. Benign Bovine Gonderiosis .. (Tzaneen Disease)	Piet Retief T Mahlabatini N Harding N
3. Brucellosis	Richmond C
4. B.W.D.	Malmesbury C
5. Fowl Typhoid	
6. Foot and Mouth Disease ...	Pilgrimsrest T
7. Lumpy Skin Disease	Mossel Bay C
8. Malignant Syncerine Gonderiosis (Buffalo Disease)	Hlabisa N
9. Mange ..... *Equine* *Bovine* *Goat*	Umtata C St. Marks C Umzimkulu C Wynberg C Peddie C Willowvale C Hlabisa N Tsomo C Nongoma N St. Marks C Ubombo N Ndwedwe N Umlazi N
10. Nagana	
11. Rabies	Brandfort O Steynsburg C Boshof O Carolina T Petrusburg C Kimberley Viljoenskroon O Schweizer Reneke Letaba T
12. Sheep Scab	Mafeking C
13. Swine Erysipelas	
14. T.B. ..... *Bovine* *Avian*	Hlabisa N Pietersburg T Bredasdorp C Durbanville Worcester C Malmesbury C Hopefield C

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DOODSBERIG Dr. H. GRAF



Hermann Graf is uit Duitse ouers in Johannesburg op 23 Augustus 1898 gebore. Hier het hy sy skoolloopbaan begin waar hy dan ook as jong seun osteomiëlitis opgedoen het. Nadat hy talle operasies ondergaan en vir bykans 'n jaar uit aksie was het sy ouers hom na sy grootouers in Duitsland gestuur om te herstel, waar hy dan ook vir twee jaar in Karlsruhe en Kirchhofen skoolgegaan het.

Terug in Suid-Afrika het hy aan die Stellenbosche „Boys High School" sy skoolloopbaan in 1917 voltooi en die matrikulasieeksamen met sukses afgelê. Gedurende 1918 volg hy die pasingestelde eerstejaarskursus in B.V.Sc. aan die Stellenbosch Universiteit wat egter aan die einde van die jaar afgeskaf is.

Die hunkering na die natuur was op die stadium so groot dat hy vir twee jaar op „die eiland" ('n eiland in die Vaalrivier naby Parys) gaan boer het.

Hierna is hy in 1920 terug na Universiteit en het aan die T.U.K. aan die einde van die jare 1922 en 1925 die grade B.Sc. en B.V.Sc. verwerf. Hy was onder die tweede groep wat 'n veeartsenykundige graad in Suid-Afrika verwerf het. Ook het hy in 1932 aan die Universiteit van Pretoria die D.V.Sc. graad behaal.

Op 4 Februarie 1926 word hy as Veeartsenykundige Navorsingsbeampte op Onderstepoort aangestel en het hy vanaf 1927 opgetree as lektor, aanvanklik in Biochemie en later in Chemiese Patologie (Dipstowwe insluitende).

Vanaf die stigting van laasgenoemde seksie in 1929 tot sy aanstelling op 3 November 1954 as Adjunk-Direkteur van Veeartsenydiens was hy hoof van dié seksie, terwyl hy ook nog vanaf 1 September 1949 reeds die pos van Onder-Direkteur beklee het.

Saam met sy pligte as Adjunk-Direkteur moes hy ook nog vanaf Januarie 1956 as Dekaan van die Fakulteit van Veeartsenykunde optree. Gedurende hierdie tydperk is hy ook in Januarie 1958 vanaf senior lektor na mede-professor bevorder en het hy gedurende Mei 1960 'n Internasionale Kongres vir Veeartsenykundige opvoeding in Londen bygewoon.

Gedurende die oorlogsjare was daar 'n gedwonge onderbreking in sy dienste op Onderstepoort toe hy op 25 September 1941 geïnterneer is. Gedurende dié jare het hy aan mede-geïnterneerdes 'n kursus in veeartsenykunde op Koffiefontein aangebied. Op 11 Desember 1945 is hy egter weer in ere herstel op Onderstepoort en het hy veral vir die laaste tien jaar 'n reuse-aandeel geneem in die afdeling se werksaamhede.

In die verenigingslewe was hy ook 'n aktiewe lid van die volgende liggame:—

1. S.A. Vereniging Bevordering van Wetenskap (S2A3) sedert 1926.
2. S.A.V.M.V. sedert 1926.
3. Stigterslid van die Fakulteit van Wetenskap van die S.A. Akademie waarvan hy dan ook later as volle lid van die akademie vir Wetenskap en Kuns verkies is.
4. S.A. Bosbouvereniging sedert die stigting in 1937.
5. S.A. Botaniese Vereniging sedert 1935.
6. Veeartsraad (Suid-Afrika)—1935 en sedert 1956.

Veral was hy 'n natuurliefhebber en het baie gedoen om ons inheemse plante en struike aan die publiek bekend te stel en die aanplant daarvan as sierbome in tuine aan te moedig.

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Sy belangstelling was besonder wyd, so ook het hy baie belang gestel in die opera en musiek. Boal was hy 'n persoon wat baie vir sy medemens probeer doen het. Persone sonder heenkome kon en het altyd 'n onderdak in sy huis gevind. Vir jare was hy in die Verkennersbeweging en later het hy sy plaas as kampeerterrein aan die Voortrekkerbeweging en ander jeugbewegings beskikbaar gestel. Hy is dan ook deur hulle vereer met die toekenning van ,,vriend van die Voortrekkers".

Of persone van hom verskil of saamgestem het, in een opsig is almal dit eens en dit is dat hy 'n persoon met integriteit was, 'n persoon wat lojaal was teenoor sy volk, sy Departement en altyd vriendelik en behulpsaam was.

Op Januarie 1926 is lêer Nr. A.31/G6/70 op Onderstepoort geopen. Op 7 Junie 1960 is dit met die afsterwe van 'n ware vriend, net na 'n komitee vergadering wat moes besluit oor die ,,Cooper Veterinary Fellowship", gesluit. In dié verband gaan ons innige simpatie uit na sy gade; Rita en kinders, Gudrun en Leana.

P. M. BEKKER.

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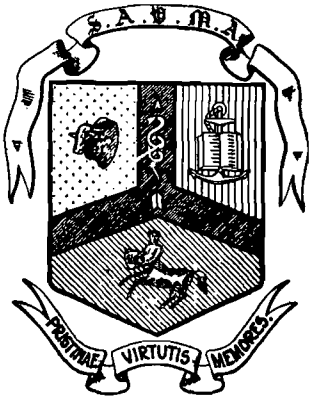
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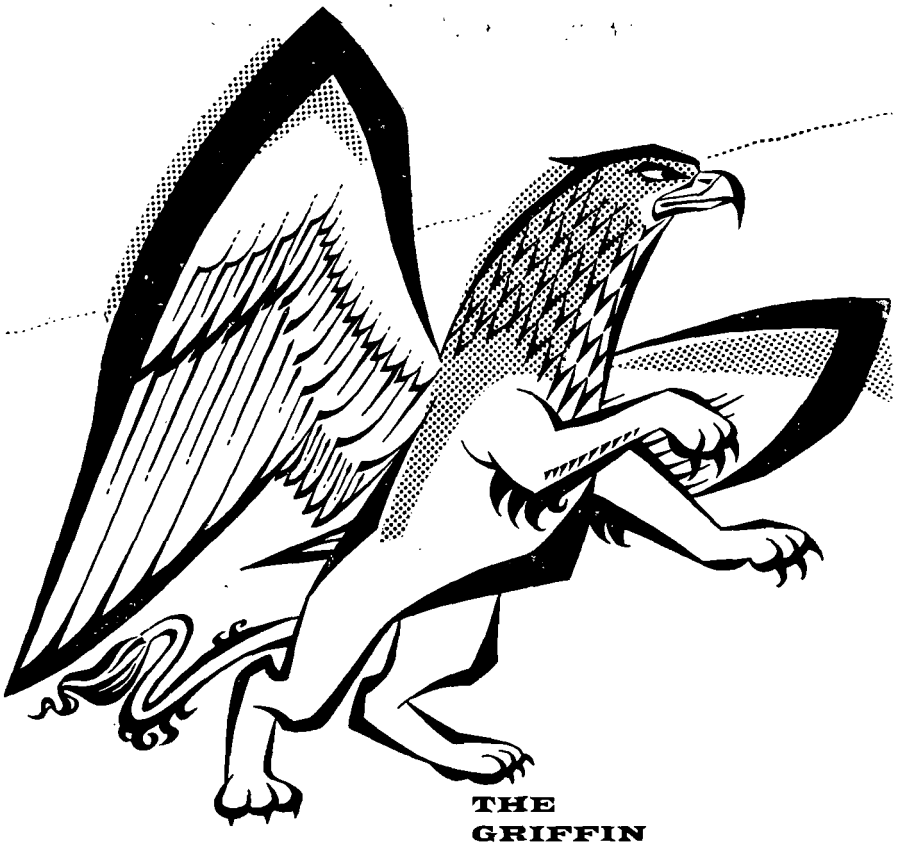
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DIE SUID-AFRIKAANSE

VETERINÊR-MEDIESE

VERENIGING

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THE GRIFFIN

This is called a GRIFFIN because it is a winged quadruped. This kind of wild animal is born in Hyperbosean parts, or in the mountains. All its bodily members are like a lion's, but its wings and mask are like an eagle's.
T. H. White *The Book of Beasts*

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SOME OBSERVATIONS ON OEDEMA DISEASE AND A POSSIBLY RELATED CONDITION OF PIGS IN SOUTHERN RHODESIA

J. R. Philip and D. K. Shone.*

(*Received for publication 17th September* 1960)

Introduction

Mortality in pigs due to oedema disease has been recognised in Southern Rhodesia since about 1955, but the recording of severe losses only began in 1957 and 1958. The disease is now recognised as a most important cause of losses in weaner pigs up to the age of 4 months. Outbreaks have been diagnosed in practically all the pig-rearing areas of Southern Rhodesia.

Laboratory records, and reports from the Field branch of the Department of Veterinary Services, reveal an incidence of more than 30 outbreaks of oedema disease during the period June 1959 to July 1960. The actual mortality in each outbreak is difficult to assess as the losses are seldom recorded in detail. In some instances the mortality has been as high as 90% of all weaners, while in others only 2 or 3 pigs succumbed.

There are reasons for believing that the incidence may be higher than official records show, as the disease is not scheduled and therefore not always reported by private practitioners or farmers. In addition, difficulty is frequently encountered in establishing a diagnosis based on the examination of a single carcase, in which the characteristic lesions may be very slight, present in atypical sites only, or particularly in the case of decomposing carcasses, absent altogether[20].

EPIZOOTIOLOGY

The disease occurs without any particular seasonal incidence, throughout the year. Outbreaks tend to be sporadic with sudden mortality, mainly in weaner pigs between the ages of 6–12 weeks, the disease frequently disappearing as rapidly as it came. Only two or three pigs in a litter may be affected in an outbreak, or mortality may be spread indiscriminately over a varying number of litters in the herd.

On some farms, however, the disease has been known to occur enzootically causing heavy mortality in one litter after another, generally about a week after weaning. Many farmers within reasonable access of dairy processing plants, feed skim milk to their fattening pigs on an *ad lib* basis. When for some reason, the supply is withheld for 24— 48 hours, losses due to oedema disease are frequently encountered within a day or two of resuming the milk diet. Occasional outbreaks have

* Veterinary Research Laboratory, Salisbury, Southern Rhodesia.

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been encountered, where a change in the diet has been excluded as the predisposing factor, and in some outbreaks mortality has occurred prior to weaning.

The symptoms when observed are generally typical of the disease as described in the literature (Erskine, Sojka and Lloyd[1]; Gauge[4]; Gregory[6,7]; Lamont, Luke and Gordon[9]; Luke and Gordon[12]; Shanks[14]; Sojka, Ersken and Lloyd[15]; Timoney[18], [20], [21]).

The classical "puffy" or swollen eyelids have only occasionally been encountered.

Macroscopic pathognomonic lesions are generally only found in relatively fresh carcases. The typical oedematous lesions eventually disappear and may not be observed in pigs that have been dead for several hours (Gouge[4], Timoney [22]), particularly under Rhodesian summer conditions when autolytic changes are extremely rapid. The most typical lesion is an oedematous thickening of the gastric wall in the region of the greater curvature. The oedema may be immediately visible and up to 2 cm. or more in width, or revealed only by detailed sectioning with a knife.

A less common but equally pathognomonic lesion is an oedematous thickening of the mesocolon, more easily observed before the viscera are removed. Ascites, hydropericardium and oedema of the lungs are occasionally noted but are not altogether characteristic.

The urine, when present, has been found in nearly every case to give a positive reaction for sugar with Benedict's reagent but the value of this test as a diagnostic aid is uncertain.

ISOLATION AND TYPING OF HAEMOLYTIC ESCHERICHIA COLI STRAINS

In almost every case where cultural examination has been undertaken, direct inoculation of the mesenteric lymph nodes onto blood plates has produced pure cultures of beta haemolytic Escherichia coli. Likewise, cultures made from the caecum and colon of typical cases have revealed a predominance of haemolytic E. coli in the gut flora. This is not always the case with the small intestine, however. Cultures from the parenchymatous organs of fresh carcases are invariably sterile.

Escherichia coli cultures from 5 different outbreaks of oedema disease were suspended in equine serum, dried under vacuum, and submitted to the Central Veterinary Laboratory, Weybridge, England for serological typing.

All of these strains were isolated from pigs exhibiting pathognomonic lesions of oedema disease.

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The results were as follows[17]:—

"E 68" type serotypes

Group designation (0141 a (*b*) : K85 (*B*) K XI (L)).

Strain No. 18. Pig Farm, Shamva District.
Isolated 8.1.60 from the colon.
History typical of oedema disease.
Two weaners submitted for post mortem.
Both showed marked oedema of the mesocolon.

Strain No. 51. Pig Industry Board Experimental Farm, Salisbury District. Isolated 15.1.60 from the caecum of a 9 week-old weaner showing oedema of the stomach wall. Mortality 3 out of a litter of 9.

Strain No. 310. Pig Farm. Marandellas District.
Isolated 23.3.60 from mesenteric lymph node.
History of sporadic mortality in several litters.
Post mortem examination showed oedema of the stomach wall.

Strain No. 377. Litter testing station. Salisbury District.
Isolated 17.4.60 from mesenteric lymph node.
History of sudden mortality in a group of weaners.
Post mortem showed oedema of the stomach wall and colonic mesentery.

"E 4" Serotypes

Group designation (0139: K82 (B)).

Strain No. 378. Gwebi Agricultural College.
Two cultures. Isolated 17.4.60 from mesenteric lymph nodes of 2 pigs.
History:—Typical oedema disease in young weaners with mortality of 14 piglets over a 5 day period in 4 separate pens.

SENSITIVITY TO ANTIBIOTICS

Twelve strains of haemolytic E. coli isolated from different outbreaks of oedema disease have been tested for their *in vitro* sensitivity to several antibiotics in common use by means of "Difco" sensitivity discs.

These strains showed a marked lack of sensitivity to the tetracycline group, penicillin, erythromycin and polymixin. On the other hand all strains were sensitive, even at low concentrations, to chloramphenicol and streptomycin.

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HAEMORRHAGIC ENTERITIS SYNDROME

A haemorrhagic enteritis syndrome that may have some relationship to oedema disease is sometimes seen in store pigs and porkers. The disease is acute, sporadic and usually affects only two or three pigs in a herd and generally only those in very good condition.

The main characteristic is sudden death without any symptoms, though occasionally a reddish tinged diarrhoea may be seen. Decomposition sets in rapidly, but the outstanding post mortem lesion is an extensive haemorrhagic and sometimes also, diphtheritic enteritis, typhlitis and colitis. The impression gained is that the pig has virtually "bled out" into the gut.

Frequent attempts to demonstrate vibrios in the lining of the gut have failed, but haemolytic E. coli can usually be isolated from the mesenteric lymph nodes. On the few occasions when fresh carcases have been available, almost pure cultures of haemolytic E. coli have been isolated from the lumen of the caecum and colon. Only two such strains have thus far been serologically typed and both were found to belong to Type E. 68 (a common "oedema disease" serotype).

Centrifuged bowel supernate in one of these cases also proved highly lethal for mice and was not neutralised by Clostridium perfringens type B. C. or D. antisera.

A very similar syndrome in well conditioned store pigs has also been observed in England[17].

EXPERIMENTAL WORK

A limited series of transmission experiments based on the work of Timoney[18, 19, 20, 21, 22]. Gitter and Lloyd[3] and Erskine *et al.*[1] has been undertaken. The trials were limited by the difficulty in obtaining fresh material and the non-availability of experimental pigs.

Experiment A.

The small and large intestines from a pig which had died three hours previously, and showed typical lesions of oedema disease, were separately perfused with 40 ml. of normal saline. The contents thus obtained were then allowed to "extract" by placing them in the refrigerator overnight at 6°C. Haemolytic E. coli predominated in direct cultures from the colon but serological typing was not undertaken.

The intestinal contents were then centrifuged at 3,500 r.p.m. for one hour, the supernatant fluid decanted and again centrifuged. A clear, greenish-tinged extract was obtained.

Two 10 week old pigs weighing approximately 50 lbs. were inoculated, one with 20 ml. of extract from the small intestine and the other with 20 ml. of extract from the large intestine. The material was given by slow intravenous injection into the anterior vena cava.

The results are tabulated in Table I.

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TABLE I

EXPERIMENTAL REPRODUCTION OF OEDEMA DISEASE BY INTRAVENOUS INOCULATION OF PIGS WITH INTESTINAL MATERIAL OBTAINED FROM TYPICAL CASES

Pig No.	Serotype Isolated	Material Inoculated	Quantity	Route of Inoculation	Date	Symptoms	Post mortem
J 50	not typed	Centrifuged supernatant "extract" from small intestine	20 ml.	Anterior vena cava	15.2.60	Slight shock following injection. Otherwise negative	—
45	not typed	Centrifuged supernatant "extract" from large bowel	20 ml.	Anterior vena cava	15.2.60	Shock dyspnoea, cyanosis followed by complete recovery	
R.H.	"E. 4"	Centrifuged supernatant fluid from small intestine	10 ml.	Ear vein	10.5.60	Immediately followed by severe shock and respiratory failure Death in 15 minutes	General venous congestion particularly of intestinal mucosa
R.B.	"E. 4"	,,	10 ml.	,,	10.5.60	,,	,,
R.T.	"E. 4"	,,	5 ml.	,,	10.5.60	Shock and respiratory failure Death in 30 minutes	Marked congestion of intestinal mucosa. Extravasation of blood into colon
6	"E. 4"	Centrifuged supernatant from small intestine—Seitz filtered	40 ml.	Ear vein	20.5.60	Followed 24 hours later by inappetence and unsteady gait Found dead after 36 hrs.	Marked oedema of mesocolon and gastric wall. Glycosuria.
Y.H.	not typed	Centrifuged supernatant from small intestine—Seitz filtered.	10 ml.	Ear vein	5.6.60	Slight shock following injection. Complete recovery. No other, symptoms seen	—
Y.B.	not typed	,,	50 ml.	,,	7.6.60	Slight dyspnoea following injection. 8.6.60 Inappetence and listlessness. 9. 6. 60 Found dead.	Diphtheritic and haemorrhagic colitis No oedematous lesions present

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It was apparent that any toxin(s) that may have been present had been severely diluted and apart from some initial shock following the injection, no symptoms were noted.

Experiment B.

Three pigs from another outbreak were used as the source of material for the second trial. Two of these were killed *in extremis* while the third had died some two hours previously.

The contents of the small intestines of the three pigs were collected, pooled, centrifuged and the supernatant fluid stored at —20°C. Haemolytic E. coli strains were isolated in pure culture from the mesenteric lymph nodes of the two pigs slaughtered *in extremis* and both were subsequently typed as "E. 4." serotypes [0139: K 82 (B)]. Bacteriological examination was not undertaken on the third pig.

Initially, three 10 week old pigs were inoculated, two with 10 ml. each, and one with 5 ml., of the supernatant fluid. All inoculations were by slow intravenous injection into an ear vein, the pigs being restrained under light chloroform anaesthesia.

This material proved extremely toxic and the injection was immediately followed by respiratory failure, and death within 15-30 minutes.

In an attempt to eliminate the toxic factor, the supernatant fluid was filtered through a Seitz E K pad. This material was then inoculated into a much older pig weighing 120 lbs. which had been previously splenectomised (for a protozoal transmission experiment.).

There was no immediate shock. Within 24 hours the pig showed signs of listlessness and inappetence followed by incoordination of gait. On the morning of the second day it was found dead.

Post mortem examination revealed a marked oedema of the colonic mesentery and an oedematous thickening of the stomach wall (greater curvature) of up to 2 cm. in thickness. The urine gave a positive reaction for sugar with Benedict's reagent. Cultural examination of the mesenteric nodes, small intestine and colon failed to reveal the presence of haemolytic E. coli.

Insufficient material remained to reproduce the condition a second time.

Experiment C.

The contents of the small intestine of a pig which died in another outbreak were centrifuged and the supernatant fluid passed through a Seitz E K filter. The haemolytic E. coli strains isolated in this instance were contaminated with a spreader organism subsequently and could not be submitted for serotyping.

An injection of 10 ml. of Seitz filtered material administered to a 10 week old pig (Y.H.) proved innocuous. The dose was therefore increased to 50 ml. and given a littermate (Y.B.). On the following day the pig was listless and showed disinterest in its feed. It was found dead on the morning of the second day. (Table 1).

The striking lesions seen on post mortem examination were an extensive diphtheritic and haemorrhagic colitis with markedly blood stained contents, very similar in appearance to the haemorrhagic enteritis syndrome described above. There was no evidence of oedema and no haemolytic E. coli could be isolated from the colon.

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DISCUSSION.

The association of certain specific serotypes of haemolytic E. coli with oedema disease of pigs has been established by Sojka, *et al*[15] Ewing *et al*[2], Gregory[8] and Sojka *et al*[16]. It was therefore of considerable interest to find, that although only a limited number of strains from locally occurring oedema disease outbreaks have so far been typed, the organisms (serotypes) involved were identical with the two most commonly occurring serotypes associated with oedema disease in Britain[16]. The exact nature of the predisposing factor(s) leading to the rapid multiplication and production of toxin(s) by these strains of haemolytic E. coli is not fully understood but it appears that the widely varying conditions of management, housing, feeding and environment pertaining in Europe, North America, Japan or Africa do not, *per se*, play an important rôle in the aetiology of the condition. Furthermore, it has been our experience that all pigs, whether run under good or bad hygienic conditions, are equally susceptible to outbreaks of oedema disease.

In attempting the artificial reproduction of the disease by means of bowel supernates on three occasions we encountered the same difficulty reported by Lamont et al[9] and occasionally by Timoney[19,20] of shock and sudden death following intravenous inoculation of intestinal material. The centrifuged supernatant fluid used initially was found on culture to be heavily contaminated with a variety of gram negative organisms, and it may be of some significance that after Seitz filtration, the same material gave rise to a condition indistinguishable from oedema disease and without any symptoms of shock.

A considerable amount of further investigational work is required into the aetiology of the condition described as an acute haemorrhagic enteritis. The fact that haemolytic E. coli strains have occasionally been demonstrated as the predominant organism occurring in the large bowel, and that two of these were found to belong to a common "oedema disease" serotype; demonstration of a toxic factor lethal to mice in the centrifuged intestinal contents from a typical case; and the reproduction of an apparently identical condition in a pig by intravenous inoculation of Seitz filtered intestinal material from a typical "oedema disease" case, have led us to believe that the haemorrhagic enteritis syndrome may be related to oedema disease.

SUMMARY

The incidence, epizootiology and pathological diagnosis of oedema disease in swine in Southern Rhodesia is described. Bacteriological findings indicate that the haemolytic Escherichia coli serotypes isolated from oedema disease cases in Southern Rhodesia, are identical with the most commonly occurring strains associated with the disease in Britain.

A syndrome indistinguishable from oedema disease, was reproduced in one pig by intravenous inoculation of Seitz filtered intestinal material, obtained from 3 pigs showing pathognomonic lesions of oedema disease.

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A condition occurring mainly in store and fattening pigs in which the characteristic lesions are a haemorrhagic, and sometimes diphtheritic enteritis, has been encountered. A limited amount of experimental work undertaken on the latter condition, suggests that it may be related to oedema disease.

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	CONTROL ANIMALS		TREATED ANIMALS	
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0	283	0.07	283	0.07
2	277	—*	279	—*
4	305	0.29	334	0.53
6	441	0.25	475	0.47

* *Liver Vitamin B12 not estimated.*

REFERENCE + *Skerman, K. D., Sutherland, A. K., O'Halloran, M. W., Bourke, J. M., and Munday, B. L., (1959), Am. J. Vet. Res., XX, 977.*



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"MULTIPLE ACANTHOMA IN THE SKIN OF SWINE"

K. C. A. Schulz* and J. A. Schutte**.

(*Received for publication* 17th *September* 1960)

Acanthomata are epitheliomas i.e. malignant tumours characterized by prickle cells and often also by "pearls" or concentric keratinization, and have a tendency to metastasize to regional lymph nodes. In this country they are by far the most frequently occurring tumours of domestic animals, especially of bovines. In the Angora goat they are equally frequent as melanotic epitheliomas. In equines they are exceeded in frequency only by "sarcoids": in the dog, only by contagious venereal tumours: and in sheep, only by (gland-cell) carcinomas[1]. They are among the rarer neoplasms of the fowl, and have not been encountered in the cat, and, until recently, in the pig.

Although affections of the skin in hogs are considered to be very common[2], the scarcity of epithelial tumours, especially malignant ones, in swine has been emphasized in several recent publications [3, 4, 5, & 6]. These observations are in accordance with previously recorded data[1, 7, 8, 9, & 10]. Hogs thus seem to be less susceptible to carcinomatous growths than such domestic animals as cattle, equines, sheep and dogs. It has even been suggested that this species possesses a marked racial insusceptibility to new growths[9]. But, since by far the greater number of hogs are slaughtered at an age less than two years, this short span of life may possibly be responsible for the apparent lack of neoplasms in the majority of these animals. It will, therefore, always be difficult accurately to determine the frequency of tumours in an animal, such as the pig. However, malignant melanomas, lymphoid tumours, and embryonal nephromas appear in swine before they reach market age.

Noteworthy is it that multiple round-cell sarcomas of the skin of a sow[11], and more recently a case of multiple skin papillomata[12], have been described.

This communication records the unique occurrence of multiple acanthoma of the skin, with a variable incidence, in different droves of Large White pigs. The material was obtained from three very widely separated sources in the Transvaal. The first case was diagnosed on a farm near Settlers in 1953, six breeding sows being affected. In 1956 an outbreak was recorded on the farm Nooitgedacht, Rustenburg District, where 15 out of 50 sows, two to three years old, were diseased. The swine were kept in well-fenced camps with thorn trees to provide shade. When the sows were due to farrow, they were moved into cement stalls previously sprayed with a 5 per cent BHC solution. This was also used as a spray against flies pestering the animals. Subsequently,

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in June 1958, several such skin lesions were received from the abattoir of Vereeniging. The exact source of the diseased hogs could, however, not be definitely determined. Presumably they had been purchased on a farm in the Rustenburg District. It is noteworthy that all pigs on the farm Nooitgedacht were sold during the beginning of that year.

It is striking that this condition appeared only in breeding sows. Boars and shoats, kept under identical conditions and in close contact with the diseased ones, remained apparently healthy. There is evidence that the affection is not transmissible by direct contact. A fourteen day old piglet, which had been placed in the same stye with a severely affected sow (fig. 1), showed no signs of the disease after several months.

Spirochaetosis, following on initial irritation by biting flies or some other mechanical agency, had been tentatively diagnosed. Although spirochaetes were found in smears from various lesions, the infection was not considered to be the primary cause. In addition, the majority of the most pronounced lesions did not conform to those previously ascribed to these parasites[13],[14].

Symptoms of Multiple Acanthoma.

In all cases the nature of the affection did not differ appreciably except in degree. The size, shape and extent of the lesions varied considerably, not only in the different animals, but even in each individual. Practically the entire body surface, except that of the lower extremities, was involved. The periorbital and aural regions of the head, the regions of the neck, shoulders, withers, back, sacrum, thigh, flank and abdomen appear to be the most vulnerable areas. The condition is manifested by numerous well-defined scabs or crusts with irregular or more or less circular outlines; up to 100 and even more of these may be encountered in one pig (fig. 1). The milder, slightly raised lesions are covered by light brown, easily detachable scabs revealing moist reddish-pink tinted erosions on removal. In the advanced, more severe affections, thick, dark brown crusts result from a large amount of sero-sanguineous secretion on drying (fig 2). These can only be detached with some difficulty and then ulcers of variable sizes are exposed. The base of such a lesion may be very irregular and have a variegated appearance; translucent (hyperplastic epithelium) and opaque (necrotic) areas being present. Some of the lesions have a tendency to bleed. Spontaneous shedding of crusts may occur after a suppurating process developed beneath it. The edges of the ulcers are either obviously elevated, serrated and firm, or occasionally undermined. They continue to expand, while the central ulcer deepens and becomes excavated, forming a crater. This is markedly accentuated when the lesion becomes fly-blown (myiasis). Some of these affections, on histological examination, prove to be ulcerating carcinomata.

Another type of lesion grows as a cauliflower-like tumour on a broad base. The elevated, more or less domeshaped surface, may stretch to a variable extent beyond the circumference of the lesion, covering a zonary area of epithelium in the precancerous stage (papillary type of squamous cell carcinoma). It may be covered with crusts or scabs, which upon removal reveal an easily bleeding papillary base.

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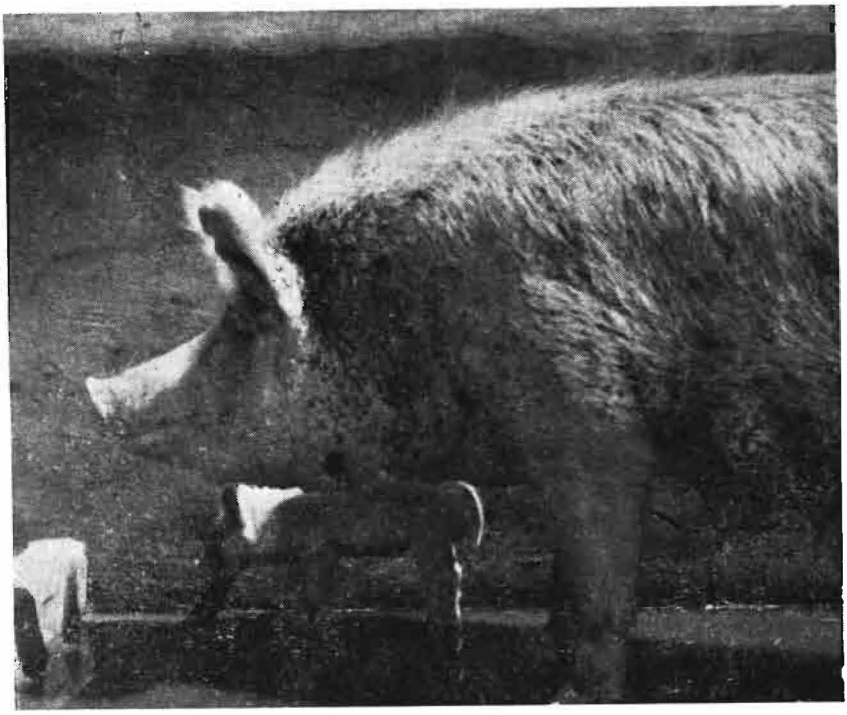


FIG. 1.—Severely affected sow with young piglet in close contact. Note the distribution of the lesions on the sow's body.



2.—Demonstrating a pronounced dermatosis
ed by numerous lesions varying in size, shape and structure.



FIG. 3.—Cut surface of a prescapular lymph node. Note the marked nodular appearance consisting of lymphoid hyperplasia and/or metastatic foci with or without necrosis.

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The consistency of the tumours, which often have a diameter from 3 to 4 cm. is usually firm. Occasionally the necrotic part of the growth sloughs off giving rise to ulceration. Sometimes raised, fungoid, verrucous lesions without ulceration are observed. The latter are characterized by a limited malignancy, being locally invasive but not metastatic, whereas the ulcerated ones may expand and cause metastasis in some of the superficial regional lymph nodes, such as the prescapular (fig. 3).

This disease does not seem to cause the patients much discomfort or distress, except a stiff gait in the hindquarters and a disinclination to rise, after they have been kept in styes for several months. The animals are not inclined to rub or scratch the body, indicating that the lesions do not cause an appreciable irritation. The appetite and food intake is apparently not impaired, since the affected pigs continue to put on weight at rates considered satisfactory. However, there may be a loss of weight during the course of the disease, when secondary conditions, such as pleuro-pneumonia, develop.

Macroscopic Lesions.

It is noteworthy that, in addition to the above mentioned skin affections, the majority of the superficial lymph nodes are markedly enlarged and nodular to a variable extent. The enlargement of the glands is mainly due to oedema, hyperplasia of the lymphoid tissue, and/or a purulo-haemorrhagic lymphadenitis, e.g. the right prescapular lymph node of one case, DOB 1692, measured 12.5 × 10 × 6 cm. and weighed 400 grams. A number of suppurating foci varying in size, and probably caused by Corynebacteria, formed the main lesions in this case. Metastasis was clearly observed in several of the superficial lymph nodes of two cases, DOB1666 and 1692; the condition being more pronounced in the former than in the latter animal. On section the metastatic lesions were well defined, firm, and generally of a lardaceous appearance. In some of them, however, central necrotic foci were observed. Lesions of minor importance are hyperaemia and oedema of the meninges; hyperaemia, oedema and disseminated foci of a non-purulent encephalitis (one case); venous stasis, central atrophy and degeneration of the hepatic cells to a variable extent; degeneration of the myocard and necrotic myositis involving especially the semitendinosus and semimembranosus muscles of both hindlimbs extensively (possibly the cause of the stiff gait and the disinclination to rise); hyperaemia, oedema and emphysema of the lungs, and an acute purulent pleuro-bronchopneumonia in one case (DOB 1666). It may be assumed that the above tissue alterations, except those in the lymph nodes, are not directly associated with the skin affection.

Histopathology.

Histological studies were made on biopsied specimens of one pig, various portions of skin and of other tissues, especially several superficial lymph nodes, of 4 pigs obtained on autopsy, and material (skin and several lymph nodes) received from the abattoir at Vereeniging. Sections from at least ten affected pigs were examined. Histologically

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two distinct types of lesions are evident in the skin; benign precursory and definitely malignant changes being present. The initial stage of the former is that of a papule or vesicle. The larger lesions seem to develop by confluence with satellite lesions at the periphery. Various transitional stages such as chronic eczema and/or dermatitis crustosa exist between it and the final squamous-cell epithelioma. The malignancy of this structure is manifested by its variably invasive character. The tumour consists of irregular masses of epidermal cells, which proliferate downward and invade the corium. The invading tumour masses are composed in varying proportions of differentiated squamous and keratinized cells and of dedifferentiated (anaplastic, atypical) squamous cells; the number of the latter being an index of the degree of malignancy. The atypicality of the squamous cells is manifested by changes such as great variation in the size and shape of the cells, the absence of prickles, keratinization of individual cells, prevalence of mitotic figures and presence of atypical mitosis. Keratinization often takes place in the form of cornified "pearls". These are very characteristic structures, composed of concentric layers of squamous cells showing gradually increasing cornification towards the centre which may or may not be completely keratinized. In addition several lymphatics are invaded by tumour cells (resulting in metastasis). Ulceration of some of the lesions occurs with a marked increase of fibroblasts, which occasionally replace the epithelial cells to such an extent that structures, simulating sarcomas, are formed. Rarely are typical lesions of spirochaetosis, or of streptothrix or of demodex seen.

Oedema, hyperplasia of the lymphoid tissue and metastatic acanthomatous foci are the most pronounced changes in the affected lymph nodes. In addition a number of giant cells and signs of suppuration are dispersed throughout sections of some of the lymph nodes. The lesions in the affected muscles are manifested by hyalin degeneration, necrosis and focal non-purulent myositis of variable degree. No significant changes are observed in sections of various other organs taken for histological examination.

Course

A number of the more benign lesions, eczematous and papillomatous, tend to heal. Relatively large areas of the skin, especially the aural, clear up after some time. However, the more severe lesions apparently do not improve. Thus, although the condition does not terminate in complete recovery, a partial spontaneous improvement takes place.

Transmission

All attempts to transmit the disease experimentally to pigs, mice, rats, rabbits and guinea pigs, failed. Material (pieces of affected skin and lymph node) obtained at autopsy was macerated and ground in a mixture of sterile sand and isotonic salt solution, and rubbed into a scarified area of the skin, and injected intradermally or intraperitoneally. A transitory dermatitis was the only lesion which developed

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on the scarified areas. Even a piglet kept in very close contact with two severely affected sows from the age of 14 days to 4½ months showed no signs of the condition. The cause of the disease thus remained obscure. However, since such a number of animals were affected at the same time, a common chronic noxis (irritant), presumably, was involved.

SUMMARY

A clinical and pathological study is reported of a unique dermatosis characterized by multiple acanthoma, affecting a number of sows in a particular drove and several pigs slaughtered at the abattoir of Vereeniging. Striking of this condition is that mild lesions, such as exzema and dermatitis, gradually but progressively may change to the more severe ones (acanthoma). Characteristics of the latter are distinct "pearl formation", hyperplasia of prickle cells, infiltrative growth, invasion of some lymphatics, ulceration on the surface of some of the circumscribed lesions and an increase of fibroblasts, which in some cases displace the epithelial cells to such an extent, that structures simulating sarcomas are formed. The occurrence of metastatic foci in a number of the superficial lymph nodes of several affected sows is noteworthy. Except the typical acanthomatous lesions, oedema, hyperplasia of the lymphoid tissue, necrobiosis and necrosis, a number of giant cells, and signs of disseminated suppuration are evident. Hyalin degeneration, necrosis and focal non-purulent myositis, involved relatively large areas of the semi-membranous and semitendinous muscles. Attempts to produce the disease experimentally failed. The cause of it remains obscure, but it is assumed that some common irritating noxious agent may be a contributing factor. The spirochaete and streptothrix infections and the demodex infestation occasionally encountered, are considered to be superimposed conditions.

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A PRELIMINARY REPORT ON pH (HYDROGEN-ION CONCENTRATION) TESTING OF MEAT BY THE NITRAZINE YELLOW METHOD, WITH SPECIAL REFERENCE TO CASUALTY ANIMALS PRESENTED FOR SLAUGHTER.

J. H. Huyser, M. M. Greathead and P. J. Meara.*

(*Received for publication 12th November* 1960).

Introduction: This subject was discussed by van den Heever[3], and Meara[2]. Briefly, the test is based on the post mortal enzymic change of muscle glycogen to lactic acid.

The development of acidity in a carcase subsequent to slaughter is important, due to the fact that it enhances the keeping quality of the carcase and has other effects on the meat. Acid meat is of open structure, light cutting, easily releases its flavour on cooking, readily absorbs sugar, brine, salt and smoke in curing processes and forms an unfavourable medium for the development and growth of bacteria, which generally prefer an alkaline or neutral medium in which to develop. Alkaline meat has the reverse properties.

The measurement of pH is usually performed only 24 hours after slaughter when the pH of muscle from physiologically normal animals lies between the range 5.6—6.4 or less.

After the pH of meat has dropped to its lowest level of 5.4 (24—48 hours after death) the acidifying process is inactivated by enzymes which cause a gradual rise in pH with the onset of autolysis and bacterial development. Signs of decomposition become evident at pH 6.4—6.5.

Nitrazine yellow as indicator gives an immediate result over the pH range from 6—7. It is relatively insensitive to delicate measurement but has the advantage of presenting a clearcut change of colour in the zone pH 6.4—6.5 which is the *critical level* in respect of meat.

The determination of pH has a definite value in the judgment of fevered, injured, or sick animals presented for emergency slaughter. It must be stressed however, that to determine the pH of meat alone, without the ordinary meat inspection methods, is no criterion for judging the fitness of a carcase.

Method. The selection of muscle samples is governed by the need not to disfigure the carcase thereby reducing its economic value. A readymade specimen is however available with the normal routine triceps brachii cut for cysticercosis in the case of cattle and pigs, the psoas major in baconers and equines, and the rectus abdominis in sheep and calves.

Precautions are taken to obtain muscle specimens with little connective tissue and free from fat and blood. These are dissected out from the carcase on the slaughter floor.

* Johannesburg Municipal Abattoir.

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As it can be accepted that the process of glycolysis is only completed roughly 24 hours after slaughter, the pH determination should normally be performed 24 hours post mortem. This period is impractical in normal slaughterhouse operation, which requires a rapid turnover of the carcases, and a rapid field test based on Callows' ultimate pH method[1] has been devised as follows, to determine the ultimate pH of freshly killed carcases by the Nitrazine Yellow Test:

Ten ml. chloroform water (40 drops chloroform in 400 ml. distilled water) are added to approximately 10 grams of the minced freshly removed muscle sample in a Petri dish and well mixed. After an hour at room temperature all the glycogen is regarded as being converted to lactic acid. To prevent dilution of the indicator, the tissue fluid is pressed out of the meat sample, which is placed in a white plastic dish and the Nitrazine Yellow (1/10,000) solution is poured into it.

NITRAZINE pH RANGE AND CORRESPONDING COLOUR CHANGES.

pH.	*Colour.*
6.0	Bright yellow
6.2	Brownish yellow
6.4	Yellow greenish
6.5	Olive green—slightly turbid.
6.7	Violet.
6.8 and over	Blue Violet to Purple.

If the indicator remains yellowish or yellowish brown the meat is generally satisfactory (pH 6—6.3), but if it becomes olive green and turbid (pH 6.4) to violet or purple blue (pH 6.8) the meat is abnormal. Care should be taken when washing the utensils and instruments with tap water, which is often alkaline. Whenever possible rinse with distilled water.

Test Results. During the period November to May the following pH tests on casualty animals were carried out, as a guide to assessing the degree of muscle abnormality for purposes of judgment during secondary meat inspection. At the same time control tests were always carried out on normal muscle specimens (carcases passed as fit for human consumption).

Specimens giving a pH reading of below 6.4 were designated negative and those giving a reading of 6.5 and higher, positive.

TABLE I

Animal Species	Number of Casualty Samples.	Result of pH Test		Condemnations During Meat Inspection.
		Negative	Positive.	
A. Cattle.	82	19	63	51
B. Sheep.	248	5	243	233
C. Pigs.	70	25	45	39
D. Calves	29	1	28	28
E. Equines.	17	12	5	3
Totals.....	446	62	384	354

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Of the 446 casualty samples tested 384 were positive and of these 354 carcases were condemned. Condemnations were recorded as follows, on secondary meat inspection:—

A. *Cattle.*

(51 out of 63 positive pH tests)

Extensive Bruising	23
Emaciation	4
Fever	18
Gangrene	6
Total	51

B. *Sheep.*

(233 out of 243 positive pH tests)

Fever	177
Septic Pneumonia	9
Extensive Bruising	17
Gangrene	4
*Caseous Lymphadenitis	4
Jaundice	8
Septic Metritis	4
Moribund	8
Emaciation	2
Total	233

* Three of the four sheep carcases condemned for Caseous Lymphadenitis showed signs of fever.

C. *Pigs.*

(39 out of the 45 positive pH tests)

Enteritis	1
Fever	13
Extensive Bruising (Including Fractures)	13
Generalised Tuberculosis with Emaciation	1
Pyaemia	3
Peritonitis	2
Arthritis	2
Gangrene	4
Total	39

D. *Calves.*

(28 out of the 28 positive pH tests)

Enteritis	13
Moribund	8
Emaciation	7
Total	28

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E. *Equines.*

(3 out of the 5 positive pH tests)

Septicaemia 3

A number of carcases (12 cattle, 10 sheep, 6 pigs and 2 equines) giving a positive test result were passed on secondary examination. In these cases, although the pH of the meat was higher than normal, no sign of any pathological condition such as fever or bruising could be found for which the carcases could be condemned.

TABLE II

CONTROL TESTS CARRIED OUT ON NORMAL MEAT (CARCASES PASSED AS FIT FOR HUMAN CONSUMPTION).

Animal Species	Number of Tests.	Result of pH Test	
		Negative	Positive
A. Cattle	94	93	1
B. Sheep	454	454	—
C. Pigs	85	85	—
D. Calves.	12	12	—
E. Equines	27	27	—
Totals.	672	671	1

Out of a total of 672 normal samples tested only one positive result was registered, that in the case of cattle. A mistake may have been made in collection of the specimens, for on the same day two casualty samples were also tested, both giving negative results, but when fresh specimens from the same carcases were retested one positive test was recorded. The small number of positive tests shows that the pH testing can play a role in the evaluation of apparently healthy carcases.

TABLE III

SPECIAL TESTS

A. pH DETERMINATION ON MEAT OF CONDEMNED CATTLE.

Disease.	Number of Samples Tested.	Result of pH Test	
		Positive.	Negative.
Gangrene	4	3	1
Fever.	25	23	2
Emaciation	3	3	—
Extensive Bruising.	20	12	8
Measles.	5	—	5
Total	57	41	16

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B. pH Determination on meat of Condemned Calves.

Disease.	Number of Samples Tested.	Result of pH Test.	
		Positive.	Negative.
Enteritis	10	10	—
Moribund	8	8	—
Emaciation	7	6	1
Totals	25	24	1

C. pH Determination on meat of Condemned Sheep.

Disease.	Number of Samples Tested.	Result of pP Test.	
		Positive.	Negative.
Fever	45	45	—
Gangrene	3	2	1
Extensive Bruising	6	6	—
Septic Pneumonia	5	5	—
Septic Metritis	6	6	—
Emaciation	8	8	—
Jaundice	5	5	—
Totals	78	77	1

D. pH Determination on meat of Condemned Pigs.

Disease	Number of Samples Tested.	Result of pH Test.	
		Positive.	Negative.
Fever	8	7	1
Extensive Bruising	6	6	—
Peritonitis	4	4	—
Gangrene	5	4	1
Measles	6	—	6
Pneumonia	3	3	—
Totals	32	24	8

Discussion.

From the results in *Table* 1 it will be seen that the percentage of carcases showing inadequate acidity, (say a positive test of pH 6.5), is high (86%) especially in the case of sheep and calves.

The high pH frequently associated with feverish or septic conditions e.g. peritonitis, enteritis, gangrene etc., can be due to a depletion of glycogen. Because of fever the metabolic rate increases, producing heat and rapidly depleting the available glycogen reserves, which are not replaced as the animal does not feed. Toxaemia resulting from gangrenous wounds also leads to an increased metabolic rate to rid the body of the toxins. A rise in temperature of 1°F above normal may lead to an increased metabolic rate of up to 20%.

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In sheep a high percentage (73%) of the samples tested were detained for fever by the Inspectors doing the meat inspection, but during secondary examination it was found that a large number could have been condemned for ketosis, travel sickness or pregnancy toxaemia, the livers showing fatty degeneration and fat phanerosis (appearance of microscopic colloid fat in the liver cells).

In casualty animals which cannot be properly rested, fed and watered, because of the need for emergency slaughter, the glycogen reserve tends to be exhausted due to starvation and strain, and the pH therefore will be correspondingly high.

In the case of calves, of the 29 tests carried out 28 were positive all of which were condemned. Most of the calves were of a very young age and therefore unable to build up a substantial glycogen reserve.

Frequently in cattle and equines an indefinite test is encountered (yellowish brown colour). This may be explained by the fact that in both species the glycogen reserve remains high even when the ultimate pH has been attained. It must however be pointed out that in quite a large number of bovines and equines recorded as casualty stock, the injuries (e.g. fractures and extensive bruising) sustained where quite often of recent origin, whilst the animals were being off-loaded or penned, the glycogen reserves in these cases should therefore not have been appreciably affected by the shock of the injury.

Pigs often travel long distances without feed or water and as the muscle glycogen reserves normally tend to be relatively low, the animals are very sensitive to the effects of fasting. As shown in *Table I*, among pigs there was a high percentage of positive pH tests, a large number coincided with bacterial infections and septic conditions and the rest were probably due to metabolic disturbances.

Feverish, septic and gangrenous conditions usually gave a strong positive pH test and if accompanied by systemic changes an even higher pH was recorded.

In case of bruising the severity and other factors such as exhaustion, fever, affected the pH readings, (below 6.4 in moderate cases, usually above 6.4 in severe cases).

Tests carried out on terminal and immature foetuses gave negative results.

The nitrazine yellow test method offers several advantages and disadvantages.

Advantages.

1. Inexpensive.
2. Simple to apply.
3. Reasonably accurate—gives a clearcut change of colour in the zone pH 6.4—6.5 which is the critical level in respect of meat.
4. Muscle samples do not disfigure the carcase, therefore the marketability is not affected.

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Disadvantages

1. Time consuming (average test takes at least 1¼ hours).
2. Test itself not sufficient for condemnation; should only be a guide in meat inspection.
3. Washing of utensils and instrument with alkaline water may upset the test.
4. In certain species, with a high glycogen reserve (e.g. bovine and equine) the test can often give an indefinite reaction.

Conclusion: The pH reading of casualty animals appears to depend on the extent of metabolic disturbance—any condition affecting the organ chiefly involved in normal metabolism, i.e. the liver, is reflected in a marked positive reaction.

The Nitrazine Yellow pH test is a useful aid in secondary meat inspection of suspected carcases.

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THE SUSCEPTIBILITY OF THE AFRICAN BUSH PIG, *POTAMOCHOERUS PORCUS MASCHONA*, LONNBERG, TO INFECTION WITH *BABESIA TRAUTMANNI*

D. K. Shone and J. R. Philip,*

(*Received for publication 18th September* 1960)

Babesia trautmanni, as evidenced by infection of domestic pigs, has a wide distribution in Africa. According to Lawrence and Shone[1] cases have been reported from Tanganyika, the Congo, Southern Rhodesia and the Union of South Africa as also Italy, Sardinia, Russia and Bulgaria.

In Southern Rhodesia there has been a considerable increase in the number of cases diagnosed, in recent years, particularly in the Bindura area[2]. Heavy mortality in adult pigs is recorded from time to time before steps are taken by the farmer to obtain a diagnosis and institute treatment.

There do not appear to be any reports of the susceptibility of the African Bush Pig, *Potamochoerus porcus maschona*, Lonnberg, to infection with *Babesia trautmanni* and the role it may play in the dissemination and maintenance of the disease in the absence of domestic pigs. The opportunity was therefore taken, when two bush pigs became available to us, to investigate this aspect.

Experimental Work

A strain of *Babesia trautmanni* from a naturally occurring case in the Bindura area was transmitted to a 4 to 5 month old Landrace cross Large White pig by the subcutaneous injection of citrated blood. Several deaths had occurred in this outbreak.

Ticks collected from sows in this group were identified by Dr. G. Theiler, Onderstepoort, as numerous *Rhipicephalus simus* and one *Rhipicephalus appendiculatus*.

Behaviour of the strain in domestic pigs: Transmission in all cases was by subcutaneous injection of citrated blood drawn from the anterior vena cava. All pigs used were between 4 and 6 months of age and Landrace — Large White crosses.

The incubation period, i.e. the interval between the injection of infective blood and the onset of the febrile reaction, was 4, 5, 5 and 6 days. Piroplasms were however demonstrable in blood smears 1 or 2 days prior to the rise in temperature though the interval between infection and observation of the organism was not less than 3 days. Two of these pigs were splenectomised, and all four made natural recoveries. The febrile reaction lasted 1 or 2 days and the peak rectal temperature lay between 106°F and 107°F.

* Veterinary Research Laboratory, Salisbury, Southern Rhodesia.

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One pig was splenectomised on the 34th day after infection and relapsed to infection with *B. trautmanni*.

In general, invasion of the erythrocytes was very heavy and in some cases it was estimated that up to 50 per cent of the cells were parasitised.

Behaviour of the strain in bush pigs: Two bush pigs, a boar and a gilt, were available for this work. Their antecedents were not very well known, but it was considered that they were from the same litter and approximately 4 months old at the commencement of this work.

On the 12th March, 1960, 20 ml. of blood was drawn from the anterior vena cava of the bush pig boar and injected subcutaneously into a splenectomised domestic pig, V.R. 7. No infection resulted. This pig was from a group which had been established to be susceptible to infection with *Babesia trautmanni*.

Immediately following the above operation, 20 ml. of blood was drawn from a domestic pig, V.R. 1, and injected subcutaneously into the bush pig boar. This blood showed an estimated 50 per cent infection rate of the erythrocytes with *B. trautmanni*.

The rectal temperature of the bush pig boar was recorded twice daily for 16 days and blood smears prepared and examined daily for 9 days, commencing the day following infection. No parasites were observed in the blood smears. For the first week the temperature was comparatively high, 103°F to 104°F, but this is thought to be due to the fact that the animals were still very nervous at this stage and were difficult to catch. In subsequent periods the rectal temperature varied from between 100.4°F and 103°F.

On the 16th March, 1960, the fourth day after injection with infected blood, 20 ml. of blood was drawn from the anterior vena cava of the bush pig boar and injected subcutaneously into a splenectomised domestic pig, V.R. 5. Six days later *B. trautmanni* was demonstrated in blood smears and a febrile reaction with a maximum temperature of 106.6°F was recorded.

On the 20th March, 1960, 16 days after infection, the bush pig boar was splenectomised under pentobarbital sodium anaesthesia. The recording of rectal temperatures was resumed on the 2nd day after operation, and continued to the 10th day. Blood smears were prepared and examined daily on the 3rd to 10th days inclusive after operation and an occasional *B. trautmanni* was demonstrated on the 3rd, 5th and 8th days but not on the other days.

On the 3rd day after operation 20 ml. of blood was drawn from the anterior vena cava of the bush pig boar and injected subcutaneously into a splenectomised domestic pig, V.R. 6. *B. trautmanni* was demonstrated in blood smears of pig, V.R. 6, four days later. A febrile reaction was also recorded.

The bush pig gilt received 20 ml. of infected blood subcutaneously on the 31st March, 1960. Daily smears, prepared for the first 7 days, were negative on examination and no febrile reaction was recorded. The rectal temperature varied between 101.8°F and 103.8°F. On the 23rd day after infection 20 ml. of blood was drawn from the anterior vena cava and injected into a splenectomised domestic pig, V.R. 8. This pig did not become infected.

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Summary

1. The African bush pig *Potamochoerus porcus maschona*, Lonnberg, has been shown to be capable of harbouring *Babesia trautmanni* for 16 days, and for a further 8 days after splenectomy.
2. The infection of *B. trautmanni* in the bush pig is an inapparent one.
3. Ticks collected from domestic sows in the course of an outbreak of *Babesia trautmanni* infection were numerous *Rhipicephalus simus* and one *Rhipicephalus appendiculatus*.
4. The incubation period in domestic pigs following the subcutaneous injection of infected blood was 4 to 6 days.

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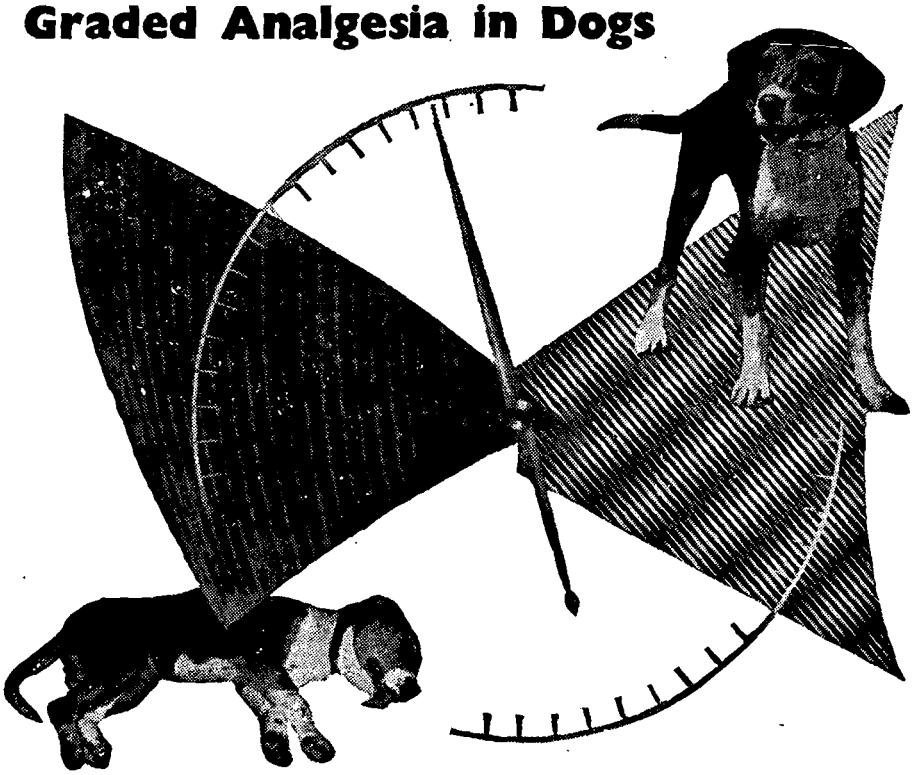
The technical assistance of Mr. B. W. Blair, is gratefully acknowledged and Mr. B. Brooks is thanked for making the bush pigs available.

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NOTE ON THE OCCURRENCE OF THE NEMATODE-*NEMATODIRUS HELVETIANUS*, IN SOUTH AFRICAN BOVINES.

R. J. Ortlepp*,

(*Received for publication* 19th *November* 1960)

In the Union of South Africa the prevalent, and up to now the only known species of the genus *Nematodirus* is the form known as *N. spathiger* (Raill., 1896); it has been found on many occasions in sheep and goats, especially in Karoo and adjoining semi-arid regions. It has also been found to be responsible for very heavy worm infestations in the Bontbok (*Damaliscus dorcas dorcas* Pallas, 1760 = D. *pygargus* Pallas 1767), in the Bontbok National Park, Bredasdorp, C.P. It has also been recovered from the Springbok (*Antidorcas marsupialis*) from Northern Transvaal and Richmond C.P. There is no record of its occurrence in S. African cattle or other wild antelopes. In fact in the Union the presence of other parasites of this genus, except in the abovenamed hosts, seemed up to now to be entirely absent. Specimens in the department's collection collected from sheep in 1925 and labelled *N. filicollis* proved on re-examination to belong to the species *N. spathiger*. Dr. R. J. Thomas, formerly of the Nooitgedacht experimental station, Ermelo, informed the writer that from an examination of larvae he suspects the presence of *N. filicollis* in sheep in the Mosselbay area. The writer has confirmed this suspicion from adult specimens obtained from this area.

Recently some round worms collected from a bovine in the Nottingham Road area, Natal, were sent by the local state veterinarian to the writer for identification. On first examination these appeared to belong to the species *N. filicollis* (Rud., 1802), but on closer examination it was found that although the spicules were very similar to those of *N. filicollis* they were appreciably longer; in addition, the dorsal bursal lobe was clearly demarcated from the lateral lobes, a condition which does not apply to *N. filicollis*, and the mature eggs *in utero* were also longer, the majority reaching a length of over 0.2 mm.

In the revision of the genus *Nematodirus*, Rajewskaja[2] described the 18 then known species; six of these species are recorded from domestic ruminants (sheep, goats and cattle). During the ensuing quarter century 11 additional species have been described of which five are from the above named ruminants. These five species differ from the Natal material either in their much longer spicules, or the shape of their distal spicular terminations or in the pointed nature of the female tail. Of the six older species from domestic ruminants only the species *N. filicollis*, *N. helvetianus*, May 1920, and *N. oiratianus* Rajewskaja, 1929, have straight and lancet- or spear-shaped distal terminations of their spicules; those of the first and last named are less than 1 mm.

* Section Helminthology, Onderstepoort.

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long, whereas those of *N. helvetianus* are longer and may reach a length of 1.25 mm. In the writer's material the length and shape of the spicules, shape of the dorsal bursal lobe, and size of the eggs, conform to those given or described for *N. helvetianus*. The writer thus has no hesitation in assigning his specimens to this species.

In the department's collection there is a single batch of *Nematodirus* worms collected from a calf in 1930 from the Albany district C.P.; these had been identified as *N. filicollis*. On re-examination it was found that these worms belonged to the same species as those from Natal, i.e. to the species *N. helvetianus*. If the hosts from which these two lots of worms were collected were bred in the areas where they succumbed, it shows that this parasite has a very wide distribution in the Union. Careful search for and examination of collected worms will probably reveal that the occurrence of this helminth is more general than is at present known.

N. helvetianus was first described in the U.S.A. from worms collected from cattle in Switzerland. Since then it has been found to be widely prevalent in the United States. It is also present in England and other European countries as well as in Asia. South Africa is the first country in the Southern hemisphere where it is also now known to occur.

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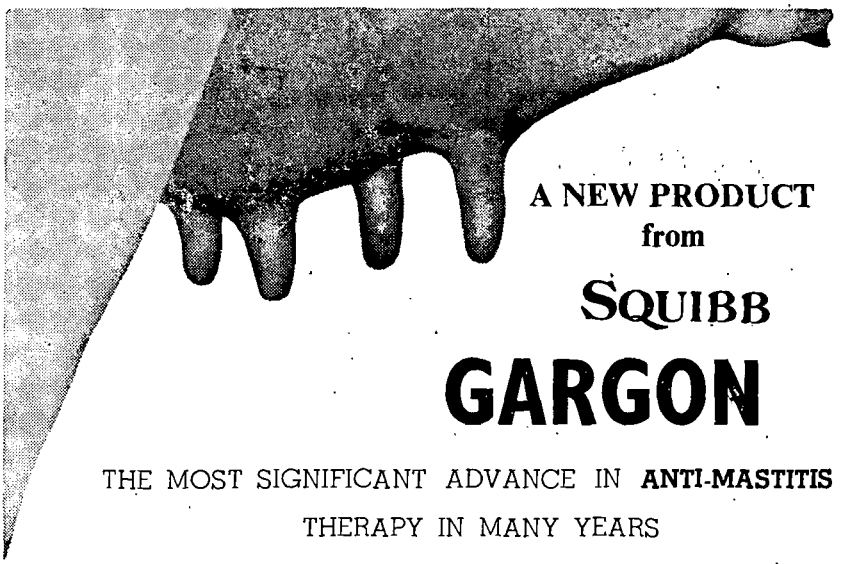
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MOVING THE BONTEBOK FROM BREDASDORP TO SWELLENDAM

K. v.d.Walt* and R. J. Ortlepp.**

Historical:

All the authorities seem to agree that in the early days of European settlement at the Cape the Bontebok, *Damaliscus dorcas dorcas* Pallas was found in an area between the Botriver in the West, the Gouritsriver in the East, the Langeberg mountains in the North and the sea in the South.

On the 11th Jan. 1689 Izak Schryver wrote: "We left the Swarte River (the present Caledon) before sunrise and passed good lands as before. On the way we saw more than 1,000 bonte bokken running".

Dr. A. Sparrman in 1775 made the following observation:

"I now for the first time, had the pleasure of seeing herds consisting of the two largest sorts of antilopes or gazels, which are called by the Dutch hartebeest and bunteboks."

Early travellers and writers like Patterson, Lichtenstein, Selous, and Barrow all referred to the Bontebok but it is clear from records left by Burchell, Harper, Thunberg, Stockenstrom, etc., that the Bontebok was often confused with the Blesbok, *Damaliscus dorcas phillipsi* Harper.

From the early writings it is also clear that the natural vegetation at that time was mainly waving grassland especially near the mountains where most of the Bontebok were found.

It is interesting to note that Thompson in 1823 already mentioned a prohibition against the shooting of Bontebok in order to prevent total extinction of the species. Cornwallis Harris (1839) recorded that a fine of 500 Rix dollars was attached to their destruction unless by special licence from the Government. The struggle for survival of this species therefore started at the beginning of the nineteenth century. Not only were their numbers decimated by shooting, but it appears that with progressive agricultural development of the fertile areas, the Bontebok was relentlessly pushed towards the bleak windswept coastal plains which were totally unsuitable from the point of view of food or rainfall.

Protected from shooting by families like the van Bredas, Albertyns, and van der Byls, never much greater than a hundred in number survived up to the present time. Unfortunately, the Bloubok, *Ozanna leucophaea* (Pallas), with a distribution, similar to that of the Bontebok, could not withstand the onslaught, and was the first species to become totally extinct in Southern Africa.

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Terrain of the Parks, past & present

In 1931 the National Parks Board took over and established the National Bontebok Park in Bredasdorp. Covering 800 morgen of flat, unhospitable, limestone area, with sparse and innutritious vegetation, the park proved inadequate for the propagation of the buck. A serious deficiency of copper and cobalt has been established in this region: in 1957 fifteen young animals succumbed to swayback. In the same year forty nine other animals died of parasitic infestation, mainly lungworm.

As a result of the above conditions, an area was acquired near Swellendam where the National Parks Board prepared a new home for the surviving Bontebok. It is interesting to note that the new park is located on the exact spot where the Bontebok was first and most frequently described in earlier days. The terrain is a basin-shaped area of about three thousand morgen covered mainly with Kweekgras (*Cynodon dactylon*) in the lower areas, and Rooigras (*Themeda triandra*) on the slopes and plateau. Seventeen hundred morgen of this area was acquired. The Southern boundary of the basin is formed by the picturesque Breederiver, the banks of which are well wooded, affording excellent protection for game. The park is situated about one mile from the town of Swellendam, on the town commonage.

Methods and procedure: Following a report of successful immobilisation of various species of wild animals in SouthWest Africa by the addition of chloral hydrate and sodium phenobarbitone to the drinking water, it was decided to investigate this method.

Mr. P. J. Barnard, biologist of the National Parks in the Cape Province, did some preliminary experiments with the addition of drugs to drinking water and reported the following reasons for failure:—

(1) It appears that about half of these animals are able either to exist without water or do not drink at the regular water supply points.
(2) Animals which do come to water drink only two to three pints of water, with the result that an effective concentration of chloral hydrate would render the water unpalatable.
(3) The animals drink at irregular intervals and stay away from water for days during cool weather.
(4) Lactating ewes drink more that the other animals in the herd which makes safe dosage-control impossible.

In consultation with the game ranger, Mr. Barnard therefore decided to erect small camps into which the game could be driven for catching. Each of these camps consisted of a triangular area of about two morgen leading into a narrow race about eighteen inches wide and ten yards long. The sides of the race were constructed of a three inch wire mesh fence, with a collapsible wire gate at the end of the crush.

The buck were driven into the small camps in lots numbering from eight to twenty, and on entry, long collapsible wire gates were closed behind them. Three or four motor vehicles were used for driving.

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A number of catchers hid themselves next to the crush, which was readily entered by the buck. On entering the crush the front gate was closed momentarily for the helpers to come up to the front; catching took place by quickly pulling out the animals by their horns and getting them onto their sides on the ground with as little struggling as possible.

Sedation

Immediately after securing the animals each one was injected intravenously with a 5 per cent solution of chlorpromazine hydrochloride ("Largactil"). All the adult animals were treated in this way. The weights varied considerably, because some animals were in extremely poor condition, but the average weight was estimated at about 70 lbs for ewes and up to 120 lbs for fullgrown rams in good condition. Lambs and halfgrown animals were not treated, mainly because they were not regarded as being a danger to themselves or other animals, when being transported.

Various dosage levels were tried, but it was soon apparent that an intravenous dose of 150 mg. (3c.c.) for a fullgrown ram had the desired effect. The dosage rate was scaled down for lighter animals.

The animals relaxed completely within about four minutes after the injection and stopped struggling. Within about ten minutes the treated animals were observed to go down, lying on their sternums with their noses on the ground, their heads often placed sideways. Some animals were inclined to roll over on their sides, and in these cases all efforts were made to keep them in a sternal position, to prevent bloat and choking by their own saliva. Profuse salivation was observed in the more heavily sedated animals, probably due to a suppression of the swallowing reflex, but no difficulty was encountered with bloat, provided the animals were not allowed to remain on their sides.

The effect and duration of sedation varied: three types could be discerned. In the first type buck were able to stay on their feet with some difficulty, but did not appear apprehensive. When let loose they were able to run away without difficulty and appeared normal. In the second type the buck were inclined to lie down when left alone, and tended to stay down for four to six hours if left undisturbed. When roused they were able to run away shakily. Some were observed to lie down after a few hundred yards, while others quickly regained normality. The third group consisted mainly of old thin animals of which some were pregnant ewes. These animals became comatosed soon after the injection and were inclined to go over on their sides. They could not be roused and stayed down for periods up to twenty-four hours.

Transportation: The buck were transported about sixty miles to the new locality in a covered vehicle (converted schoolbus). The interior was quite dark. The animals were loaded on to thick straw and bedded down next to one another in a sternal position. Attendants with the animals prevented them from lying on their sides and injuring one another. It was observed that the animals which were still able to stand, lay down soon after the vehicle started moving.

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The journey took about two to two and a half hours, and the buck were unloaded and released immediately on arrival at their destination. Those which could not be roused, were left unmolested in the open.

Results:

Of the eighty four animals originally captured, sixty one survived the critical first few days in the park, and it can be assumed that they are successfully established in the new environment. A detailed analysis of the cause of deaths is not possible as there were several contributory factors.

A ewe had to be destroyed on account of a fractured femur, but as this animal was found in the veld, the cause of the injury remains unknown.

Four animals were found dead in the water of the Breederiver, after their release at a point about a mile from there. It must be assumed that they died of over indulgence, having gone without water for some days, and having subsequently been chased.

Two out of six half-tame young Red Hartebeest *Alcelaphus buselaphus* Pallas, which were released in the park at the same time as the Bontebok, were subsequently found dead from what appeared to be prussic acid poisoning. It was surmised that some of the Bontebok had died of the same cause, as they had been found dead in the veld.

About eight of the animals arrived dead at their destination. Post mortem examinations were performed on most of them. Without exception these animals showed severe lungworm infestation, but the outstanding symptom was gross endocardial haemorrhages, ascribed to over exhaustion. The lesions appeared to be so severe that they could not have survived.

A few of the animals were unloaded in a comatosed condition and could not be roused. These animals died within the next twenty-four hours without moving, and it must be assumed that the main cause was over sedation. They were, however, exceptional animals in poor condition: some were heavily pregnant. Auscultation in some of these cases revealed marked conduction disturbances in the heart which were probably caused by endocardial haemorrhages.

Discussion

When judging the results of the operation the following factors should be borne in mind.

(1) All the animals in the herd including young, old, pregnant and thin animals were rounded up and captured without any selection.
(2) As described previously important reasons for moving the animals were severe parasitic infestation and malnutrition.
(3) The administration and dosage rate of the traquilliser was on an empirical basis, as no previous information was available.

The first eighteen animals were dosed with phenothiazine after sedation. This was accomplished by using a rubber extension tube on a dosing syringe, passed into the oesophagus. Each adult received two

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ounces of a watery suspension of microfine phenothiazine containing 10 grams of the remedy per ounce; younger animals according to age and size received the equivalent of 10 or 15 grams phenothiazine. The animals were dosed in a kneeling position with the head held horizontally. No difficulty was encountered in passing the tube down the gullet and no untoward symptoms were observed after dosing. Examination of the three animals which died *en route* to the new park showed that no phenothiazine had passed into the lungs. After it was established at post mortem that they were suffering mainly from parasites (lungworms and conical flukes) not susceptible to phenothiazine, dosing was stopped. The actual administration of a worm remedy after sedation is however quite a feasible procedure. Unfortunately no '*Dictycide*' was available. This treatment seemed to be indicated for the lungworm infestation.

On the whole, sedation by means of intravenous injection of chlorpromazine hydrochloride was remarkably effective at the dosage rate used. After this experience, however, it was considered advisable not to exceed 1 mg. per pound live weight in order to prevent oversedation. Finally, attention should be drawn to the rôle played by prevailing weather conditions when wild animals are being captured. Cool, overcast conditions on the last days of March 1960, when this operation took place, undoubtedly contributed much to its success.

ACKNOWLEDGEMENTS

Opportunity is taken of expressing our appreciation to the Director of Veterinary Services for sanctioning participation in this undertaking and allowing publication of the results; also to Mr. Rocco Knobel, Director of the National Parks Board, and Messrs R. J. Labuschagne and P. J. Barnard, professional officers on his staff, for inviting us to the expedition and putting the relevant publications and information at our disposal.

SUMMARY

An entire herd of Bontebok, Damaliscus dorcas dorcas *Pallas, was transferred from the District of Bredasdorp (Western Cape Province) on account of malnutrition and parasitic infestation.*

The historical background, capture, sedation with chlorpromazine hydrochloride, and transportation is described . Twenty-three animals out of a total of eighty-five were lost . The reasons for this are discussed.

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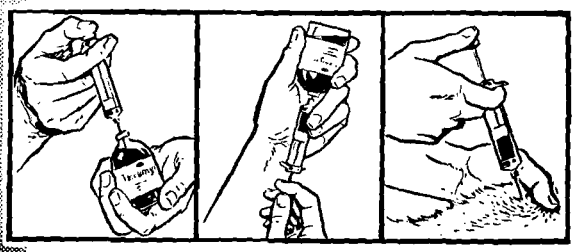
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A NOTE ON THE INCIDENCE OF PORCINE TUBERCULOSIS IN SOUTH AFRICA

S. J. van Rensburg* and F. B. W. Du Casse**

(*Received for publication* 18*th January* 1961)

The prevalence of tuberculosis in pigs in South Africa was noted by Robinson[1], who drew attention to its possible significance when a campaign against the disease in cattle was undertaken. However, no consecutive study of the incidence of tuberculosis-like lesions and their relative occurrence in the body appears to have been recorded in this country.

The substance of this report is based on findings in baconers slaughtered at the Estcourt Bacon Factory. As the pigs originated from widely separated areas in Natal, the Transvaal, and the Orange Free State, they were considered to constitute a representative sample of the country's baconer population,

Incidence of lesions resembling Tuberculosis.

A total of 13,000 pigs were consecutively surveyed during 1959. Examinations of the head, mesenteric and bronchial lymphatic nodes were recorded. Lesions localized in one site in the carcass were observed in 7.4 per cent of the animals (Table 1).

TABLE 1.
OCCURRENCE OF LOCALIZED LESIONS IN 13,000 PIGS.

Head lymphatic nodes	447 — 3.69%	7.14%
Mesenteric lymphatic nodes	432 — 3.32%	
Bronchial lymphatic nodes	19 — 0.15%	

Little difference existed between the incidence in head and mesenteric nodes. The percentage of carcasses condemned for multiple lesions was 0.52 (Table 2), the head and mesenteric nodes being afflicted in the majority of these animals. Therefore, out of 13,000 baconers, a total of 995 (7.66 per cent) were found to have lesions macroscopically suspected of being tuberculous.

TABLE 2.
OCCURRENCE OF MULTIPLE LESIONS IN 13,000 PIGS.

Head and mesenteric lymphatic nodes	56 — 0.431%	0.516%
Head and mesenteric and bronchial lymphatic nodes	7 — 0.054%	
Head and bronchial lymphatic nodes	3 — 0.023%	
Mesenteric and bronchial lymphatic nodes	1 — 0.008%	

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** Division of Veterinary Services, Estcourt.

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Examination of lesions for Acid-fast bacilli.

Ziehl-Neelsen and Giemsa stained smears, made from tuberculosis-like lesions in the lymphatic nodes, were each searched for approximately ten minutes for acid-fast bacilli and other bacteria. Head, mesenteric and bronchial lymphatic nodes, collected consecutively, both from carcasses with localized and multiple infection, were examined (Table 3).

TABLE 3.
MICROSCOPIC EXAMINATION OF SMEARS OF LESIONS.

Lymphatic. Node.	No.	Acid-fast. bacilli *	Non-acid-fast Bacteria.	Negative.
Mesenteric	45	69%	13%	18%
Head.....	165	18%	53%	29%
Bronchial.	19	16%	68%	16%

* Mixed infections with non-acid-fast bacteria in 11% mesenteric, 2% head, and 5% bronchial nodes.

In the survey of lesions resembling tuberculosis, 67 of the 13,000 pigs were found to have multiple lesions, and in each case the whole carcass was condemned in order to conform to existing regulations. Smears of lesions in the lymphatic nodes of the head were made from all these condemned pigs (Table 4), except in the instance where one animal had no lesions in the lymphatic nodes of the head, when the bronchial node was examined. Non-acid-fast organisms appeared mainly to resemble corynebacteria, which rarely tended to be acid-fast. Streptococci, staphylococci and non-acid-fast bacilli were also encountered.

TABLE 4.
SMEAR EXAMINATION OF HEAD NODES FROM PIGS WITH MULTIPLE LESIONS.

Number	Acid-fast bacilli *	Non-acid-fast bacteria	Negative
67	57%	25%	18%

* Mixed infection with non-acid-fast bacteria in 5%.

Macroscopic Pathology of Lesions

Correlation of the presence of acid-fast bacilli and other bacteria in lesions, indicated that colour, consistency, and the presence of calcification in affected nodes, were unreliable criteria for distinguishing tuberculous from non-tuberculous nodes. Only 4 carcasses, having tuberculosis-like lesions of one gland, had what was considered to be typical corynebacterial abscesses of another gland, and were not classified as "generalised". These abscesses were in the sub-parotid nodes and, microscopically, appeared to be due to corynebacteria.

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Small lesions were commonly encountered; their detection in the course of routine inspection presenting considerable difficulty. Approximately 40 per cent of the pigs having lesions in more than one situation in this survey, had single to multiple small lesions (1-2 mm.) of the head lymphatic nodes; the majority having a soft to dry caseous consistency. Acid-fast bacilli were most consistently found in small and in large extensive lesions.

Non-acid-fast organisms invariably occurred in lesions of intermediate size (approximately 5 mm.), except on two occasions when they were found in small lesions. Small lesions were also commonly found in the mesenteric node.

DISCUSSION

Robinson[1,2] reports that in the great majority of cases lesions are confined to the lymphatic nodes of the head, with only occasional ones in a mesenteric. In this survey we noted an almost equal incidence of lesions in head and mesenteric nodes. Acid-fast bacilli, however, were demonstrable in 69 per cent of the mesenteric nodes, and in only 18 per cent of the head nodes. When lesions were not confined solely to the head, acid-fasts were demonstrable in 57 per cent head nodes. Kleeberg[3], however, examined 55 specimens of tuberculosis-like lesions confined to the lymphatic nodes of the head, microscropically, bacteriologically, biologically, and histologically, and found 82 per cent undoubtedly tuberculous.

In this survey, pigs originating from several piggeries with the highest incidence of tuberculosis, not uncommonly had mesenteric node lesions in excess of 50 per cent, while only occasionally were more than 10 per cent of the heads affected. These results are similar to the experimental infection experiments of Robinson[1,2] who failed to produce infection limited to the lymphatic nodes of the head, and confirm his suspicion that, if mesenteric glands are examined carefully, more lesions may be found.

A striking divergence exists between the results of this survey and official statistics of other abattoirs in the country. For 1959, the percentage of pigs detected with multiple lesions was 0.094, whereas the figure in this survey conducted by veterinarians was 0.516 per cent. A similar observation was made by Kleeberg[4] regarding tuberculosis in cattle. He necropsied 110 tuberculin-positive reactors and found lesions in 97 per cent. Thirty other reacting cattle in the same experiment were sent to two different abattoirs but in only four of them (13 per cent) were lesions of tuberculosis detected. It can therefore, be concluded that abattoir statistics regarding the occurrence of tuberculosis in South Africa do not reflect a true picture of the incidence.

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SUMMARY

A consecutive survey of the total and relative incidence of tuberculosis-like lesions in 13,000 baconers revealed that 7.66 per cent were afflicted. Evidence that mesenteric nodes are as frequently involved as head nodes is presented. The bearing which the high frequency of mesenteric tuberculosis and small lesions in baconers has on problems of meat inspection, is emphasised.

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WHITE MUSCLE DISEASE IN AN ANGORA GOAT.

R. C. Tustin.*

(*Received for publication 18th November* 1960) .

A diagnosis of white muscle disease was recently made at autopsy of a ten day old female Angora goat kid. Typical macroscopic and microscopic lesions of the disease were observed in the majority of skeletal muscles and a white sub-endocardial plaque was present in the left ventricle. The appearance of the lesions suggested that the animal had shown symptoms for at least four or five days before death.

The dam was one of a batch of Angora goat ewes which had arrived from the Karoo 16 months previously. During this period they were kept in a small camp in which no natural grazing was available and were fed a concentrate ration consisting of maize cob meal 90 per cent, blood meal 6 per cent, bone meal 2 per cent, salt 1 per cent and urea 1 per cent. Each animal received ½ to 1 lb. of the mixture and approximately 2½ bls. of lucerne hay per day. Fresh lucerne was occasionally fed to the animals. The lucerne hay fed on the station originates mainly from the Upington area or the Vaalhartz irrigation scheme.

White muscle disease was also diagnosed in a two and a half month old Merino lamb whose dam during pregnancy and lactation received the same ration as the goats. The lamb and the kid died within a period of three weeks of each other.

ACKNOWLEDGEMENT

The Director of Veterinary Services is thanked for permission to publish this article.

REFERENCE

Tustin, R. C., (1959): *J.S.A. Vet. Med. Ass.* Vol. 30, pp. 451.

* Section of Pathology, Division of Veterinary Services, Onderstepoort.

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COMPARATIVE DENTAL PATHOLOGY* (WITH PARTICULAR REFERENCE TO CARIES AND PARADONTAL DISEASE IN THE HORSE AND THE DOG.)

C. F. B. Hofmeyr**

(*Received for publication 12th September* 1960)

As the title of this paper embraces such a wide field as to make it impossible to present coherent information in a limited space of time, unless the material is handled so superficially as to be without any value, it was decided to concentrate largely on two species—the horses and the dog, with only odd references to other species. More attention will be given to those conditions which are of especial importance in human dentistry—caries and paradontal disease.

For the purpose of orientation it will be necessary to consider a few anatomical and physiological features particularly of the permanent dentition of the two species named.

(1) The Horse.

The formula for the *permanent teeth* is:—

$$2(I\,\frac{3}{3}\;C\,\frac{1}{1}\;P\,\frac{3 \text{ or } 4}{3}\;M\,\frac{3}{3}) = 40 \text{ or } 42.$$

Deciduous teeth:

$$2(Di\;\; Dc\;^{1}\; Dp\;\tfrac{3}{3}) = 24.$$

The permanent teeth erupt as follows:—,

I	—	$2\frac{1}{2}$ years
I2	—	$3\frac{1}{2}$ years
I 3	—	$4\frac{1}{2}$ years
C	—	4-5 years
P 1	—	5-6 months
P 2	—	$2\frac{1}{2}$ years
P 3	—	3 years
P 4	—	4 years
M 1	—	10-12 months
M 2	—	2 years
M 3	—	$3\frac{1}{2}$-4 years.—

This table refers to the upper and lower jaw except P3 and 4 which may erupt 6 months earlier in the mandible.

Equine teeth have a unique feature i.e. the infundibulum, which is a deep invagination of all the layers in the occlusal surface of the tooth

* Address delivered before the Odontological Society of South Africa (Johannesburg Branch) at the Dental School, Witwatersrand University on 27 July, 1960. Published by kind permission of the Director of Veterinary services.

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and is filled with cement. As will be indicated later, the pattern varies in the different teeth but remains basically the same in all.

The teeth of equine animals are long crowned i.e. they grow out to compensate for wear. That is why they are deeply embedded in young animals and very shallow in the very old. The incisors at 5 years of age may be about 7 cm. long and at twenty or older only short stubs.

In passing it may be mentioned that in ruminants the cheek teeth are also long crowned but the incisors (only in the lower jaw) are not. When the crown wears away with increasing age the gum recedes so that the root projects to permit prehension. In rodents, again, the cheek teeth are fairly short crowned, but the incisors, particularly in certain species, are tremendously deeply embedded, the whole tooth forming a perfect segment of a circle and extending from the symphysis of the mandible to the temporo-mandibular joint. In the pig family again all the teeth are short crowned except the canines—that is why old boars have such long tusks. As a further interesting point, the lower canines of the pig have enamel on the outside only. The softer cement and dentine on the lower canines wear away against the upper canines thus maintaining a razor sharp edge on the lower canines.

To revert to the equine: the incisors are flattened from the labial to the lingual side, and curved. The apex is flattened in the opposite direction. This is why increased wear shows alteration of the shape of the tooth. Instead of an enamel cap, the infundibulum is present as referred to before. There is a single pulp cavity.

A cheek tooth is basically more or less square viewed from the occlusal surface. It resembles a rectangular block as seen from the side and it is more or less curved posteriorly depending on the particular tooth.

The occlusal surface is oblique, sloping outwards and downwards from the lingual side. The infundibulum is double and forms an intricate pattern resembling the gothic letter "B". The tooth has root cavities of involved pattern. The roots of the molars and sometimes half the last premolars project into the maxilary sinus which is well developed in the equine. The mandibular cheek teeth resemble the maxillary cheek teeth except that they are narrower from side to side and show only half the "B" although they have two infundibula and pulp cavities. Their occlusal surfaces slope downwards and outwards from the lingual side.

The basic structure of all equine teeth from without inwards to the centre of the infundibulum is cement, enamel, dentine, enamel, cement.

The incisors of the horse are used for prehension of food, and the cheek teeth are grinders with a side-to-side movement.

An important feature is that the approximate surfaces are in very firm contact, forming a continuous table. The gum also very closely and firmly invests the tooth.

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(2) The Dog.

The formula for the permanent dentition is:

$2(I\ \frac{3}{3}\ C\ \frac{1}{1}\ P\ \frac{4}{4}\ M\ \frac{2}{3}) = 42.$

The permanent teeth erupt as follows:—

I 1
I 2
I 3
C
} 4-5 months.

P 1
P 2
P 3
P 4
} 5-6 months.

M 1—4 months
M 2—upper 5-6 months.
lower 4½-5 months.
M 3—6-7 months.

All the teeth have short crowns, and the conventional enamel cap. The root is covered with cement and just below the gum line the cement is thinnest.

All the crowns (except those of the canines) are tridenticulate.

As indicated, the cheek teeth are normally $\frac{6}{7}$ but in brachycephalic breeds may be $\frac{5}{7}$ or even $\frac{4}{5}$, reduction occurring at either end of the buccal arcade. P4 above and M1 are much larger than the rest and are called carnassial or sectorial teeth. The teeth behind the carnassial teeth are more tuberculate. Where the incisors are used for prehension and the canines for tearing, the cheek teeth are used for cutting like scissors except the tuberculate ones which are employed in crushing (bones etc.).

With this background, dental pathology can now be considered. The term is used here in the restricted meaning of disease of the tooth itself and not in its wider connotation of oral pathology.

It can be classified as follows:

(1) *Congenital and Developmental Abnormalities.*

Oligodontia, polydontia, macrodontia, prognathism, brachygnathism, rotated teeth, misplaced teeth, dentigerous cysts, odontomata etc.

(2) *Trauma*: Fracture of tooth or alveolus, abnormal wear due to pathology elsewhere in the mouth or due to bad habits etc.

(3) *Chemical injury*:

(4) *Neoplasia*: Adamantinoma etc.

(5) Changes due to inflammation of dental and peridental tissues or to specific diseases.

It is proposed to centre the attention more particularly on item (5).

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5.1 *Dental carries*:

5.1.1. *The Horse*: This is almost never seen in the incisors. It is a condition which may start at one of two primary sites.

5.1.1.1. On the occlusal surfaces.

5.1.1.2. On the buccal, lingual or approximate surfaces.

5.1.1.1. The first upper molar is most often affected (in nearly 50 per cent of cases), then upper M2 and PM4 in this order, then comes M1 of the lower arcade and the PM3 and M3 (upper) and the other cheek teeth at the bottom more or less equally. This condition is not uncommon.

The caries starts in the anterior or posterior cement lacunae on the occlusal surfaces where there may be small canals which are actually defects. When feed mixed with sand or particles of metal is ground between the teeth, damage may also be inflicted on the cement, forming hollows where food stagnation may occur. This is particularly likely to happen when cereals are masticated, as the lower cheek teeth have a side to side grinding movement against the upper teeth and as the complex "B" of the upper teeth readily accommodates feed particles, it can be understood why the upper cheek teeth are more vulnerable to caries than the lower. The reason why the upper M1 is more commonly affected than any other tooth may be explained by the fact that it erupted a long time before the other teeth in the upper buccal arcade and is situated in part of the arcade most used for grinding of feed. The latter reason is also a feasible one to explain the incrimination of the other most commonly affected teeth (i.e. those on either side of M1 (upper)).

The caries is not a rapidly progressive condition. There may be attempts by the body to wall off the diseased process by laying down of cement. Even so the process proceeds until eventually the pulp cavities are exposed. There is usually surprisingly little pain associated with the caries and quite often one may find merely a shell of the tooth or a few fragments still in situ, without spectacular symptoms associated with the teeth.

The pulpitis is progressive and eventually the periodontal membrane is affected. The disease then usually proceeds to assume the characteristics of paradontal disease which will be discussed further under this heading.

5.1.1.4. The second form of caries starts on the lingual, buccal or approximate surface of the cheek teeth. Here they are covered with cement. The occurrence is associated with some form of trauma due to sand, grit or metal in the feed. This causes a chipping of the enamel which in turn provides a depression in which particulate matter can collect and ferment. The rest of the development is similar to caries of the occlusal surfaces.

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Up to now active aetiological factors have been considered. Less clearly defined but important are the predisposing causes—teeth that are structurally poor due to nutritional and disease factors or from genetic causes.

Comparatively young horses are sometimes seen with the cement and the enamel on the labial surface of the incisors worn away. Vices like crib-biting lead to an abnormal wear of the teeth but then usually in the vicinity of the occlusal surface. The condition mentioned is regarded as being due to structurally poor teeth—a weakness often revealed by the additional wear associated with close grazing on sandy pastures. Despite the damage to the dental tissues of the incisors, caries does not supervene—a clear identification that a combination of factors is necessary for caries to manifest itself, as is the case in the cheek teeth.

5.1.2. *The Dog*: Dental caries is fairly rare in this species. It occurs more often in dolichocephalic than in brachycephalic breeds, the comparative freedom in the latter being because of abnormal occlusion. To elaborate this point—as the opposing molar teeth do not occlude properly, there is much less tendency in the case of short headed breeds, as compared with long headed breeds, for cereal food to be impacted on these occlusal surfaces. Further explanation is given later. The predilection sites are the upper molars. The lower molars are seldom affected. It is very rare for caries to show itself on the inside, outside or approximate surfaces of any tooth.

As indicated under the discussion on anatomy, the function of the cheek teeth differs. The denticulate premolars have a cutting function like scissors. Food particles do not readily adhere to their sharp surfaces. The molars are used for crushing and have tuberculate occlusal surfaces. The normal food being meat and bones, there is not much chance for adherence to the teeth. Where soft cereals are fed there is no need for attempting to crush the food and again food particules do not tend to become impacted on the molars. With the advent of dog biscuits, however, the crushing function of the molars has to be brought into action and the cereal readily attaches to these occlusal surfaces. It is strongly surmised that cereal biscuits are generally to blame for dental caries in the dog.

In the dog abnormal attrition of the teeth does not lead to caries. It is a common sight to see dogs with grossly worn teeth as a result of playing with a tennis ball or stones—yet caries remains absent. The same applies to dogs with the labial surface of the front teeth worn due to incessant biting of a chronic dermal pruritis. This is seen especially in long, coarse-coated dogs. These remarks also apply to those cases with prematurely worn teeth due to excessive gnawing of bones—a practice encouraged to unintelligent extremes by most dog owners.

5.2. *Paradontal Disease.*

5.2.1. *The Horse*: Paradontal disease can be called the dental scourge of the equine family. As indicated previously the mucoperiosteum fits very snugly around the teeth into the interproximal areas.

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The first foreboding of this disease is destruction of the mucoperiosteum at its dental border. This arises as a rule from some injury which could be brought about by grass, thorns, or very rough feed, sharp foreign particles in the feed or even by ordinary hay which is often cut into short lengths with a cutter. Such hay can more easily cause injury to the mouth than long pieces which are gradually masticated and softened by the saliva.

As soon as separation of tooth and mucoperiosteum has taken place, a small more or less triangular cavity is present and traps feed, debris and grain particles. Due to bacterial action and fermentation there is progressive infection of the periodontal membrane and a progressive disease of the alveolar wall. The tendency of the infection is to spread away from the tooth. This is limited by a degree of the density of the bone. The bone of the maxilla is therefore affected more readily than that of the mandible.

The supporting structures of the tooth are gradually destroyed until the tooth becomes loose in the alveolus.

In the case of the upper cheek teeth the infection may break through into either nasal cavity, or empyaema of the maxillary sinus may result—the roots of the molars and often half of the last premolar being separated from the sinus by a thin layer of bone only. Eventually, through force of gravity the tooth is inclined to come away unless professional help has been given before.

In the case of the mandibular teeth gravity keeps them in place. The infective process burrows deeper, often resulting in tooth fistulae discharging at the ventral mandibular border.

It is patent that a horse with diseased teeth is unable to masticate the feed properly. This in turn usually brings a train of digestive disorders in its wake. In addition chronic sepsis may cause arthritis, synovitis, neuritis etc.

Caries on the occlusal surface, as indicated before, very often ends up in paradontal disease with a pathological picture similar to that just described, the difference being a simultaneous, gradual disintegration of the tooth.

In young horses there are conditions of the alveolus that must be referred to, although the pathology differs from that of paradontal disease.

Chronic ossifying alveolar periostitis may be seen in horses from one to five years, although the greatest incidence is among those from two to a little over three years old. The tooth most commonly concerned is the third large upper premolar although the second is sometimes involved. It is manifested by a swelling of the bone over the root of the tooth and resembles osteofibrosis except that it is mostly unilateral. If bilateral, it is usually not symmetrical and the swelling is not as extensive as in osteofibrosis.

The inflammation is low grade and not purulent. The root of the tooth may be distorted or shell-like as the nutrition to the tooth has been hampered or destroyed. An indication of the exact aetiology is difficult, but there may be an associated impaction of the corresponding developing permanent premolars.

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Another form of alveolar periostitis of young horses occurs especially in the lower P1, P2 and M1. It occurs particularly in those animals 2 to 4 years of age and seems more common amongst the lighter breeds. This condition is recognised by swelling over the roots of affected teeth at the ventral border of the mandible. Fistulae may be present at these sites. Where no fistulae occur, the swellings disappear in horses towards 6—8 years. These enlargements are formed by dental sacs at the roots and are associated with overcrowding of the teeth, which has a genetic basis.

It will be noted, of course, that the swellings make their appearance at a time when the relevant permanent teeth are about to erupt.

Treatment is not within the scope of this paper. In view of the profound influence, however, of exodontia on the function of the remaining teeth and because of the new train of pathological processes that may be started by this operation or by any dental pathology, this is a convenient moment to consider some aspects of dental physiology and the consequences of malfunction.

As stated at the beginning, equine teeth are long crowned and grow out continuously to compensate for normal wear. That means that each tooth is worn down by its counterpart in the opposing jaw. Whenever then a tooth is absent through removal by surgery or disease the opposite tooth is no longer subjected to normal wear and elongates eventually damaging the gum and the jaw at the interdental space. Where local pain discourages mastication at the site, jaw movements may become restricted or distorted. This is reflected in the dental wear. The buccal arcades may thus present various types of deviating appearances as follows:—

(1) Undulating or wavy mouth—a description of the appearance of the occlusal surfaces.

(2) Stair- or stepmouth—cheek teeth of unequal height arranged like steps. This is liable to occur after pathology in several teeth.

(3) Shear mouth (parvignathism). If the lateral movement of the mandible is restricted (usually due to dental pain), then this condition is produced. It is recalled that earlier on it had been stated that the maxillary teeth are longer on the buccal than on the labial side. The madibular teeth are the reverse and are also, in addition, somewhat narrower than the top teeth. If proper side to side movement does not take place it means that the outside of the maxillary teeth and the inside of the mandibular teeth (as a rule on one side only) grow out, causing a scissors effect.

In addition to what has been said above concerning the causes of various dental conditions, heredity, nutrition and intercurrent disease would decidedly influence the liability of an equine animal to develop dental pathology. Unfortunately it is quite impossible to allocate the exact responsibility that each might bear in diseases of the teeth. It will, indeed, be an extremely complicated study to clarify the possible rôle of each in predisposing to dental disease.

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Up to this point no mention has been made of dental tartar in the horse as a cause of disease. In this species the rôle is not a prominent one and is in sharp contrast to the dog where it is of extreme importance.

5.2.2. *Paradontal Disease in the Dog.*

As dental tartar or calculus is an invariable precursor of this condition, these accretions are to be considered first.

5.2.2.1. *Dental Tartar.*

There are two types:

5.2.2.1.1. Salivary calculus.

5.2.2.1.2. Serumal calculus.

5.2.2.1.1. *Salivary calculus* consists of calcium phosphate and carbonate held together by a compound calculus agglutin'n which is formed after the tartar attaches to the teeth. This is soft and grayish white initially but alters into a white and a clear substance. The latter becomes caseous, breaks up and disappears while the former forms primary calculi which in time discolour due to attached particles and products of putrefaction and gingivitis.

5.2.2.1.2. *Serumal calculus.* Arises from and attaches below the gingival margin. Serum oozes out and forms calcoglobulin which changes and leaves the serumal calculus which is harder than salivary calculus. The colour is brownish black or black.

These two processes are not to be regarded as entirely distinct. The process is commonly mixed.

There are various conditions which encourage and facilitate the formation of tartar:

(*a*) *Oral disease.*

(i) Vincent's gingivitis caused by *Spirochaeta vincenti* and *Fusiformis bacillus.*

(ii) Leptospira stomatitis.

(*b*) *Debilitating disease* (like distemper or nephritis etc.) This often leads to a softening of the gums, some drawing away from and furring of the teeth—*foetor ex ore.*

(*c*) *Deficiency of Certain Vitamins*: Here Vitamin A is very important as it is associated with the integrity of mucous membranes The deficiency of Vitamin A causes hyperplasia of the subgingival epithelium and predisposes to subsequent bacterial invasion. Vitamin D deficiency causes defects in alveolar bone and aids invasion by micro-organisms. In a less direct manner Vitamin B_2 (nicotinamide) plays a rôle,

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particularly at the stage where the deficiency is so severe as to cause black tongue. As the dog elaborates Vitamin C in its digestive tract, its deficiency is not often manifested and when it is, it tends to take the form of Möller-Barlow's disease.

(*d*) *Any debilitating condition* is a potential starter of dental tartar. Prolonged inappetance causes a lack of the cleansing action of the tongue, lips and cheeks on the teeth and as such encourages the deposit on the teeth.

The above are pathological factors which encourage tartar formation. The diet, however, plays a very important rôle.

Harking back to the anatomy and physiology of canine teeth it is remembered that they are used for tearing, and reducing the food to proportions to permit deglutition, while the last teeth in each jaw are used for crushing bones and the like. Mastication is therefore reduced to a minimum in case soft foods are fed and with it, the rubbing and cleaning action of the tongue, lips and cheeks. Given a natural diet of fairly large pieces of meat, cleaning of the teeth automatically takes place.

The partial or almost complete cereal diet of many dogs tends to cause adherence of food particles to the teeth, particularly if the cereals are very refined. In the case of grossly pampered lap dogs the food position is beyond comment, and, usually, so are the teeth.

There is no tooth in the dog's mouth that is immune from tartar but certain teeth are more prone to the deposit than others.

First of all the teeth of a dolichocephalic breed are less readily affected than in the case of a mesaticephalic breed and the teeth in a brachycephalic head are most easily effected, i.e. the position is the reverse to that of dental caries.

As everybody knows the palate of the dog abruptly widens after P3. The upper buccal arcade swings outwards tending to leave a bit of a pouch, where the cheek is not in such very snug apposition. This is particularly noticeable in brachycephalic skulls. In the dog these skulls are also invariably prognathic with the lower incisors usually exposed over the lip.

This is why the cheek teeth up to P3 and the incisors more than the other teeth lack the cleaning massage of lips and cheeks, and tend to be most readily affected by tartar.

As has been shown, the dental calculus not only encroaches on the gingival margin but may, especially in the case of serumal calculus, be below it. The irritation it causes and the separation between gum and tooth invites invasion by bacteria and very soon the first step in paradontal disease is in evidence—variously called pericementitis, alveolar periostitis etc. During this process the alveolar wall and the bone further afield are affected to a greater or less extent—the severity being dependent on many factors, but, inter alia, the density of the bone and the virulence of the infection. The maxillary bones, being more cancellous, are generally more widely affected than the mandible. In the maxilla it is not uncommon for a discharging fistula to form over

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the antrum as a result of paradontal disease in the carnassial (P.4) tooth. This may be due to empyema of the antrum or, very often, to caries of the external plate of the antrum.

As has repeatedly been pointed out, besides the exciting causes, predisposing causes are to be considered. In this case again, genetic predisposition (anatomical or humoral) has to be considered. However, as the integrity of the dental apparatus is dependent on proper nutrition and good health particularly during the critical period between 8—24 weeks in pups, it can readily be appreciated that dietetic abuses would pave the way for subsequent diseases—poorly formed teeth, weak alveoli and thin gums. A very interesting observation can be mentioned here and that is that distemper if contracted by a pup during 2—3 months of age, almost invariably leads to hypoplasia (or aplasia) of the enamel, even where other pups that suffered from severe rickets at this age may show normal looking permanent teeth. It is suspected that the distemper virus has a specific action on the enamel organ.

It is generally known that bone, like all other tissues in the body can only be saved from atrophy if it is used. Disuse atrophy may show itself radiologically in bone as early as 14 days. When bone has to do work, it becomes denser with a better blood supply. The trabeculae are also directed towards the lines of greatest stress. The jaws should therefore, have work to do by being given suitable food to bite and tear i.e. food that is not finely cut up, as well as an occasional bone. This helps the perialveolar wall to be of maximum density—a barrier to the spread of paradontal disease. In addition the integrity of the pericementum is safeguarded—thus helping to prevent the changes resulting in paradontal pathology. The habit of feeding dogs bones excessively is unintelligent, as they do not require excess calcium-phosphorus, and they are exposed to excessive dental attrition and, often, chronic constipation.

Paradontal disease in the dog is potentially serious. The loss of teeth is the least important aspect. Much more important is it that this disease very often, through chronic toxaemia, plays a major rôle in the causation of nephrosis (Bright's disease), myocardial degeneration and possibly liver damage.

Before concluding, a word about dental conservation. Various methods akin to those employed in man have been described. Technically there is no reason why these methods should not be applied to animals, except that few cases would be seen in time to preserve the affected teeth, the matter of expense to the owner is an important factor while, last but not least, one is faced with an essentially antagonistic patient. Taking all this into consideration, the limited demand would not justify the outlay at this stage.

It is hoped that this paper has shown how very akin the problems are of those who have made the prevention and treatment of disease their calling, irrespective the species with which they are preoccupied. It is suggested that through mutual discussion and joint experimentation we may be enable to lighten each other's burdens.

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CLINICAL COMMUNICATION

Sedation of the Equine

C. J. Muller*

(*Received for publication 20th September* 1960)

The use of chloral hydrate, pethidine and chlorpromazine to sedate the equine is discussed.

Chloralhydrate.

Chloral hydrate was one of the first narcotic agents to be used in veterinary medicine and to this day it still proves to be one of the most effective drugs, provided of course it is administered intravenously. Perivenous deposition can largely be prevented, if the site of injection is desensitized with a local anaesthetic, previous to the introduction of a trocar and cannula (12 gauge and 3.5 inches long), which is preferred to a long needle that may pierce the vein if the animal flexes its neck while the injection is being given.

The dosage level is in the range of 1.5 grams of chloral hydrate per 50 kilos, suitably diluted, and given intravenously. Usually ½ ounce chloral hydrate is required for the average sized animal (400 kilos). Chloral hydrate is an ideal drug when the feet are examined because most horses sedated with this dosage of chloral can balance on three legs.

Chlorpromazine hydrochloride ("Largactil").

Given intravenously this phenothiazine derivative has produced very good results when used as a sedative to the bovine. When however, it is given intravenously to the equine, undesirable side-reactions occur in almost all cases. To avoid the undesirable reactions, pethidine has been used in conjunction with chlorpromazine, either mixed in the same syringe or the pethidine given prior to the administration of chlorpromazine. The dosage level is 1 mgm/Kilo of each, slowly administered intravenously. It must be given slowly, particularly when the two drugs are mixed, in order to prevent the possibility of drug shock. Symptoms of pulmonary distress, tachycardia and profuse sweating may occur if the administration is accelerated. Urticaria may occur, as pethidine is known to stimulate histamine production: it can be controlled by an anthistaminic.

* Faculty of Veterinary Science Onderstepoort.

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The separate administration of the two drugs was used successfully on 15 aged horses. In each instance the drugs were given as rapidly as possible at 3 minute intervals in order deliberately to produce undesirable reactions. None occurred. When the drugs were mixed in the same syringe, it became evident from the first case, an aged mule that died 5 minutes after the injection, that accelerated administration would not give good results. By slow intravenous administration however, it was used successfully on 14 horses and mules.

The physiological effect of this combination is noticed after 3 minutes, and reaches its maximum at approximately 8 minutes after administration; the animal being sedated for a period lasting from 3—6 hours. The patient is very unsteady on its legs, and will only in exceptional cases be able to control itself on three legs for purposes of shoeing or examination.

It was also evident in five of these cases that chloral hydrate can be used successfully for anaesthesia after the animals are sedated with pethidine and chlorpromazine. The chloral hydrate is given slowly until the desired effect is reached. Usually a $\frac{1}{4}$ ounce chloral is sufficient at this stage to make the animal lie down quietly and in this state of anaesthesia, it can be prepared for any operation.

ACKNOWLEDGEMENT

I wish to thank the Director of Veterinary Services for providing the facilities to test these drugs.

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CLINICAL COMMUNICATION

Outbreak of Blackquarter

Susanne Solomon*

(*Received for publication 21st November 1960*)

History

No animals in the herd had been vaccinated against Blackquarter for three years. The herd comprises 26 animals. In recent years there has been no outbreak of Blackquarter in this area, which lies within the White River district known as Kiepersol, and forms part of the Lowveld.

On May 21st, 1960 an 18 months crossbred Jersey heifer was brought in for post-mortem examination. She had been found prostrate and in great pain early in the morning, and had died on the same afternoon.

Post-mortem

After the first few incisions which exposed the inner thigh muscles, crepitous swellings were visible in one side, the muscles being spongy and dark red. As these lesions were considered sufficient for the diagnosis of Blackquarter no further examination of this carcase was made.

The owner later disclosed that four other bovine animals were showing swellings and one in particular seemed extremely sick.

Examination

The farm in question was visited in the early morning of the following day. All unaffected animals were inoculated against Blackquarter and each one was temperatured. As a precaution it was decided that each beast would receive a daily injection of Penicillin for one week, according to its estimated weight. The owner agreed to check the temperature of each animal daily.

I. Heifer, 19 months old.

Symptoms

Recumbent and appeared in great pain. Temperature 97.2°F The dewlap was swollen and tense, and felt spongy to the touch.

* Veterinary Practitioner, White River, Eastern Transvaal.

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Diagnosis: Blackquarter.

Treatment: 3 million units of Penicillin were injected i/m daily for one week.

Course: On the same evening the heifer was taking a little food and water. The crepitous dewlap swelling improved gradually over period of two months. At the end of this period the heifer appeared normal.

II. Cow. 2 years 9 months old.

Symptoms

Complete drop in milk yield. Extensive swelling of the upper portion of the right hind quarter. Stiff and painful gait. Temperature 103.6. Great loss of condition and appetite.

Diagnosis: Blackquarter

Treatment: 3 million units of Penicillin i/m daily for one week.

Course: 3 days later appetite returned and temperature was normal. Within 6 weeks the swelling disappeared and the cow returned to normal.

III. Cow. 6 years 4 months.

Symptoms

Temperature 99.4°F. Drop in milk yield, loss of appetite, elongated crepitous swelling along lumbar region.

Diagnosis: Blackquarter

Treatment: 3 million units Penicillin injected i/m for seven days.

Course: There was great improvement within six days. Milk returned on the seventh day. Swelling persisted for two months.

IV. Calf (heifer) 6 months.

Symptoms

Temperature 102°F. Loss of appetite, extensive crepitous swelling of lower jaw extending towards dewlap, and a similar swelling on the left hind quarter,

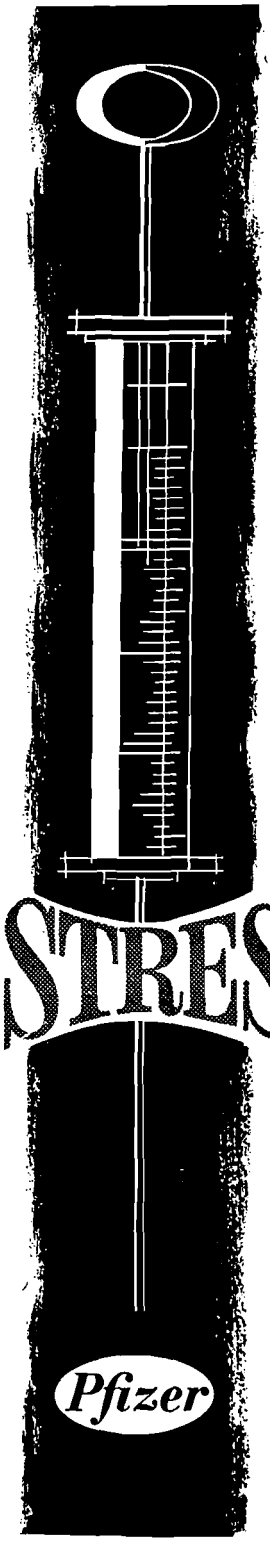
Diagnosis: Blackquarter

Treatment: 1 million 500,000 units of Penicillin injected i/m daily for one week.

Course: The swelling subsided within four days and had completely disappeared at the end of one month.

No further cases of Blackquarter have occurred on this farm in the four months which have since elapsed.

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REPORT ON THE VIth INTERNATIONAL BLOOD GROUP CONGRESS IN MÜNCHEN

21—23 August 1959.

D. R. Osterhoff.*

The study of blood groups of our domesticated animals has made much progress during the last 30 years and in the past 10 years the results of this research on practical breeding has become more significant.

The VI. International Blood Group Congress held in München, Germany on August 21, 22 and 23, 1959 demonstrated not only the practical applications of this new field of research but indicated also new ways for a better understanding of other problems of heredity.

Blood group experts from the following European nations took part in the proceedings: Belgium, Czechoslovakia, Denmark, Germany, Finland, Sweden, Switzerland and Yugoslavia. The South African blood group laboratory also sent in a progress report and took part in the International standardisation and repeatability tests.

The first paper was presented by Dr. J. Rendel of Sweden on *A study on relationships between blood groups and production characters in cattle.* Approximately 1,100 animals were tested with the blood typing reagents detecting blood groups in 10 different blood group systems. Records on butterfat yield and fat percentage of the first lactation as well as data on heart girth measurements were collected from each cow. Relationships between blood groups and these production characters as a result of pleiotropy, linkage or heterotic effects of the blood genes were investigated. To avoid an effect of selection all cows which were blood tested more than 18 months after their first calving were discarded. All statistical tests were carried out within groups of paternal half sibs.

The results indicated a definite relationship between the B-system of blood groups and fat percentage of the milk. Thus cows carrying the allele $B^{BO_1Y_2D'}$ produced milk with approximately 0.16 units higher fat percentage than those lacking this allele. Animals heterozygous at the B-locus had a significantly higher fat percentage than those homozygous. The J and M genes within the J and M systems respectively seemed to have an increasing effect on the fat percentage, while the L gene had a strongly depressive effect in this character. It could not be established if this relationship was due to pleiotropy, linkage or heterosis or any combination of these factors. There was no consistent influence of any of the blood group genes on the total butterfat yield or the heart girth measurements.

* Blood Group Laboratory, Division of Animal Husbandry and Dairying, c/o Division of Veterinary Services, Ondersteporrt.

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Similar studies were performed in Denmark and a paper presented by Dr. A. Neimann-Sörensen included: *Blood- and Serum-group investigations on cattle, pigs and dogs in Denmark.* In a material of 185 bull progeny groups from the Danish Progeny Testing stations including about 3,000 heifers, the possible association between blood types and production characters has been studied. Preliminary analyses of the results seem to show that certain B- alleles have a statistically significant influence on fat percentage and milk yield. It could again be demonstrated that the $^{BO_1Y_2D'}$-group had a highly statistically positive effect on fat percentage, other B-groups however had a depressive effect on both fat percentage and milk yield. It could be established that the share of blood group genes in the total genetic variance amounts to 7 per cent for fat percentage and 5 per cent for milk production.

By starch gel electrophoresis 6 different beta-globulin types could be distinguished in cattle. The original work was done by Smithies who could reveal that the serum types were controlled by three allelic genes. Studies in Denmark have demonstrated the value of application of serum types in parentage control in cattle. Other investigations have been published which point at a certain relationship between the serum types and embryonic death. When both the dam and the embryo possess the E-gene, it seems that the possibilities of fetal survival are diminished. Furthermore, other investigations have indicated an association between the serum type of an animal and its milk production capacity.

For blood grouping of pigs 16 specific test sera have been produced. The use of these test sera in blood typing of 516 pigs from the Danish Progeny Testing Stations and their respective parents has shown each of the 16 factors to be inherited as dominant characters, and controlled by genes on 9 apparently independent loci. The significance of this work in relation to practical pig breeding, is discussed.

Investigations on the blood groups of the dog have revealed five blood group factors, G, H, I, J and K. The J antigen is serologically related to the bovine J factor. On the red cells, it occurs in three subgroups, J_1, J_2 and J_3 and it is supposed that these three factors are analogous to the factors A—A', previously described by Young et al. By further immunizations 4 new types of antibodies could be produced but the investigations are not finalized.

The third paper presented at the congress was given by Dr. A. Tolle, Germany on *"Basic theories and experimental results on relations between blood group factors and first lactation performances."*

Tolle discussed the problem of possible ways of relations between blood group factors and physiological or morphological characters and he assumed the following ways:

1. Genes determining the formation of blood group factors also affect the formation of other characteristics.
2. Genes determining the formation of blood groups are linked to those genes which influence the formation of characteristics for performance and body conformation.

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The main point for any search for relations between the blood group factors and performance characteristics is the selection of a reliable material. Tolle suggested therefore the study of bull families i.e. the comparison of daughters of the same bull and furthermore the performance results of only the first 200 days of the first lactation. In the material studied—1005 cows—the difficulties of a selection, mainly the exclusion of the strong influence of environmental factors, was explained.

The results can be summarised as follows: It could be shown that the blood factor M may exert a negative effect on length of lactation. Furthermore it could be demonstrated that blood factor M and U_2 had a depressive effect on milk production, and factor Y_2 an increasing effect on milk production. In one herd with largely constant environmental conditions these factors (Y_2, M and U_2) showed a relation to protein and S.N.F. yield, and were correlated to milk yield and fat content.

These and other results show that further investigations on the influence of different factors and on the influence of homo- and heterozygosis concerning all blood group systems are advisable, to throw more light on the question of relations between blood groups and production characters in cattle.

Dr. D. G. Gilmour, England, gave a very comprehensive survey of *Serological and genetic investigation of blood cell antigens of chickens.* Four highly inbred lines of White Leghorn chickens were studied, each originating from entirely distinct foundation stocks, and maintained at Reaseheath, England by annual brother-sister matings. Agglutinating iso-immune sera were used to identify the antigenic factors. Genetic evidence from inbred and backcross matings showed that the genes determining these antigenic factors occurred at five independent loci, A, B, C, L and N. Tests with American reagents showed that three of the loci corresponded with the A, B and C loci previously reported by Briles and his co-workers, while the other two (L, N) were new. A further antigenic factor which had been identified but the mode of inheritance of which had not been clearly worked out was shown by such tests to be determined by an allele at the E-locus of Briles. The close linkage of A and E was confirmed.

A detailed study of the B-locus showed the presence of nine multiple alleles each determining a separate antigen, distinguished by different cross reactions, absorption and immunisation characteristics, among the four lines studied.

Attempts were made to identify further antigen loci which may have been fixed in homozygous condition in the lines, and which would not have been identified by within-line immunisations, by making immunisations between both backcross progeny and individuals of different lines, but without success. Most of the sera so produced identified B-locus antigens.

The continuing segregation of several loci in each of the four lines at very high levels of inbreeding is powerful indirect evidence that heterozygous individuals at these loci were at a selective advantage under the conditions at Reaseheath. The basis of selection was family

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size, the largest two or three groups of full sibs in each being chosen each year, to provide the breeding stock for the following season. It was shown that the largest families were always those on which the maximum number of antigen loci were segregating. This relationship followed from a general superiority of the heterozygous individuals in reproductive performance and livability. Comparisons between heterozygous classes of individuals at each of the A, B, C and L loci showed that heterozygous sires were strongly superior in fertility, heterozygous hens generally superior in egg production, both annually and during the hatching season, and heterozygous progeny superior in survival from fertilisation until a few months of age. It was not possible for various reasons to make such comparisons for the other loci.

The existence of a connection between antigen heterozygosity and superior performance in inbred lines suggest the possibility that the various heterozygous classes occurring in commercial stock, may also differ significantly in performance. Evidence that this is so has been obtained by at least one large American company specialising in the sale of "hybrid" chickens.

The different activity reports of the single blood group laboratories included reports from the following laboratories:

Gent, Belgium
Libechov, Czechoslovakia
Copenhagen, Denmark
Helsinki, Finland
Göttingen, Germany
München, Germany
Wageningen, Netherlands
Oslo, Norway
Warsaw, Poland
Onderstepoort, South Africa
Uppsala, Sweden
Ljubljana, Yugoslavia

Because many of the laboratories mentioned have only recently started their blood group work, the main points discussed in the respective reports were the production of bovine anti-sera and blood group reagents. Only the South African laboratory gave some more results of studies on other subjects as well as the seasonal variation of the naturally occurring iso-antibodies for the J-substance and the first results of a blood group classification of East and South African cattle breeds.

For the first time an international standardization of animal blood group reagents was initiated at the congress. For this purpose 89 different anti-sera from 10 countries and 268 blood samples of different breeds from 13 countries were tested at the München blood group

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laboratory. A committee for standardization selected the anti-sera and the participating delegates from the different laboratories recognised 15 anti-sera as standard blood typing reagents. One of these recognized standard reagents had been produced at the Onderstepoort laboratory. For the selection, storage and use of standard blood typing reagents in cattle, specified rules were worked out and accepted by the meeting.

SUMMARY

The congress has shown that much progress has been made in this new field of research, both in the sphere of blood group serology, and the further investigation of the genetical structure of animal blood groups. The bearing of the results of this research on practical applications such as proof of identity, determination of parentage, determination of monozygous twins, and diagnosis of sterile heifers in mixed twins, has been widely accepted by all cattle breeders and breeders' associations, as shown in the reports of the participating delegates at the meeting. Besides the known facts in connection with the advantage of heterozygosity in different blood group systems in chickens with regard to livability and several other production traits, which had been proved by Gilmour as shown above, three independent studies could be presented at the congress on similar relationships between blood groups of cattle and production characteristics. These investigations are very important and the bearing of the results on relations between the hereditary character of the blood groups and signs for productivity could lead to broad practical applications in the selection of breeding animals.

New studies of blood groups in pigs and dogs could be reported. In pigs the Danish laboratory succeeded in designating 16 blood group factors belonging to 9 genetical systems.

Through international cooperation, the first standardization of blood typing reagents for cattle blood groups could be achieved and rules for the selection, storage and use of standard blood typing reagents in cattle be worked out.

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JOHAN GODFRIED BEKKER. †

Met die heengaan op 12 Oktober 1960 van dr. Bekker het die veeartsenykundige professie 'n baanbreker verloor.

Boet, soos hy algemeen bekend was, het met 'n B.Sc. Agric. graad (Stellenbosch), hom in 1923 laat inskryf as student op Onderstepoort. Hy was 'n briljante student en het sy graad B.V.Sc. in 1925 behaal. Hy was van 'n opgeruimde geaardheid en bemin waar hy hom begeef en veral by sy kliënte toe hy in die privaat praktyk gestaan het.

Dr. Bekker het 'n aanstelling op Onderstepoort aanvaar en is verplaas na Armoedsvlakte waar hy navorsing verrig het in lamsiekte. Hy het gou op die idee geraak om fosfate deur middel van drinkwater aan beeste toe te dien, en het in 1933 sy D.V.Sc. behaal met 'n verhandeling oor die aangeleentheid. Die wêreldberoemde biochemikus dr. H. H. Green het in 'n resensie die stuk navorsing baie aangeprys en dit as baanbrekerswerk beskou in die probleem van fosfaatvoeding.

In 1930 is dr. Bekker na Bradford Engeland gestuur om woltegnologie te studeer, met die oog om die pas-gestigte Afdeling in dié vak waar te neem.

Nie lank na sy terugkeer het hy egter bedank en in die private praktyk gegaan saam met dr. Kind. Hier ook het hy weer baanbrekerswerk verrig, met sy deeglikheid en kennis het die publiek algar die waarde van die private praktisyn besef, en so het hy die weg gebaan vir baie wat hom sou volg.

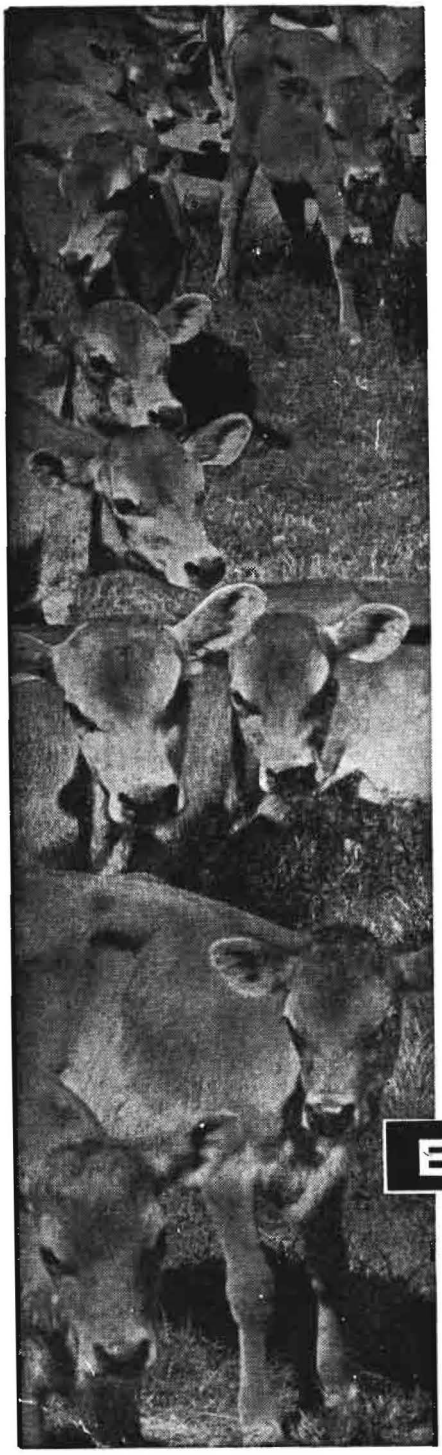
In die laaste paar jaar het sy gesondheid afgeneem en toe hy sy vrou, Cea, die meeste nodig had, het sy hom ontval.

Ons wil ons innige meegevoel uitspreek met sy seun Johan en dogter Annita met hulle droewige verlies.

Hy was in sy sestigste jaar.

P.S.S.

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Unieke nuwe Pfizer-inspuiting bestry onvrugbaarheid by beeste ... verhoog mate van bevrugting

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Met Embryostat word teelkoste derhalwe verminder en dit het gesonder troppe tot gevolg.

*Wat die wetenskaplikes sê oor EMBRYOSTAT **

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S.A.V.M.A. INFORMATION SERVICE.

Events.

Conferences.

A conference on the publication of the results of research sponsored jointly by the C.S.I.R. and the Department of Education, Arts and Science, is to be held in the University of South Africa buildings Pretoria, on February 2 and 3, 1961.

Enquiry Committees (Commissions).

1. Komitee van Ondersoek in verband met Skemas om Kunsmatige Inseminasie uit te bou ter bevordering van die Veeteeltbedryf. (v.d. Wath-Komitee).

   This committee has now submitted its report to the Minister of Agricultural Technical Services.

2. Committee of Enquiry into the organization and functioning of the Technical Services of the Department of Agricultural Technical Technical Services. (Rautenbach Committee).

   The Association is busy preparing the evidence which it will lay before this committee. It is likely that the evidence will be given during March or April 1961.

3. Commission of Enquiry into the Uses of Ionised Radiation.

   The Association expects to give evidence before this Commission as there is a possibility that it might recommend training and registration of the persons using this type of apparatus.

Association News.

1. *Animal Reproduction Group.*

   The newly formed Animal Reproduction Group has now formulated its objects. Dr. I. S. McFarlane, Onderstepoort, is the Hon. Secretary.

   The Association now has five Branches and two Groups.

2. *The constition of the Association* is being prepared for printing and should be available for issue to members by April 1961.

3. *The Next Annual Congress* and *Fifty-Sixth* Annual General Meeting is to be held at Durban on September 25-28, 1961. The meeting will be held at the Killarney Hotel where accommodation at reduced rates will be available provided the management receives

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the necessary patronage from members. Further particulars regarding the congress can be obtained from the Hon. Secretary of the Natal Branch (Dr. W. C. Viljoen, P.O. Box 397, Pietermaritzburg) or from the Hon. Sec. S.A.V.M.A. Onderstepoort.

4. A *New International Veterinary Journal* is to be published in England, viz. The Journal of Small Animal Practice. It is sponsored by the British Small Animal Veterinary Association. The subscription is £4 per annum (Price in Britain). Enquiries should be directed to—

Messrs. Pergamon Preis Ltd.,

(Journals Department)

Headington Hill Hall,

Oxford,

England.

Professor C. F. B. Hofmeyr, Department of Surgery, Onderstepoort, is the South African Consultant Editor.

Personalia.

Dr. P. J. J. ("Flippie") Fourie retires at the end of December 1961 from the post of liaison officer which he has held since his retirement from the permanent service of the Veterinary Division, six years ago.

He will be sorely missed and the wishes of all members go out to him in his role of public relations officer to a private enterprise.

Dr. R. J. Frean has taken Dr. Fourie's place at Onderstepoort.

Overseas Visits by Members.

We believe that Drs. Schalk van Rensburg, Steve van Heerden, Hofmeyr, and Ted Edwards will be going overseas next year and we wish them the very best of luck and happy landings.

"Schalk" is proceeding to Europe and Britain in his private capacity. He will attend the Fourth International Congress on Animal Reproduction at The Hague on June 5–9, 1961. He will represent the Association at a meeting of the Permanent Committee of the World Veterinary Association which is to be held in Paris on the 18th May, 1961.

He will also attend the meeting of the Society for Animal Breeding in London on May 30th, 1961.

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"Steve" will attend the Congress on Animal Reproduction at The Hague and will also attend a meeting of the F.A.O. Expert Panel on Livestock Infertility in Rome from May 29th to June 3rd, 1961. In addition he will attend the Eighth International Congress on Animal Production which is to be held in Hamburg on June 12th-16th 1961. He will also visit various institutions and universities in Europe and Britain where the subject of reproduction in animals is studied.

"Ted" will attend the congresses at The Hague and Hamburg, and in addition will visit the Animal Quarantine Stations at The Hook of Holland, Hamburg and those in Switzerland and Scandinavia. He will make a study of the methods adopted in Europe and Britain for the prevention of spread of notifiable diseases.

"Hoffie" will attend the Congress of European Professors of Veterinary Surgery which is to be held at Cambridge on 5-8 April, 1961. He will also attend the Congress of the World Small Animal Veterinary Association and British Small Animal Veterinary Association to be held in London during April, 1961. On invitation as guest professor for four weeks he will lecture at the Department of Surgery of the Old University of Berlin and he will visit a number of Faculties in Europe and Britain concerned with Veterinary Education.

We wish all these colleagues the best of luck and a safe return. We look forward to the talks and contributions to the Journal, which we feel sure they will give us on their return.

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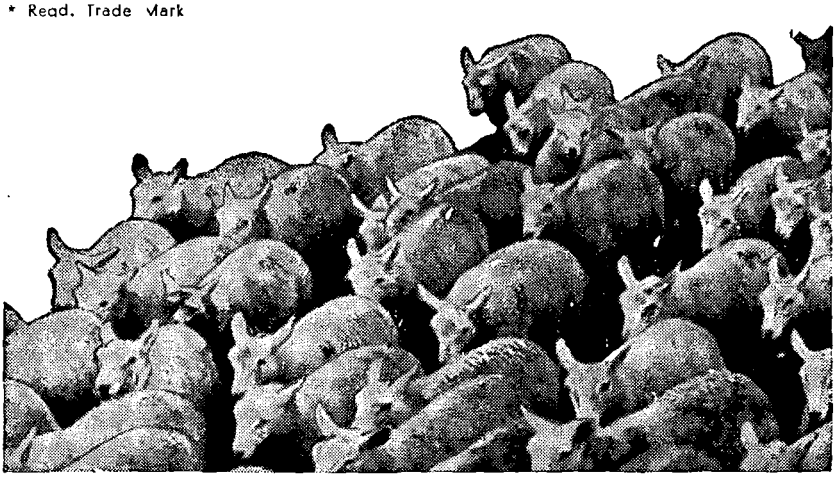
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TREATMENT — Diseased tissue should be carefully pared away and Chloromycetin Tincture 10% applied with a stiff brush. Supplied in bottles of 2 and 16 fluid oz. with brush.

1. Aust. Vet. Jl. 1954, 30:209.
2. Vet. Rec. 1955, 67:74.

* Regd. Trade Mark



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ONDERSTEPOORT STUDENTS' HOSTEL.

CHAIRMAN'S REPORT.

Once again the jacarandas are beginning to blossom, and examinations are all too close at hand, both indicating the formidable approach of the end of yet another year. No more suitable time to look back, and ponder over what has been done, and what should have been done over the past year.

It is often difficult in a chairman's report not to single out the good from the bad and make the past appear rose-coloured. I will try not to concede to this fault. By and large, however, the past year has been one well within keeping the festival spirit of the 50th year of Union, and the anniversary of our University.

From the start, a high pitch was set, which we tried to maintain throughout the year. In the Interhouse swimming Gala, much to our own surprise, we managed to take second place to "Dagstudente": a feat which I think can be largely attributed to the legendary O.P. spirit.

This spirit, too often misunderstood and condemned, is a spirit created amongst both Afrikaans and English speaking sections of our people, forming a situation which is, perhaps, unique among faculties of our South African Universities. As I have mentioned, it is something which is not fully understood; which appears foreign and alien to Tukkies, and yet which forms such an intimate part of Tukkies, a University to which we are proud to belong. It is a spirit, created among men of many creeds, a spirit of co-operation, and above all understanding. For every problem, for every point of view, for every action, there are, and must always be, two sides; it is in creating an atmosphere for a better and deeper understanding of these sides, that we will be able to progress.

By this means I am sure we will be able to alleviate the antagonistic feelings which appear to exist sometimes at galas and sing-songs. Believe me, I am in no way trying to excuse us; we are as much to blame as anyone else. I only wish to point out, that too often a place and an institution are judged by the conduct of only a very small percentage of its members.

But on the other hand let us be constructive in our criticism, and let us not be buried by our so often single minded thoughts. Above all, let us not envy, or deny, the accomplishments of others. In an unselfish spirit, competition has never harmed anyone. In fact, it has made good many an error in the co-existence of a community.

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Rag this year also brought us success, with a prize for the most humorous float. For this effort I must thank our third year class for their work and determination.

At the beginning of the year the Onderstepoort students unofficially affiliated with the Occupational Therapy Faculty, a move welcomed by many, which did not perhaps succeed as had been hoped. We look forward to a continued affiliation next year.

In the field of sport our success was varied. We had one of the best rugby teams we have had in years, and seemed to be set to win our league division. But through injury, and perhaps lack of interest in the latter part of the season, we threw our chances away. Our trip to Swaziland was, however, both a social and rugby success! Thanks must go to our Captain, Marius van Tonder, and his committee for the season.

Golf is a sport which has come to the fore this year and with the restoration of our course, many pleasant hours have been spent chasing after the little white ball. We hope next year to be able to hold interhouse competitions.

Again the Intermedics sports meeting proved a great success, and proved invaluable in maintaining a fellowship between the three medical faculties. O.P. just managed to hold off a determined bid for victory by Dentistry.

Once again Sonop showed us a thing or two about Cross-country and we slipped back to fourth position. But from defeat comes the inspiration for the future.

This year, for the first time, O.P. took part in a "Studentevaria" programme of its own. Our thanks for this privilege must go to Rina Thom and Josua du Toit for making this possible, and also to R. de la Rey, E. Genis and Mrs. Lombard for their work in the preparation of the programme.

Our thoughts and deepest sympathies in their hour of need were with the families of Linda Wissekerke; Jean Malherbe; Mrs. Bisschop; and Prof. Graf.

In memoriam to our late Dean, Prof. H. Graf, I wish to say a few words. At all times his interest in the student and student community was foremost in his thoughts. The work and energy he devoted to the establishment of our student library will not go unforgotten. His supreme diplomacy, and willingness to listen to, and help students in their troubles, made him a man respected by all. His example throughout his long career at Onderstepoort both as research worker and lecturer will not fade fast. He was indeed a "white man", whom I am proud to have had the privilege of knowing.

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To his successor, Prof. R. du Toit I extend, on behalf of the students, every wish for success with his appointment. I am sure it is no easy task to take over from a man like Prof. Graf.

Finally it remains for me to thank my House Committee for the past year for their co-operation and support at all times, which certainly went to making my task so much easier. Special thanks to Dr. Adelaar, our Warden, for his guidance and help in all matters during the past year, as well as to Prof. de Boom.

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Just Published

DISEASES OF THE PIG

by **D. J. Anthony, M.R.C.V.S., M.V.S.M., F.R.S.H.**

Chief Veterinary Surgeon, Brierley Hall, Staffs; Examiner in Meat and other foods to the Royal Society for the Promotion of Health; Formerly Examiner in Anatomy to the Royal College of Veterinary Surgeons.

and **E. F. Lewis, M.R.C.V.S., F.R.S.H.**

Department of Medicine, Royal Veterinary College, London; Examiner in Veterinary Medicine to University of London; Examiner in Meat and other foods to the Royal Society for the promotion of Health.

A general textbook on pig diseases and their treatment with a short introduction on breeds, housing, feeding and management. Intended for Veterinary Practitioners and Students, Public Health Officials and all others who require information on a wide range of topics associated with pig-rearing.

Pig farming plays an important part in the economy of many countries, and the maintenance of healthy stock is essential to good husbandry. The guidance given in this book will not only aid in the treatment of diseases, but also in its prevention, and thus ensure the economical production of sound animals.

In this new edition an extensive revision has been made and a considerable amount of new material is included. The description of methods of therapy have been brought up-to-date, and sections have been pruned, rearranged or re-written for greater convenience of reference. A new chapter on diseases of newly born and young pigs marks the economic importance of this aspect of the subject. New photographs replace some of the less informative illustrations, and the list of references have been greatly amplified.

5th Edition 1961

416 pages, 97 illustrations. Price 35s. postage 2s. 3d.

Price quoted is for United Kingdom

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OUTBREAKS OF NOTIFIABLE DISEASES—QUARTER ENDING 31ST DECEMBER 1960.

ANTHRAX.

Transvaal:

Nelspruit, Carolina, Barberton, Sibasa, Middelburg, Potchefstroom, Wolmaransstad.

O.F.S.

Frankfort, Brandfort, Bethlehem, Harrismith.

Natal:

Mahlabatini

Cape.

Vryburg, Stutterheim, Alexandria.

BENIGN BOVINE GONDERIOSIS (TZANEEN DISEASE).

Natal:

Umzinto, Harding, Kranskop, Port Shepstone, New Hanover, Dundee.

Transvaal:

Nelspruit, Sibasa, Piet Retief.

BRUCELLOSIS.

CATTLE	*Natal*	Camperdown
GOATS	—	
SHEEP	—	

B.W.D.

O.F.S.

Frankfort, Ficksburg.

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Transvaal:

Ermelo, Kempton Park, Standerton, Vanderbijlpark.

Natal:

Pinetown, Camperdown.

Fowl Typhoid.

Natal:

Dundee, Newcastle.

Transvaal:

Heidelberg, Springs.

O.F.S.

Harrismith

Cape:

Malmesbury, Somerset West.

Foot and Mouth Disease.

Transvaal:

Barberton.

Lumpy skin disease.

Transvaal:

Potchefstroom, Germiston.

Natal:

Dundee, Estcourt.

O.F.S.

Rouxville.

Cape.

Worcester, Somerset West, Swellendam, Robertson, King William's Town, Aliwal North, Barclay West, Heidelberg.

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Mange: Equine

Cape: Namakwe.

Mange: Goat

Cape: Umtata, Middeldrift, St. Marks, Indwe, Glen Grey, East London Mquanduli.

Natal: Ngotshe, Hlabisa, Mgwavuma.

Nagana.

Natal.

Hlabisa

Rabies.

Cape.

Kuruman, Vryburg, Postmasburg, Albert, Kimberley.

Transvaal.

Letaba, Soutpansberg, Potgietersrus, Wolmaransstad.

O.F.S.

Bloemfontein, Boshof, Zastron, Hoopstad, Bothaville.

Swine Erysipelas.

Cape.

Steynsburg.

Tuberculosis: Bovine.

Transvaal.

Potchefstroom, Pretoria, Klerksdorp, Ventersdorp, Piet Retief, Bethal, Vereeniging, Ermelo, Volksrust.

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Cape

Beaufort West, Caledon, Swellendam, George, Delareyville, Worcester, Mossel Bay, Bellville, Riversdale, Mt. Currie.

Natal.

Ixopo, Richmond.

O.F.S.

Hoopstad.

AVIAN.

Cape.

Caledon.

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JOURNAL OF THE SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

VOLUME XXXI

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