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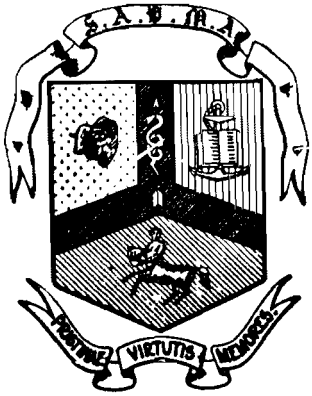

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VAN

DIE SUID-AFRIKAANSE VETERINÊR-MEDIESE VERENIGING

METHYL-THIO-URACIL IN FATTENING RATIONS OF YOUNG AND MATURE STEERS.*

H. J. J. TERBLANCHE**

SUMMARY

A series of trials using methyl-thio-uracil as an additive to fattening rations and involving 328 steers is reported. In general the results with two-year old steers were not promising, there being a negligible increase in dressed carcass weight with however a fair increase in feed conversion efficiency. Most of the apparent advantage in liveweight gain was not reflected in dressed carcass weight.

In mature steers the results were more rewarding there being a moderate increase in dressed carcass weight, fair feed conversion and an improvement in grading. The response, however, largely depends on the initial condition of the animals.

Residual M.T.U. in the tissues of treated animals was found to be negligible.

The possibilities of M.T.U. feeding under specific conditions are discussed. In general the results were not impressive.

INTRODUCTION

Goitrogens are substances which interfere with the synthesis of thyroid hormones. The resulting hypothyroidism tends to depress the metabolic rate and hence increases fat deposition. In response to the low level of thyroid hormone in the blood, the anterior pituitary produces large amounts of thyrotrophic hormone with resultant goitre formation.

The effects of goitrogens on stock have been widely studied. Those that have received particular attention include thio-urea, thio-uracil, methimazole (Tapazole) and, more recently, methyl-thio-uracil. These have been studied on goats, sheep, pigs, poultry and cattle. Observations have been made on growth and metabolic rate, thyroxin secretion, absorption and elimination, toxicity, reproduction and calcium metabolism. Those interested are referred to references 1 to 13.

The results with cattle in particular can be summarised as follows. Thyroidectomised steers gained weight at an increased rate during the first six to eight post-operative weeks after which the rate dropped, reaching pre-operative levels at about 20 weeks[1 2]. Dosages of 2.0 to 6.0 g thio-uracil per steer per day did not depress growth rate while dosages of 2.0 to 4.0 g per day increased weight gains slightly while tending to improve dressing percentage, degree of finish and feed conversion[6]. Essentially uniform levels of thio-uracil were maintained by oral and subcutaneous administration at 12 hourly intervals and the drug was eliminated from the blood stream within 24 hours of cessation of administration[7]. Feeding thio-uracil to yearling heifers at the rate of 4.0 g per day for 140 days did not noticeably affect growth rate, feed consumption or efficiency of feed conversion[9]. There was a poor response to the addition of methimazole to a fattening ration[11]. Methimazole had no influence on weight gains, dressing percentage or depth of loin fat coverage and little or none on feed consumption. Feed conversion was improved slightly, as was grading.

Results with methyl-thio-uracil (M.T.U.) can be summarised as follows: In a trial on eight cows it was concluded that its usefulness in fattening is negligible[14]. It made no significant difference to the dry matter or fat content of the flesh or livers when fed to old cows[15]. Better weight gains of up to 35% shown by treated cows was due to an increase in the weight of the stomach contents[16]. A significant increase in liveweight and liver weight was obtained in cows. Zinc, manganese and molybdenum were lowered significantly but copper and cobalt were unaffected[17 18]. Steers fed 0.5 g M.T.U. daily per 100 lb. body weight showed negligible amounts of residual M.T.U. in ribeye muscle, liver, kidney, digestive tract and depot fat[19]. Treatment increased weight by about 26 lb. and liver weight by about 5 lb. and spectacular gains in liveweight of as much as 11 lb. daily over the last week were recorded[20]. In a comparative trial with stilboestrol on cows, liveweight gains were better in the M.T.U. group which also showed no significant effect on ovarian function. In a trial in Italy on Friuli and Red Pied Bulls, the Friuli bulls showed signs of myxoedema after

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20 days. Liveweight gains were improved in the Friuli bulls by 10% and in the pied bulls by 65%. Feed conversion was better in the treated bulls. The author emphasises that breed differences should be considered when using M.T.U.[22]. In young bulls, daily weight gains of 5.61 lb. and 3.04 lb. were shown by treated and control animals respectively[23]. The authors also report the following details:

	Controls	M.T.U. Group
Pounds of feed/lb. weight gain	3.31	1.73
Slaughter percentage	57.27	56.64
Meat Analysis: Water content	75.6	76.6
Crude Protein	21.95	20.96
Crude Fat	0.8	0.7

In general these results indicate that M.T.U. feeding may cause better liveweight gains and feed conversion efficiency. Unfortunately no trials on mature steers have been reported.

In South Africa the slaughtering of younger animals is the exception rather than the rule. There, is, however, an increase in the fattening of adult cattle in feed lots for a few months prior to marketing. In such adult animals bone and muscle growth has ceased and rapid, economical fattening is the aim. Under these circumstances the use of M.T.U. might well be advantageous.

A series of trials was therefore conducted in order to ascertain to what extent M.T.U. feeding in both young and mature cattle would have a beneficial effect on weight gain, feed consumption and meat quality. Tissue residues of M.T.U. were also investigated.

TRIALS I AND II

YOUNG AND MATURE FEEDER STEERS

Materials and Method:

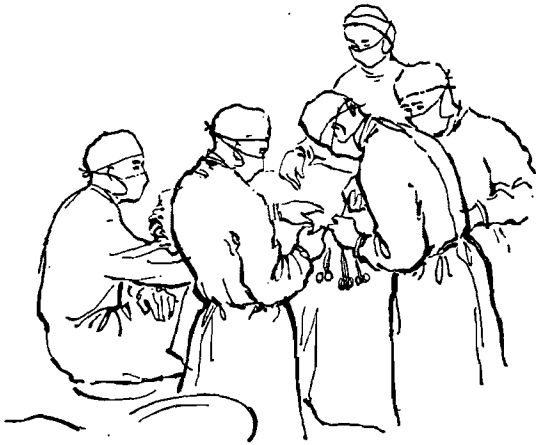
Fifty young Afrikaner steers, aged approximately two years and in fairly good condition were used in Trial I. In Trial 2, fifty well-grown, large framed Afrikaner oxen aged approximately six years were used. Half the animals in each trial received approximately 3 g of M.T.U. daily in the feed, whilst the other half served as controls. The mature group were wild veld types in low condition, and had to be introduced to maizemeal and lucerne hay feeding. The young group was in fair condition, and had been introduced to manger feeding prior to their arrival on the trial premises. Milled good quality maize and lucerne hay were used. The animals were each held in small pens measuring about ten yards square with individual mangers but with common water troughs between adjoining pens. Fresh water was available *ad lib.*

The introductory feeding period lasted six days. During this period the animals received ½ lb maize meal per 100 lb. body weight and an *ad lib* supply of hammer milled lucerne hay. They were ear-tagged and dewormed. They were weighed twice at the end of the introductory feeding period (initial liveweight); once 20 days later; once a further 15 days later and twice at the end of the trial, i.e. after 40 days of trial feeding. During the trial feeding period each animal was offered 1 lb. maize meal per 100 lb. body weight and lucerne hay in excess of the daily requirement. Both meal and hay intake were accurately weighed and recorded. Methyl-thio-uracil powder was premixed

Results and Discussion of Trials I and II.

TABLE 1.

	TRIAL I *Young animals*		*TRIAL II* *Mature Animals*	
	Control	*Treated*	*Control*	*Treated*
Initial condition of animals	Fair	Fair	Med.	Med.
Total Feeding period (days)	46	46	46	46
Preliminary Feeding period (days)	6	6	6	6
M.T.U. Feeding period	—	40	—	40
No. Steers per group	25	25	25	25
Average initial liveweight (lb.)	533	525	831	832
Average final liveweight (lb.)	615	630	953	993
Average gain (lb.)	82	106	121	161
Average daily gain (lb.)	2.4	3.0	3.5	4.6
Average dressed weight (lb.)	299	299	491	508
Dressed weight as % initial liveweight	56.1	56.9	59	61
% Carcasses grading prime or 1st grade	28	52	40	40
Average daily feed consumption (lb.)	14.3	10.4	22	18
Overall fat	2.96	2.80	3.7	2.9
Plinimeter	42.7	46.2	57.4	62.8



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with maize meal, di-calcium phosphate and molasses in such a concentration that two ounces of this mixture contained 3 g of M.T.U. Control animals each received two ounces daily of the identical premix without M.T.U. Mangers were cleaned every morning and the residual feed weighed. Measurements of the ribeye muscle (longissimus dorsi) were taken on the chilled carcasses and the marbling, overall fat and grading recorded.

In both trials the treated group took less food and gained more liveweight than did the controls. In Trial I, however, there was no difference between the dressed weights of treated and control animals, indicating that the liveweight advantage of the treated group was due to a greater weight of the digestive tract contents. In Trial II (mature animals) the treated group showed an average advantage of 17 lb. per dressed carcass and a saving of 4 lb. of food per head per day as compared to the controls.

When dressed weight is expressed as a percentage of the initial liveweight, the mature animals also showed a better response to M.T.U. feeding than did the younger group. On the other hand the younger group appeared to show a better response with regard to carcass grading. The low grading of the mature group can probably be ascribed to their poor initial condition.

Residual M.T.U. in muscle and liver was determined. An average blank value was obtained from tissues from the controls and this was used in reading the results from the treated animals. Grote's reagent was used and optical density measured at 680 mμ. All specimens tested contained less than 1 p.p.m. M.T.U. which is the limit of accuracy for the method. It would therefore appear that residual M.T.U. in the meat does not constitute a hazard.

The thyroids of the treated animals were distinctly larger and heavier than those of the controls.

TRIAL III

As M.T.U. is extremely bitter it was decided to test its effects on the palatibility of rations to which it was added with and without materials to disguise the taste.

Eight steers were grouped into four pairs and M.T.U. fed as follows:

PAIR A. Pure M.T.U. powder mixed with the maize meal ration.

PAIR B. M.T.U. formulated with black sugar, salt, di-calcium phosphate and aniseed oil mixed in the maize meal.

PAIR C. M.T.U. in a fluid formulation of molasses, aniseed oil and an emulsifier poured over the maize meal.

PAIR D. M.T.U. suspended in a neutral oil injected into the dewlap.

The first three pairs took their ration well from the second day onwards. Pair D lost their appetite and looked miserable after a week.

This trial indicated that M.T.U. had little or no effect on the palatibility of a ration.

TRIAL IV

YOUNG FEEDER STEERS

In order to gain more information on the effects of M.T.U. feeding on young feeder steers, this trial was planned to study feed consumption, weight gains, carcass grading and optimal period of treatment.

Thirty young steers aged about two years were allotted into three groups of ten each on the basis of liveweight. Feed consisted of maize meal offered at 1 lb. per 100 lb. body weight and teff and soya bean hay *ad lib*. The meal was fed in equal portions twice daily. Three grammes M.T.U. powder were added to the daily meal ration of group A for the entire feeding period of 56 days. Group B. received a similar M.T.U. dosage for the last 28 days only while Group C served as untreated controls. All steers were in good condition and tame when they entered the trial.

Results and Discussion of Trial IV

Although the initial liveweights of all three groups were for all practical purposes the same, Group A gained 31 lb. liveweight per steer more than the controls. However, none of this gain was reflected in the dressed sarcass weight. Group B gained 29 lb. liveweight more per steer than the controls. Of this gain only 9 lb. or 30% was reflected in the dressed carcass weight. From this it can be concluded that M.T.U. feeding has a pronounced effect in causing ingesta to accumulate in the digestive tract.

As shown in Fig. 1. the liveweight gains of Group A were more rapid over the first 28 days than over the second.

The gain shown by Group B over the first period was lower than that of the controls although both groups were then receiving identical rations.

The administration of M.T.U. to Group B caused a rapid increase in liveweight gain parallel to that of Group A during the first period.

The average daily dry matter intake of the treated groups was significantly less than that of

TABLE 2

	TRIAL IV		
	Control	*Treated A.*	*Treated B.*
Initial condition of animals	Good	Good	Good
Preliminary feeding period (days)	—	—	28
M.T.U. feeding period (days)	—	56	28
No. of steers per group	10	10	10
Average initial liveweight (lb.)	707	697	701
Average final liveweight (lb.)	825	856	854
Average gain (lb.)	118	159	153
Average daily gain (lb.)	2.1	2.8	2.7
Average dressed weight (lb.)	453	452	462
Dressed weight as % of initial liveweight	64.1	64.8	65.9
% Carcasses grading prime or 1st grade	100	100	100
Average daily feed consumption (lb.)	27	23	24
Daily cost feed/steer (cents)	40	32.5	35
Overall fat	6.5	6.5	6.5
Marbling	0.7	1.1	0.7

the control group. In group B the food consumption over the second 28 days was significantly less than over the first 28 days.

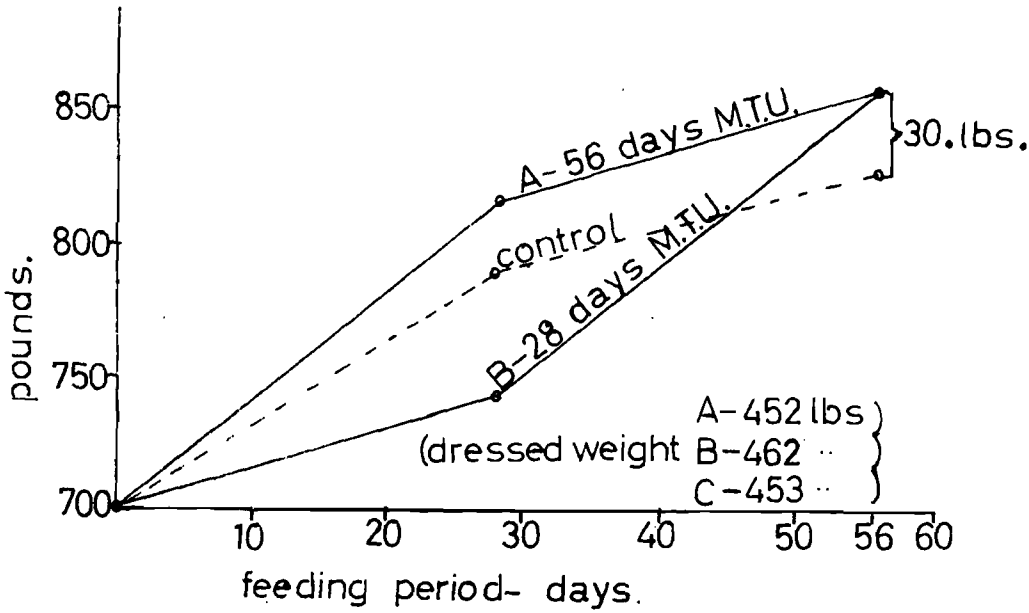
There was no difference between groups in regard to grading, marbling or overall fat. The excellent grading of all steers in this trial was no doubt due to their good condition at the start of the trial.

TRIAL V

MATURE FEEDER STEERS

In this trial 95 well grown Afrikaner type steers (6 to 8 tooth) were used. This is the first trial conducted on a cattle ranch under practical farming conditions. Facilities for the weighing of animals and transportation to the abbatoir were available. During the introductory feeding period, the steers were offered 1 lb. of maize meal daily with some silage and veld hay ad lib. The feeding pens were practically constructed but no overhead shelter was provided, representing average conditions appertaining to cattle feeding in this country.

The trial ration consisted of maize meal, a high quality sorghum and maize silage and good veld hay. The steers were kept in nine feeding pens and divided into groups as detailed in Table 3.



One fifth (19) of the steers served as untreated controls. Feed quality was excellent and offered *ad lib.* Rations were not identical for all groups since the farmer wished to experiment with different mixtures of the available feeds in order to devise the most economic ration. Different periods of introductory and M.T.U. feeding were also applied. Details are listed in Table 3.

All steers were dewormed prior to entering the trial and spraying for tick control was carried out at weekly intervals. The M.T.U. was offered by mixing a specially formulated wettable powder with water, and then pouring the freshly mixed suspension over the meal ration once daily (mornings) to the equivalent of 3 grammes active ingredient per animal per day. The medicated meal was readily consumed. The meal feeding was followed later in the day by silage and hay. Leftovers were weighed and recorded.

was obtained by treated animals over controls. This finding is vastly different from that obtained in Trial II and is probably directly related to the much better initial condition of the steers in Trial V as compared to the poor condition of the animals in Trial II.

In studying feed cost per 100 lb. gain, it became clear that extending the 35 day M.T.U. feeding period to 38 and 41 days, had no advantage. When considering the grading of carcasses it was found that the 35 day group produced 72%, the 38 day group 58% and the 41 day group 60% first grade and better carcasses. The 35 day M.T.U. feeding period therefore seems practical and probably a near optimal.

No deduction can be made from the lengthening of the introductory feeding period from 11 to 24 days. It seems that this period can differ provided animals are fed into a stage of good condition.

TABLE 3

(TRIALS V AND VI)

	TRIAL V		*TRIAL VI*	
	Control	*Treated*	*Control*	*Treated*
Initial condition of animals	Fair	Fair	Fair	Fair
Preliminary feeding period (days)	11 to 24	11 to 24	20	20
M.T.U. feeding period (days)	—	35 to 41	—	35
No. of steers per group	19	76	23	72
Average initial liveweight (lb.)	824	827	775	758
Average final liveweight (lb.)	906	934	—	—
Average gain (lb.)	82	107	—	—
Average daily gain (lb.)	2.3	2.9	—	—
Average dressed weight (lb.)	432	452	419	447
Dressed weight as % of initial liveweight	52.1	54.7	54.1	58.9
% Carcases grading prime or 1st grade	42	64.6	43	62.7
Average daily feed consumption/steer (lb.)	49.5	46.4	—	—
Total daily ration/steer (lb.)	—	—	43	43

Results and Discussion of Trial V.

The average initial liveweights of both groups were practically the same (824 and 827 lb.). The treated group produced a better final average liveweight per animal by 28 lb. This liveweight advantage was reduced to 20 lb. in dressed carcass weight, an unexpectedly high proportion compared with Trial II (grown steers) where the respective figures were 40 lb. and 17 lb.

The figure expressing the dressed weight as a percentage of the initial liveweight, is considered to be a reliable reflection of the efficacy of M.T.U. In this trial these percentages are 54.7 and 52.1% for treated and controls respectively. This is fairly similar to the results obtained in Trial II where the difference was 2%.

A significantly better carcass grading of 22.6%

In conformity with results obtained in Trials I, II, and IV, the feed conversion in the treated group was significantly better than in the controls, the reduction in feed-intake per treated animal per day being 3.1 lb. compared with about 4 lb. in Trial II.

Considering the treated steers in this trial producing somewhat better liveweights, slightly better dressed weights, significantly better feed conversion and better carcass grading (meat quality), it seems that M.T.U. feeding to grown steers holds some promise of economic advantage.

In order to obtain a clear picture of the effects of M.T.U. feeding on dressed carcass weights and grading, a further final trial was planned. This was again conducted on a ranch under practical farming conditions and on mature steers.

TRIAL VI

MATURE FEEDER STEERS

A group of 95 steers were used of which approximately one quarter (23) acted as untreated controls. The other 72 steers were given about 3 grammes M.T.U. each once daily as in Trial V. All the steers were older than three years and on the whole they were lighter than those in Trial V. The average initial liveweight per steer was slightly higher for the controls than for the treated steers viz. 775 lb. and 758 lb. respectively.

Feed offered consisted of good quality bean hay, sorghum and maize silage, corn-and-cob meal of high grade and good quality veld hay. The corn-and-cob meal was medicated with M.T.U. as in Trial V. Thereafter bean hay and silage was offered in the mangers while the veld hay was available ad lib in feed racks. Quantities offered are detailed in Table 3. The feed used in this trial is considered better than that offered in Trial V. The high daily silage ration of about 36 lb. per animal used in Trial V, was reduced to 25 lb. The ration proved very palatable. Feed consumption was not recorded since dressed weights and grading were to be the main criteria.

Unfortunately on account of a faulty weighbridge no reliable final liveweights could be taken and these figures are therefore omitted from Table 3.

All steers were held in pens, without roofing, in groups of twelve. Treatment against internal and external parasites was carried out as in Trial V.

Results and Discussion of Trial VI.

Notwithstanding the fact that the treated animals had an initial liveweight of 17 lb. less per steer than the controls, they finished with an average of 28 lb. more dressed weight per animal. The final liveweights and particularly the liveweight gains must therefore have been much better in the treated animals.

In this trial there was a difference of 4.85% in the figures expressing the dressed weight as percentage of the initial liveweight. This figure is much higher than those of Trials II and V (mature steers) where the figures were 2% and 2.6% respectively.

Treated animals graded distinctly better than the controls, as is reflected by the figure of 19.7%. This finding is fairly similar to that in Trial V. where a 21% better grading was obtained.

If it is assumed that both treated and control groups consumed the same quantities of feed, then the treated group registered a much better feed conversion since they started with a deficit of 17 lb. in liveweight and ended with 28 lb. better dressed weight per carcass.

Considering the findings in this trial it would seem that this particular ration had combined well with M.T.U. feeding.

CONCLUSIONS

The results obtained with two-year old steers were poor. The apparent liveweight gain obtained was mainly due to an increased weight of the digestive tract contents. The advantage in dressed weight was not more than three pounds per carcass. There was, however, a slight saving in feed used to produce virtually the same dressed carcass weight i.e. better feed conversion but this would be largely offset by the cost of the M.T.U.

The response of the mature steers was better. In the three trials in which this type of animal was used, the overall gain in dressed weight was some 22 lb. per carcass. When the dressed carcass weight is expressed as a percentage of the initial liveweight, the treated animals showed an overall advantage of some 3%. In two of the three trials the treated steers graded better than the controls. Overall, 58% of the treated steers (total 173) graded first grade or better as compared to 42% among the 67 controls.

In all the trials a better feed conversion was shown by the treated animals. The overall saving in feed amounted to some 4 lb. per animal per day.

The use of M.T.U. would therefore appear to hold some promise provided the following conditions are complied with:

1. The animals must be fully grown.
2. They must be in fair condition before M.T.U. is added to the ration. The initial condition appears to be the major factor in determining the grading and also influences the response to M.T.U. An adequate preliminary feeding period must therefore be allowed.
3. The optimal period of M.T.U. feeding would appear to be about 35 days.

The possible danger of M.T.U. residues in the meat to the consumer must be considered. In some countries the use of M.T.U. is prohibited. In the present tests, however, the level of residual M.T.U. in the tissues was zero or below 1 p.p.m. which would not represent any hazard. Nevertheless it is not impossible that consumer resistance to such "treated meat" may be met with should the practice become common.

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OBSERVATIONS ON A *HEPATOZOON*-LIKE PARASITE IN THE IMPALA

P. A. BASSON*, R. M. MCCULLY**, R. D. BIGALKE***, J. W. VAN NIEKERK****.

SUMMARY

A previously undescribed *Hepatozoon*-like parasite is reported in the impala (*Aepyceros melampus* (Lichtenstein, 1812)) from the Kruger National Park and its association with mild focal disseminated hepatitis and lymphadenitis described. The organism resembled *Hepatozoon canis* (James 1905)[1,2] very closely, was invariably intracellular and apparently represented a schizont with a single large merozoite.

Comparative histochemical studies of the organism in impala and *H. canis* in dogs showed that in both cases the residual cytoplasm of the schizonts as well as certain granules within the merozoites were intensely argyrophilic and the former also P.A.S-positive and mildly Gram-positive.

INTRODUCTION

Species of *Hepatozoon* have been described from reptiles, birds and mammals, being particularly common in rodents[3]. *H. canis* from the dog was originally discovered by Bentley[2] and its development in the intermediate host, *Rhipicephalus sanguineus,* initially described by Cristophers[4] and later by Wenyon[5]. Dogs apparently become infected by ingestion of ticks harbouring sporozoites of *H. canis*[3]. Schizonts have been seen in the spleen, bone marrow and liver[4,5,6]. Merozoites varying in size and number have been described; some are thought to perpetuate schizogony while others invade leucocytes to form gamonts[5]. Prior to formation of merozoites the schizont has an abundance of foamy cytoplasm, most of which is retained as a residual structure in the mature schizont. The parasites may be encountered in apparently healthy animals, but are also said to cause serious disease and death[6,7,8]. The first fatal case in dogs in South Africa was reported by Porter[7]. In wild ruminants so-called leucocytogregarines, probably gametocytes of a *Hepatozoon* sp., were found in the mononuclear leucocytes of a reedbuck (*Redunca arundinum*) as well as in the lymphocytes of a giraffe (*Giraffa camelopardalis*). In the cases in the impala that are being presented, organisms resembling *Hepatozoon* were found in association with hepatic and lymphoglandular lesions.

MATERIALS AND METHODS

During a study of besnoitiosis in impala[11], banana-shaped micro-organisms were found within hepatic microgranulomas. Subsequently all tissues collected from impala for this or other studies and livers from those shot for meat rations and other purposes were studied microscopically for the presence of similar lesions and parasites. Seventy-five impala were thus examined during March-April (1965, 1966) and 38 during August-September (1964, 1965). The tissues, which were preserved in 10% formalin, were embedded in paraffin wax, sectioned at 3 microns thickness with a sliding microtome and stained with haematoxylin and eosin (H.E.). Giemsa, Gram[12], oil red 0[13], Gomori's methenamine-silver nitrate[14] (G.M.S.) and Hotchkiss periodic acid-Schiff (P.A.S.)[15] techniques were used as special methods in order to study some histochemical aspects of the parasite. Nine organisms were measured with the aid of an ocular micrometer.

Specimens from positive cases in dogs were collected, preserved and treated similarly for comparative parasitological studies.

RESULTS

Some of the livers examined showed a few small and widely distributed inflammatory foci (Fig. 3). Their distribution in the lobules varied, but was usually either midzonal or within the portal areas. The lesions consisted mainly of hypertrophic reticulo-endothelial (r.e.) cells and a small number of lymphocytes and plasma cells

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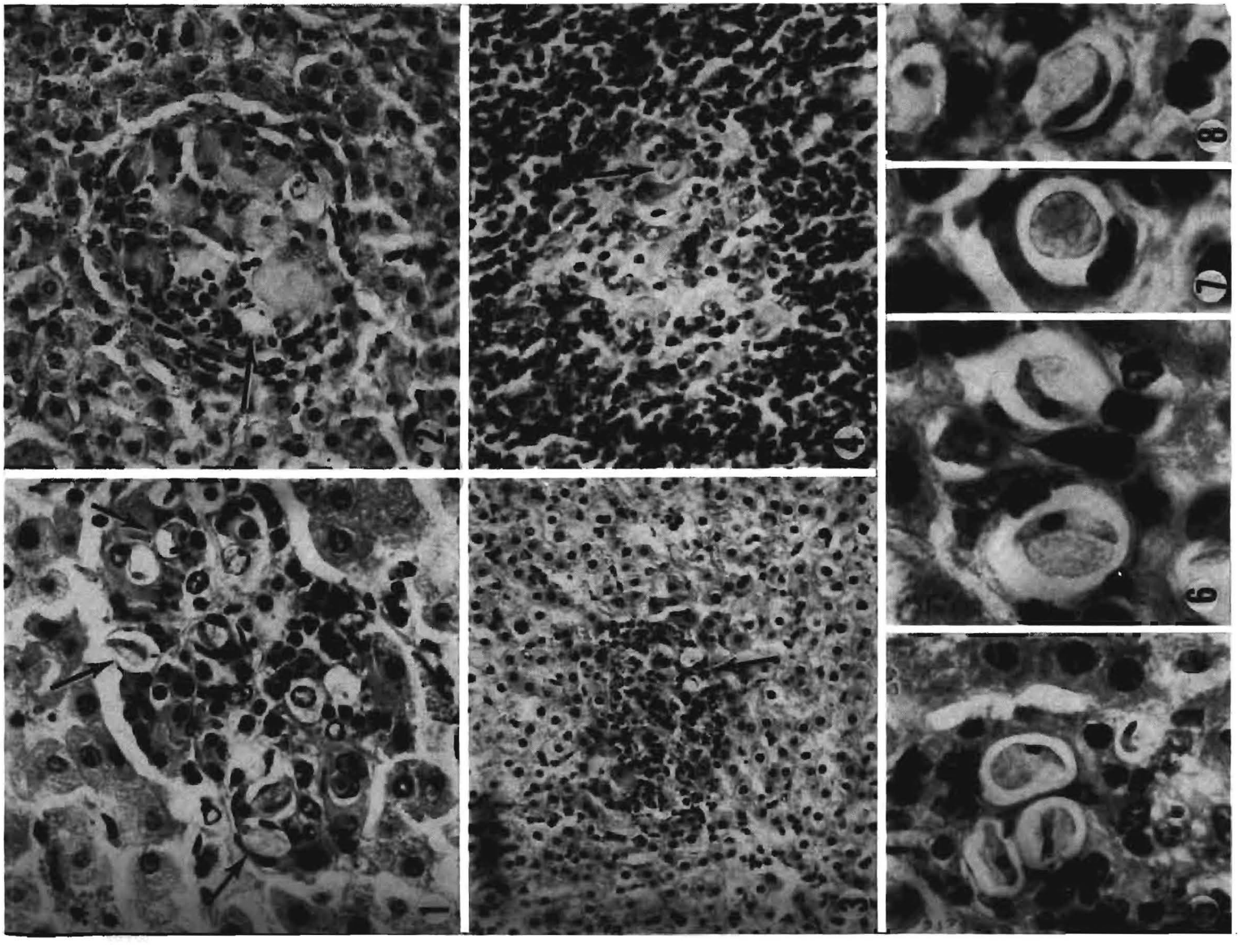

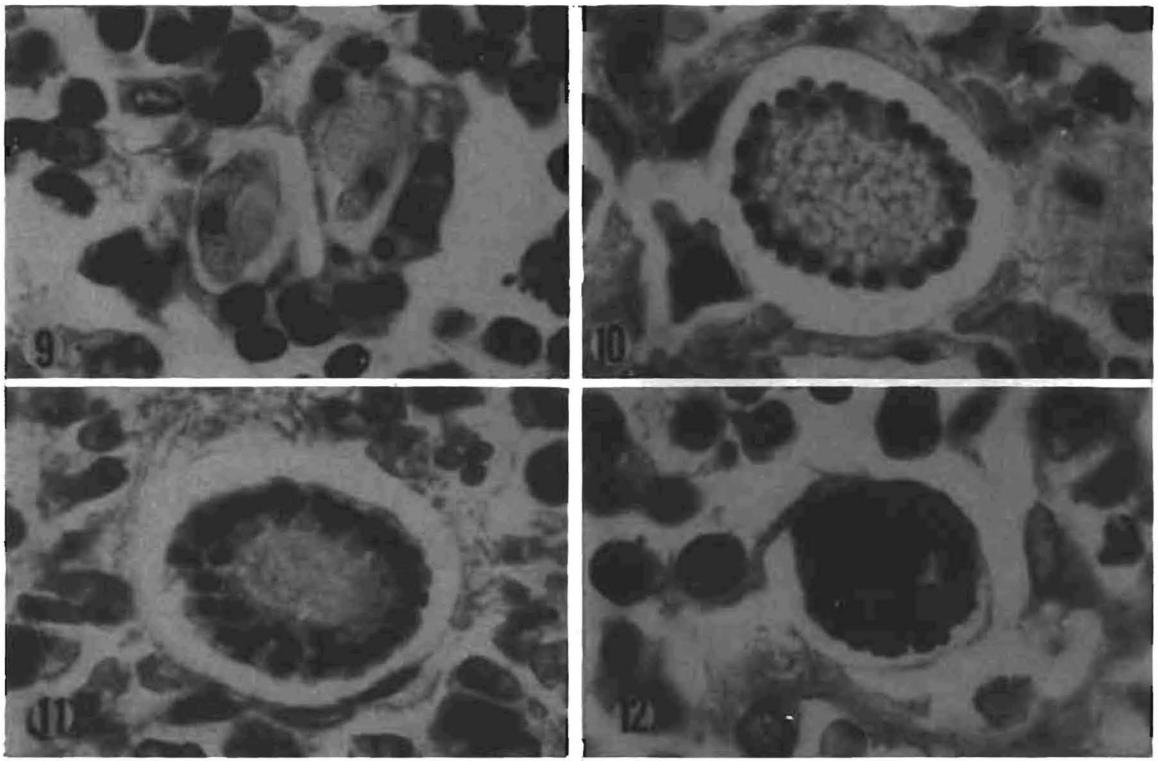

PLATE I.

Photomicrographs of lesions and organisms in three impala (1, (2-7), 8).

Fig. 1: Liver. Mild epithelioid and round cell reaction and several organisms in longitudinal and cross section (arrows). H.E. X 480.

Fig. 2: Liver. Microgranuloma with several giant mononuclear epithelioid cells. One organism is present on the left (arrow). H.E. X 305.

Fig. 3: Liver. Another microgranuloma containing several organisms. H.E. X 200.

Fig. 4: Lymph node. Small focus showing the epithelioid reaction. An organism is present on the right. H.E. X 305.

Fig. 5: Liver. Four parasites in a lesion. H.E. X 770.

Fig. 6: Liver. Two organisms with their typical banana-shape and adjacent residual mass of cytoplasm. H.E. X 1200.

Fig. 7: Liver. Parasitized cell showing the residual cytoplasm only. H.E. X 1200.

Fig. 8: Liver. Section through a parasitized cell with a compressed eccentric nucleus. H.E. X 1200.

PLATE II.

Hepatozoon canis in the dog.

Fig. 9: Lymphnode. Two schizonts, each with a single large merozoite. The adjacent residual cytoplasm can also be seen. H.E. X 1200.

Fig. 10: Spleen: Typical schizont showing the developing peripheral zone of small merozoites and inner residual foamy cytoplasm. H.E. X 1200.

Fig. 11: Spleen: Well-shaped small merozoites in a more mature schizont. H.E. X 1200.

Fig. 12: Lymphnode. Schizont showing the intense argyrophilia of the residual cytoplasm. G.M.S. X 1200.

(Fig. 1). Some of the enlarged r.e. cells exceeded the parenchymal cells in size and contained an abundance of eosinophilic cytoplasm (Fig. 2). Banana-shaped intracellular organisms with an adjacent residual foamy and faintly basophilic cytoplasmic mass were invariably present in these lesions and measured approximately 15 x 7.5 μ (Fig. 5-8). The individual width of the parasite, without the adjacent residual cytoplasm, varied between 2.5 and 3 μ. Its nucleus was distinct, circular and usually situated centrally. The organisms were located within the cytoplasm of the r.e. cells and clearly surrounded by a halo. Each parasitized cell apparently contained only one organism and the eccentric nucleus was frequently compressed to a semilunar structure. Some cells appeared to contain a more basophilic cytoplasmic mass without well-defined organisms.

Of the 113 impala examined, livers from every animal, spleens from 15% and lymph nodes from 9% were studied microscopically. Livers of eight animals (7%) contained lesions and organisms; seven were obtained during late winter to early spring (August-September) and only one in late summer to early fall (March-April). Specimens from spleen and lymphnodes were studied in only three of the eight positive cases. One of these lymphnodes contained foci of hypertrophic reticular cells (Fig. 4), in which the same organisms were present as seen in the liver. Examination of all other tissues, including the spleen, was negative for similar lesions and parasites.

The comparative morphological studies on the parasite in the impala and *H. canis* showed that it was very similar to the large merozoites of the latter (Fig. 9). Furthermore, the histochemical studies revealed similar affinities for certain stains. With G.M.S. the residual foamy cytoplasm, which with H.E. was either refractory or faintly basophilic, proved to consist of deeply staining argyrophilic globules (Fig. 12). It was also mildly Gram-positive (light cobalt blue) and P.A.S.-positive, but refractory to Giemsa and oil red 0. The organisms also contained a few G.M.S.-positive granules within their cytoplasm.

The histopathological and other findings on cases of canine hepatozoonosis will be described in a subsequent report.

Discussion

The invariable association of the organism concerned with the microgranulomatous lesions leaves no doubt that these were provoked by the parasite. In our comparative study of the localization, morphology and staining characteristics of the organisms, the close similarity of the parasite in the impala to the large merozoite of *H. canis* was evident. It also corresponds with the description of the large merozoite of *H. canis* by Wenyon[5], who reported certain schizonts in dogs containing one or more of these large merozoites. However, the typical *Hepatozoon* schizont (Fig. 10, 11) with the collar of small peripheral merozoites surrounding the central mass of residual foamy cytoplasm could not be found in the eight positive cases in the impala; a possible explanation for this is the somewhat remote possibility that the impala is an aberrant host to the parasite. It spite of the close similarity of the organism of the impala to known *Hepatozoon* spp., it is therefore evident that further research is required before it can be identified unequivocally.

The argyrophilia of the residual cytoplasm and certain intracytoplasmic granules in the merozoites of the parasites of both dogs and impala, as well as the mild Gram-positive and P.A.S.-positive nature of the former, show promise of being useful aids in the examination of tissues for, and the identification of, these haemogregarines.

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THE DIAGNOSIS OF MASTITIS BY DIRECT AND INDIRECT CYTOLOGICAL METHODS

W. H. GIESECKE*
and
L. W. VAN DEN HEEVERϕ

SUMMARY

The cytology of normal and abnormal bovine mammary secretion is discussed and the diagnosis of mastitis by direct and indirect methods of cell content determination is critically examined. The limitations of such tests in an organised mastitis control programme are pointed out, and their unjustified sole use in assessment of faulty milking techniques and the success of recent mastitis therapy is emphasized.

INTRODUCTION

Recent publications show widespread interest in the availability of the California Mastitis Test (CMT)[1–4] and propose its use as a simple, reliable and ideal method for a national mastitis control programme[1]. It therefore seems expedient to direct attention to the cytology of milk in general and to the available literature regarding the CMT in particular.

Every increase in cell content of milk, without necessarily being associated with clinical symptoms, may arouse suspicion of the existence of an inflammatory process in the mammary gland, whether of infectious or aseptic aetiology. However, a variety of other factors which cause a physiological increase in cell numbers in milk have to be excluded, e.g. during the colostral period[5 6] and at the end of lactation[7], the effects of individuality or age[7 9], breed or regional differences[10 12] or factors responsible for induction or predisposition of the udder to mastitis and thus possibly influencing the cell content of milk, such as inadequate or faulty care, management, nutrition, milking technique and hygiene, seasonal climatic influences, milk yield and duration of lactation, hormonal influences, constitution or inherited disposition[13–32].

The widely fluctuating variation in the symptoms of pathological processes in the mammary gland results from a reciprocal interaction between "irritational capacity of the micro-organisms" — or of the traumatic stimulus in the case of aseptic or non-specific mastitis — and the "reactional capacity of the udder tissue[33–36]. These are factors which occasionally succeed in spontaneous elimination of mastitis organisms from the ducts of the mammary gland. However, with the removal of the infectious or non-infectious harmful agents there is no parallel immediate termination of the inflammatory process in the udder tissue[37], the latter following the former rather gradually, and resulting in a slow reduction of the cell content of the milk before its eventual return to normal. Severe cellular reactions have also been observed after intracisternal administration of modern mastitis therapeutics[37–41] so that in summary it can be stated that an increased cell content does not provide any evidence of an infectious cause but is simply to be judged as an expression of an inflammatory defence mechanism.

Although there is no doubt that a certain number of cells occur in every normal milk, the various factors which influence the cell content have resulted in a variety of opinions concerning the value of determinations of the kind, number and percentage of its cellular components. On the one hand, any wider importance is denied[42 43], whilst on the other hand the significance of a satisfactory quantitative cytological examination of milk for human consumption is emphasised[44]. This point of view is particularly stressed by the existence and increasing incidence of aseptic or non-specific forms of mastitis and their importance as inducing or predisposing factors in the epidemiology of infectious mastitis[45], as well as the observations made when testing the reaction of udder tissue to the intracisternal administration of modern mastitis therapeutics. However, for the purpose of mastitis control and the diagnostic necessity of obtaining details regarding the causative agent as well as of damage done to the udder tissue, both cytological and the usually recom-

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TABLE 1.—TYPES OF CELLS IN NORMAL MILK OF COWS.

BACHMANN 1932	ZLOTNIK 1942	BLACKBURN & MacADAM 1954	SCHONBERG 1956
1. *polymorphonuclear leucocytes* *a.* neutrophilic polymorphonuclear leucocytes *b.* eosinophilic polymorphonuclear leucocytes	1. *polymorphonuclear leucocytes* *a.* polymorphonuclear leucocytes *b.* atypical neutrophile polymorphonuclear leucocytes *c.* eosinophilic polymorphonuclear leucocytes	1. granular leucocytes *a.* neutrophilic. *b.* eosinophilic *c.* basophilic	1. polymorphonuclear leucocytes *a.* neutrophilic polymorphonuclear leucocytes. *b.* eosinophilic polymorphonuclear leucocytes. *c.* basophilic polymorphonuclear leucocytes.
2. lymphocytes	2. lymphocytes	2. lymphocytes	2. lymphocytes.
3. monocytes *a.* common monocytes *b.* large monocytes *c.* mononuclear cells with fat inclusions and fat-loaded epithelials.	3. monocytes		3. monocytes.
4. epithelials	4. epithelials *a.* epithelials with clear cytoplasma *b.* epithelials with neutrophilic foamy cytoplasma *c.* epithelials with acidophilic foamy cytoplasma *d.* epithelials with basophilic foamy cytoplasma *e.* pseudopolymorphonuclear cells (degenerated epithelials) *f.* micropseudo-cells (disintegrating pseudopolymorphonuclear cells)	3. epithelials *a.* with vacuoles in cytoplasma *b.* without vacuoles in cytoplasma	4. epithelials *a.* squamous. *b.* cylindrical *c.* cuboidal.
	5. large squamous cells (from teat skin and teat canal)		5. giant cells
			6. erythocytes
			7. disintegrating forms of all kinds of cells (Nissen's bodies)

TABLE 2.—AVERAGE PERCENTAGES OF DISTRIBUTION OF THE CELLULAR COMPONENTS IN NORMAL BOVINE MILK.

Types of cells in milk	% of Total Cell Count ml Milk					
	Wirth (1930)	Bachmann (1932)	Zlotnik (1947)	Schönberg (1956)	Teute & Welz (1961) cells per micro-field of Prescott-Breed milk smear	Dilbat (1963)
neutrophilic polymorph. leucocytes	44	60.7	—	50–70	0.7	—
eosinophilic polymorph. leucocytes	3	0.4	—	0–3	—	—
polymorph. leucocytes	—	—	—	—	—	35.2
lymphocytes	36	26.7	1–18	25–35	0.5	23.4
monocytes	—	5.9	—	5–15	—	—
mononuclear granulocytes	17	—	—	—	—	—
mononuclear cells with fat inclusions	—	0.2	—	—	—	—
large squamous cells	—	—	1–5	—	—	—
epithelials	—	6.1	55–85	—	4.5	41.4
pseudopolymorphonuclear cells	—	—	6–40	—	—	—
micropseudo-cells	—	—	1–20	—	—	—

mended bacteriological examinations[43–46] are necessary. Only a combined examination[47], preferably consisting of several but at least two of the methods mentioned above can furnish satisfactory data essential to the establishment of a diagnosis which is as correct as possible. Such a diagnosis is necessary to permit reliable prognosis and advice, for the application of the most economical and effective treatment and to gain the cooperation and trust of the dairy farmer. The farmer's willingness to collaborate is generally regarded as fundamental for the success of any mastitis control programme.

Determination of the cell types and the average percentage of their distribution in milk is based on the findings of various authors[48–54] and summarised in table 1 and 2.

One group of authors[14 42 48 49 51 52 55–60] regards the neutrophilic polymorphonuclear leucocyte as the prevailing type of cells in normal bovine milk. Others[50 61–63] consider epithelials predominant and the appearance of neutrophilic and eosinophilic polymorphonuclear leucocytes or monocytes in milk as a qualitative shift from the normal nature of udder secretion to indicate the onset or existence of a secretional disturbance or even mastitis. A third group of investigators[9 12 45 64–66] agrees on the prevalence of epithelial cells and the existence of white blood cells in normal milk as long as the latter do not exhibit clumping, phagocytosis or degeneration.

Similarly, opinions differ regarding the total cell content/ml of normal raw milk. Summarising[11 12 49 52 54 56 62 63 67-77], table 3 indicates that recent publications generally agree on a maximum total cell content of 150 000-300 000 cells/ml in normal raw milk of individual samples[12 52 54 62 63 73 74].

TABLE 3.—THE TOTAL CELL CONTENT/ML OF NORMAL RAW BOVINE MILK.

Author	Year	Cell count/ml
Prescott & Breed	1910	1,485 000
Skar	1912	450–500 000
Christiansen	1929	963 000
Bachmann	1932	59 750
Cherrington a.o.	1933	50 000
Grassi	1933	120 000
Wayne & Macy	1933	1,252 000
Wilde	1938	200 000
Klimmer & Schönberg	1939	20 000–500 000
Könz	1955	150 000
Schönberg	1956	150 000
Whaby & Nasr	1957	250 000
Klastrup	1960	180 000
Moursy & Obiger	1960	300 000
Obiger	1961	300 000
Seelemann & Meyer	1963	300 000
Dilbat	1963	160 000
Kästli	1963	300 000
Seelemann	1964	100 000

Analysis of cellular milk components is usually performed by microscopic examination of a stained milk sediment smear[52 78 79] or of a whole milk smear[67 68].

Although the variable cell distribution in the sediment of centrifuged milk has been repeatedly investigated and discussed[49 54 59 64 67 68–74 78 80 86] such sediment slides are still employed for cell differentiation as well as for routine examination of milk samples for mastitis. A relatively even cell distribution in the smear examination of some microscopic fields permits a practical estimation of the cell content[47]. This is sufficiently accurate for the evaluation of milk cell content[9 46 87] and can be standardized[60 88] to some extent. In addition, this method more frequently permits the demonstration of specific mastitogenic micro-organisms than is the case when examining a whole milk preparation[78], even though the latter is recommended as a more accurate enumeration of the cellular milk components[59 81 89]. Despite an average error of 15-30%[07 90] which is particularly evident in milk of low and very high cell content, errors of counting and differentiation are generally within a limit which nevertheless warrants use of this whole-milk smear method for comparative and evaluation purposes. These relatively exact laboratory methods — however much effort and time they require — permit evaluation of the total cell or leucocyte content and the diagnostically important detection of leucocyte clumping, phagocytosis of micro-organisms, cell degeneration and nuclear disintegration.

For many years the examination of milk for secretional disturbances of the mammary gland under field conditions was largely limited to macroscopic examination with a strip cup or indicator papers. By these methods mostly only quarters already showing clinical symptoms or with advanced secretional disturbances could be identified. However, since the introduction of the Whiteside test[90], the modified Whiteside test[92], the antiformin test[93], and particularly by the further modification of the Whiteside field test[94] to the California Mastitis Test (Schalm Test)[95], it is now possible to obtain a more convenient estimation of the cell content directly in the stable.

The California Mastitis Test (CMT) is a presumptive double test and has been described as a provisional method for estimating the cell content of milk[96]. As a screening procedure it is thought

capable of detecting glands producing milk of high cell count before macroscopically visible changes in milk secretion occur and the gland parenchyma furnishes evidence of severe functional disturbances[87]. Apart from brom cresol purple to serve as pH indicator and furnish colour contrast to milk, the CMT test solution contains alkyl- or alkylary-sulphate or -sulphonate or other detergent substances [12 25 47 98–102] some of which constitute the basis of modifications of the CMT[103–105].

Existing literature indicates the principle of the CMT to be the reaction of an anionic detergent with proteins of the milk cells. By their denaturation and by forming a protein-detergent-complex a macroscopically detectable increase in the milk vicosity is produced. The specific cell-protein group included in this reaction was found to be particularly associated with the nuclei of the leucocytes[74] (D.N.A.) which increases in direct proportion to the cell content in cases of mastitis[86 88 98 105–107]. Besides the cellular components of milk, other factors such as fat and protein content were once thought to affect the CMT[87 100] but the false results found in applying the CMT were not associated with cream, casein, albumin, and globulin in the milk[46]. Finally, the mechanism of the CMT was explained as a reaction between the anionic surface-active test fluid and the cationic cell membrane whereby the membrane potential is broken down. The membrane itself disintegrates, the detergent reacts with the DNA of the nucleus, and the mixture becomes more viscous. For this reason a negative CMT reaction in milk of increased cell content could be explained by decreased electrochemical activity of the cell membrane[99].

The CMT method has been considered capable of detecting mastitis in the incipient stages, to locate quarters affected with chronic mastitis, and to evaluate the effect of mastitis therapy and changes in management on udder health. Using the original and substituted detergents, the CMT has been subjected to numerous investigations at different lactation periods[87 108 109], in chronic udder infections[97 110 111], on can milk samples[87 108 111–113] against known cell content, and in comparison with catalase thybromol reactions and chloride content of milk [87 109 111 113–115]. From these investigations the value of the simple, rapid, inexpensive CMT appears to be its ability to provide an estimate of the cell content of milk within certain limits, to detect secretional disturbances of the mammary gland when taking into acount physiological conditions, and to distinguish healthy from diseased animals in herds to be treated. These favourable opinions on the CMT and other rapid field screening tests[22 25 54 87 98 101 102 108 115 119] are opposed by many who are sceptical[36 46 47 99 109–111 120–124]. Although admitting the generally orientating ability of the CMT, these latter authors reject the test as a screening method for mastitis control because its accuracy is contested and its ability to give reliable information about the cell composition and to detect chronic infections is questioned. Furthermore, the reaction appears to be rather variable in the critical cell content ranges, it is influenced by the number of micro-organisms in the milk sample, and often gives positive results particularly with can milk in which an increased cell content cannot be confirmed. For these reasons and the particular importance of the hidden and subclinical cases in a mastitis control programme, one is inclined, after scrutiny of the available information in the literature and own investigations concerning the CMT as diagnostic aid for the detection of mastitis, to consider the CMT of rather limited value as a basic method for a mastitis control program. This opinion is formed with regard to accurate diagnoses, specific therapy with modern mastitis therapeutics, and the culling of intractable or uneconomical mastitis cases — all basically necessary in a mastitis control campaign. Because of numerous false results the CMT is neither capable of minimizing the acknowledged importance of a combination of various methods [46 47 108 124 126] nor of replacing the bacteriological and microscopic-cytological examination.

Conclusions

As an initial test rather than the ultimate one, the CMT has limited practical value as a screening test to assess the udder health situation of a herd, the influence of milking techniques, or the slow recovery of a recently treated diseased quarter. However, it is not advisable to base milk sampling for laboratory examination, therapy, or herd segregation on results of the CMT. For this, more accurate diagnostic methods are required to show whether or not there is an infectious cause of the secretional disturbances.

It is considered necessary to stress that in assessing the effect of various milking techniques and the functional efficiency of milking machines, it is most hazardous to recommend or practice the test without certain knowledge of its advantages and disadvantages, and that it is most unwise to interpret the CMT reaction without previous knowledge of the bacteriological status of the udders or quarters concerned. Assumption that positive CMT reactions are the result of milking trauma would certainly be incorrect in many South African herds where lowgrade or subclinical

bacterial forms of mastitis are frequent. Similarly, the practice of using only the CMT to assess the success or otherwise of recent antibacterial mastitis therapy cannot be too strongly condemned in view of the known irritant effect of many available mastitis therapeutics for intramammary use and the slow return of cellular content of the secretion of successfully treated quarters. It is clear that there is as yet no single readily performed test for mastitis which can take the place of conventional laboratory methods. The danger lies in failure to provide facilities for laboratory examination of milk for mastitis as long as simpler and less expensive methods are available.

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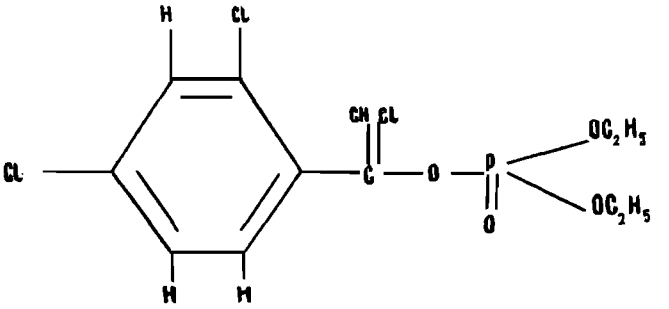

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PARATUBERKULOSE (JOHNE SE SIEKTE) BY 'N INGEVOERDE DUITSE MERINO RAM

O. T. VAN NIEKERK* en K. VAN DER WALT**.

SUMMARY

A case of Paratuberculosis (Johne's disease) in an imported German Merino ram is described. This is the first known report of this disease in a sheep in South Africa.

OPSOMMING

'n Geval van paratuberkulose (Johne se siekte) by 'n ingevoerde Duitse Merino ram word beskrywe. Dit is die eerste bekende rapport van hierdie siekte in 'n skaap in Suid-Afrika.

INLEIDING

In die afgelope 15 jaar is paratuberkulose op 17 verskillende plekke in die Republiek gediagnoseer. Al hierdie gevalle het in beeste voorgekom waarvan 11 by ingevoerde diere was. Ten spyte van gereelde invoer van skape uit Brittanje en Wes-Europa is geen gevalle voorheen in ingevoerde of inheemse skape gediagnoseer nie.

GESKIEDENIS

'n Groep bestaande uit een ram en vyf ooie is in November 1965 van dieselfde stoetery in Duitsland ingevoer.

Die ram is op 9 September 1966 in die departement Geneeskunde te Onderstepoort opgeneem met 'n geskiedenis van diarree gedurende die voorafgaande vyf weke. Gedurende hierdie tyd het die ram, volgens die eienaar, baie gewig verloor en het die dier 'n onderbroke koors en 'n variërende aptyt gehad. Daar was deurgaans 'n hardnekkige maagwerking wat, ten spyte van behandeling met antibiotika en antidiarreemiddels, nooit volkome herstel het nie.

Voor die begin van die siekte was die ram in 'n uitstekende kondisie en het hy 'n aantal ooie bevrug.

SIEKTETEKENS

Met opname het die ram 'n koors van 103.4°F gehad en was daar tekens van 'n groen waterige diarree. Die dier het origens gesond en lewendig voorgekom en het geen ooglopende afwykings getoon nie.

Misondersoek was negatief vir parasieteiers. Totale plasmaproteïen was 5.46 gram per 100 ml met albumien 1.40 en globulien 4.06 gram per 100 ml respektiewelik. Totale leukosiettelling was 21,000 met 82% neutrofiele en 18% limfosiete. Alle ander laboratoriumbepalings se waardes het binne die normale grense geval.

Gedurende die twintig dae in die hospitaal was die aptyt deurgaans redelik tot goed en die mis was meesal styf maar nie korrelrig nie. Die koors het tussen 103°-104°F varieer en van die tiende tot die veertiende dag na opname is die dier daagliks 440 mg rolitetrasiklien* binnespiers gespuit met geen noemenswaardige verbetering van die koors of kliniese beeld nie.

Twee dae voor dood het die koors tot 105°F gestyg, die asemhaling het versnel, eetlus het verminder en die dier het lusteloos voorgekom. Die mis het vloeibaar geword en enkele ure voor die dood was daar tekens van strawwe koliek met rekbewegings, geskop na die buik en steungeluide. Hierdie tekens het kort voor die dood bedaar toe tekens van ineenstorting reeds teenwoordig was.

POST MORTEM

By nadoodse ondersoek was daar kongestie en sianose, hemorragiese enteritis, mesenteriale limfadenitis, buikwatersug, degenerasie van die miokard en lewer, en 'n nefrose.

Behalwe die proksimale 25 cm van die duodenum was die dermkanaal met 'n rooibruin bloederige vloeistof gevul. Die slymvlies was verdik, dof en die oppervlakte was gerimpeld. Daar was dilatasie van die caecum en colon en vergroting van die mesenteriale limfknope, die grootste waarvan 7 x 5 x 5 cm gemeet het. Die limfknope was baie edemateus, sonder duidelike afbakening van korteks en medulla. Die gesnyde oppervlakte was vlekkerig en dof met petechiale bloedings. Redelike

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hoeveelheid vet was nog teenwoordig en daar was geen tekens van uittering nie.

Smere van die dermslymvlies het die teenwoordigheid van suurvaste organismes getoon en histopatologies was daar verdikking van die dermslymvlies met massale infiltrasie van epiteloïedselle, limfosiete en plasmaselle, met enkele polimorfe in die mucosa en submucosa. Groot getalle suurvaste organismes was in die sitoplasma van die epiteloïedselle teenwoordig en 1g. selle met suurvaste organismes is ook in die limfknope waargeneem.

BESPREKING

Paratuberkulose word beskryf as 'n langdurende chroniese siekte gepaard met uittering (Marsh 1965). In hierdie geval was die totale verloop van die siekte slegs sowat agt weke en die skielike ontwikkeling van 'n bloederige enteritis wat tot die dood gelei het kon, in die lig van beskrywings van die tipiese verloop van die siekte, nie verklaar word nie.

Uit vorige ondervinding met besmette beeste in Suid-Afrika skyn dit nie of die besmetting tot dusver inslag gevind het nie, maar of dieselfde vir skape geld is uit die aard van die saak glad nie bekend nie. Dit is bevind (Lovell *et al* 1944) dat die organismes vir so lank as 246 dae, maar nie vir 284 dae nie, in mis kan voortleef, en gesien in die lig van 'n moontlike bedreiging van ons nasionale skaapkudde sal hierdie fokus van besmetting vir 'n lang periode fyn dopgehou moet word.

BEDANKINGS

Graag wil ons waardering uitspreek vir die hulp en inligting deur dr. P. R. Mansveldt van Veeartsenydienste, verskaf, en die Hoof, Veeartsenykundige Navorsingsinstituut bedank vir toestemming tot publikasie hiervan.

VERWYSINGS

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MARSH, H. (1965). Newsom's Sheep Diseases (3rd Ed.) *London. Bailliere Tindall and Cox.*

BOOK REVIEW

Radioaktivität und Veterinärmedizin. K. H. Wegener. 1966. Paul Parey, Berlin and Hamburg. 228 pages including index, 33 illustration, 49 Tables and numerous appendices. D.M. 43.00.

This book represents an excellent presentation of the biological effects of high-energy radiations on the tissues of domestic animals. Although its opening chapters deal with the physical principles concerned in electro-magnetic radiations, ionisation, nuclear fission and the measurement of such phenomena, it is by no means a book for use by the casual reader. The author presupposes a fairly comprehensive knowledge of radiation phenomena. The text is in German and free use is made of German scientific terminology, which makes this book difficult to read for those whose knowledge of the language is elementary. It is however a book of great value to those with a knowledge of German scientific literature.

The opening chapters are followed by sound discussions on the effects of ionizing and electromagnetic radiations on living tissues, the radiation syndrome as seen in animals and birds, pathological considerations and basic principles involved in treatment of the syndrome. The problem of fall-out in relation to animal and human health is well covered and will be of great interest to those veterinarians concerned with public health. In this regard there is a comprehensive discussion on the use of animals which have suffered radiation damage and the dangers inherent therein to the human consumer. Contamination of milk consequent to fall-out receives the necessary attention. World-wide fall-out in relation to the testing of nuclear devices is dealt with in the same context. A useful section on the use of radiation in food preservation and storage follows. The discussion is unfortunately rather in the nature of an introduction to this important topic.

The use of isotopes in research and practice is covered very briefly. This is surprising in view of the mass of world literature in relation to this aspect of Veterinary science.

The latter third of the book is devoted to the control of isotope use, particularly in Germany, dosimetry, organization of work with radionuclides and radiations and various legal aspects. The sections concerned give valuable hints to persons concerned with this aspect of radioactivity. The book is concluded with a comprehensive bibliography.

Printed on good semi-glossy paper, well-bound and profusely illustrated with line drawings and tables, this book should make a very useful addition to the bookshelves of all interested in this subject.

J. M. M. B.

A NOTE ON THE TOXICITY OF THE PLANT *PACHYSTIGMA THAMNUS.* ROBYNS

T. F. ADELAAR and M. TERBLANCHE*.

SUMMARY

Details are presented showing that the plant *Pachystigma thamnus* is toxic and causes the disease "Gousiekte".

INTRODUCTION

Gousiekte is a chronic disease of ruminants characterised by sudden death due to cardiac failure following on a chronic interstitial myocarditis[1 2]. There is a latent period of 1-8 weeks after ingestion of certain plants. *Pachystigma pygmaeum* (Schtr.) Robyns was incriminated as a cause of the condition by Theiler in 1923[1]. Later, *Pavetta harborii* S. Moore[3] was also proved to cause an identical syndrome. It has long been accepted that *Pachystigma thamnus* is the cause of Gousiekte in ruminants in the north eastern parts of Natal[4 5] but no experimental proof of this has yet been presented in the literature.

MATERIALS AND METHODS

The plants were collected in north eastern Natal and identified by the Botanical Research Institute, Pretoria. The material was dried in the shade and then ground to a fine powder. This was suspended in water just prior to dosing through a stomach tube to Boer-goats at a rate of 200 g plant material per day. Four-tooth to adult female and castrated male goats were used.

RESULTS

Three of the five experimental goats developed typical Gousiekte symptoms[6 7] and lesions[2]. (See table 1). The fourth, which received a relatively low dosage, developed a systolic murmur but survived. It was eventually slaughtered on the 180th day. Post mortem examination showed hydrothorax and a dilated heart. On histological examination a mild focal fibrosis of the myocard was observed which is indicative of gousiekte. The fifth goat, which received an even lower dosage, developed no symptoms or lesions.

DISCUSSION

Pachystigma thamnus Robyns occurs mostly in north eastern Natal i.e. in the districts of Utrecht, Newcastle and Dundee and differs from the well-known *Pachystigma pygmaeum* (Schtr.) Robyns (Gousiektebossie) mainly in that it has smooth leaves whereas *P. pygmaeum* has yellow hairy leaves[5]. The plant is toxic to ruminants and is one of five plants now known to cause Gousiekte.

These are:

Pachystigma pygmaeum (Schltr.) Robyns[1]
Pavetta harborii Moore[3]
Fadogia monticola Robyns[8]
Pavetta schumaniana F. Hoffm.[9]
Pachystigma thamnus Robyns

It is noteworthy that all these plants belong to the family Rubiaceae.

TABLE 1.—RESULTS OF DOSING 200 G DRIED *Pachystigma thamnus* ROBYNS MATERIAL DAILY TO GOATS.

Goat No.	Weight in kg	No. of dosages	Total plant dosages		Exper. day of death	Histological Result
			Actual (kg)	as g/kg		
1	23.7	60 in 89 days	12.0	ca 500	89	positive
2	37.2	62 in 92 days	12.4	ca 330	93	positive
3	39.1	41 in 50 days	8.2	ca 210	51	positive
4	40.0	18 in 24 days	3.6	ca 90	Slaughtered on day 180	suspicious
5	44.5	15 in 18 days	2.9	ca 67	Slaughtered on day 100	negative.

*Veterinary Research Institute, Onderstepoort.

ACKNOWLEDGEMENTS

The Chief, Veterinary Research Institute, Onderstepoort is thanked for permission to publish in this journal as are the State Veterinarian, Ladysmith for submitting the plants and Prof. J. D. Smit for the histological examinations.

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VETERINARY OPERATIVE SURGERY

DR.DR.h.c. EWALD BERGE AND DR.DR.h.c. MELCHIOR WESTHUES

Foreword by G. FORMSTON

Translated by WALTER G. SILLER AND J. A. FRASER

282 illustrations pp. XI + 411. Publishers Medical Book Company, 7 Aaboulevard, Copenhagen, and Bailliere, Tindall & Cassell, 7, 8 Henrietta Street, London, W.C. 2.

This book is a translation of "Tierärztliche Operationslehre" by Berge and Westhues, 28th edition 1961. The German edition has been reviewed in this Journal.

The German authors have written a book which has been widely accepted on the Continent of Europe as a standard work on veterinary surgery. They have only described procedures which have proved themselves in their hands and thus not "all" operations. To the practising veterinarian and the student this is in reality an advantage, as many of the newer operations that have been described in the literature have not stood and will not stand the test of time. The inclusion of any operation in this book is therefore a guarantee of its usefulness. It should be borne in mind, however, that the translation is of a book published in 1961, so that some good techniques which have proved themselves since that date are naturally not described.

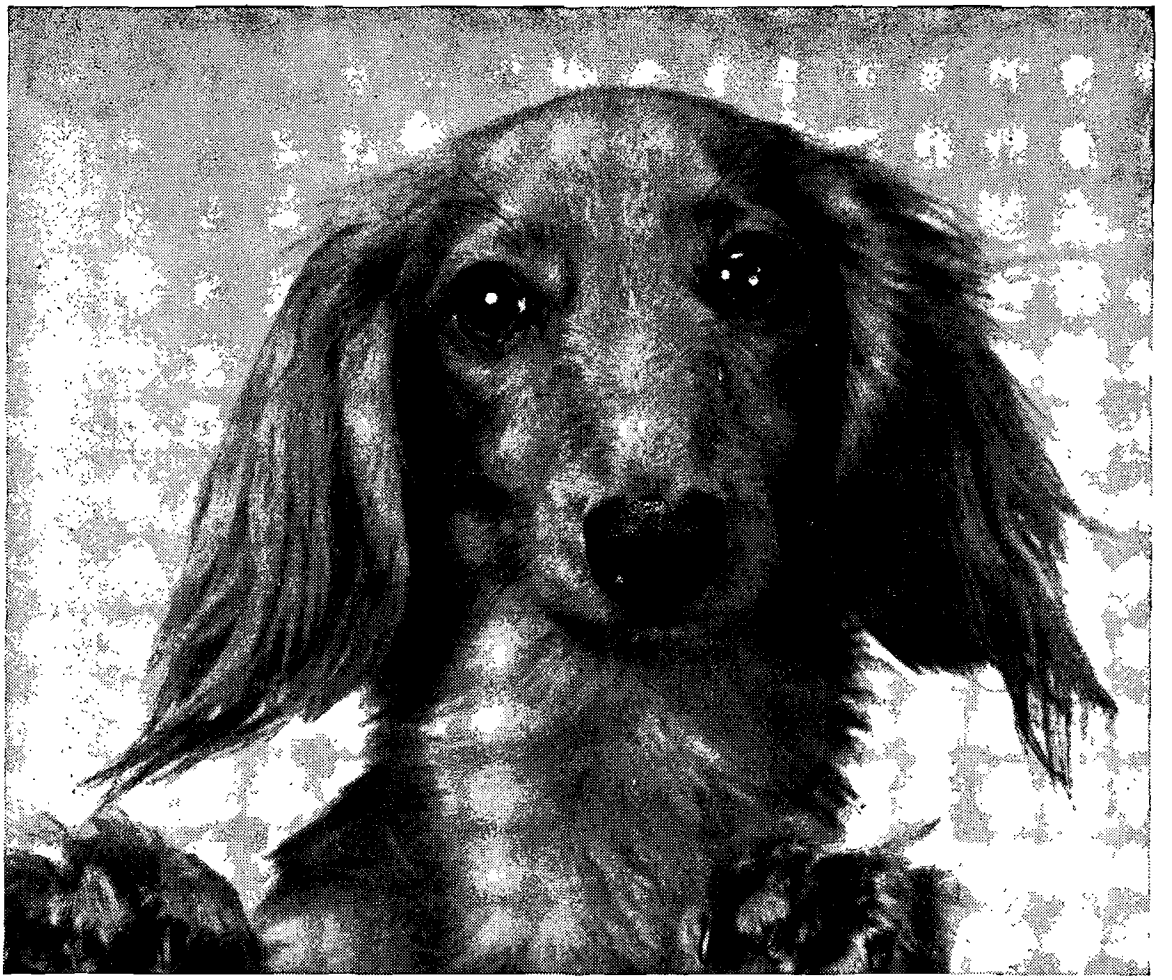
The surgery of all the domestic species is included which has much to commend it, because of the great value of the comparative approach.

General surgical principles, embracing suturing and osteosynthesis techniques, occupy 40 pages. The next 60 odd pages describe general anaesthesia and local analgesia. This is a time honoured practice for surgical textbooks, but, in the face of excellent textbooks on anaesthesia, less and less of this subject will in future be seen in works on surgery. But for the last nearly 20 pages devoted to the use of drugs and to methods of restraint, the remainder of the book is concerned with operations on regions and systems. Where necessary the surgical anatomy of the area is briefly outlined, followed by the indications and a list of instruments required. The best type of anaesthesia is then advised and succeeded by a description of the operative technique. This system is succinct and allows location of the required information without prolonged searching.

As many veterinarians are unfamiliar with German it is to be commended that this book has crossed the language barrier. It is likely to be in demand by practitioners and students alike as it is such a ready reference.

Printed on glossy paper, with well set out printing, and finely executed, artistic illustrations, and strongly bound it makes a functional and aesthetically pleasing book.

C. F. B. H.



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A PRELIMINARY STUDY ON THE SYMPTOMATOLOGY AND CARDIODYNAMICS OF GOUSIEKTE IN SHEEP AND GOATS*

P. J. PRETORIUS** AND M. TERBLANCHE***

SUMMARY

The symptoms of gousiekte during the latent period in sheep and goats were studied. The auscultation findings of tachycardia, hyperpnoea, dyspnoea systolic murmurs, gallop rhythm, splitting of the first heart sound and arrhythmias were confirmed and studied by means of phono- electro- and ballistocardiography. Cardiac insufficiency was definitely established.

INTRODUCTION

Gousiekte is a plant poisoning in ruminants caused by five different plants of the family *Rubiaceae,* namely : *Pachystigma pygmaeum* (Schltr.) Robyns[1] *Pachystigma thamnus* Robyns[2], *Pavetta harborii* S. Moore[3] *Pavetta schumaniana* F. Hoffm[4] and *Fadogia monticola* Robyns[5].

Although premonitory symptoms such as lagging behind, lying down with head and neck stretched out, dyspnoea and coughing, for up to 28 days before death have been described, symptoms of gousiekte are usually given as sudden death following a latent period of approximately 2-6 weeks[1 6 7].

Post mortem examination of gousiekte cases reveals congestion and oedema of the lungs, dilatation of the heart and varying degrees of hydrothorax, ascites, hydropericardium, hyperaemia of the rumen and small intestinal walls and occasionally liver changes[1 8 9]. A pathognomonic chronic focal myocarditis is seen histologically[1 8].

To determine any abnormalities of heart function which might develop during the latent period, clinical examination, auscultation, and phono-, electro- and ballistocardiographical studies were undertaken on developing gousiekte cases.

MATERIALS AND METHODS

Twenty-eight adult Merino sheep of both sexes and two adult Boer goats (an upgraded native goat) were dosed with or fed on *Pachystigma pygmaeum* (Schltr.) Robyns, while seven adult Merino sheep and thirteen adult Boer goats, again of both sexes, received *Pavetta harborii* S. Moore.

The dosing rate was usually at 200 g dried plant material per day or 1 kg fresh plant material daily, until death occurred. These animals died after an average interval of 56 days (range 23 - 104). One case, which had received 150 g/day, died 222 days after dosing was commenced. In eight cases, in which the dosing was discontinued after 70.4 to 143 g/kg dried plant material had been administered, a latent period of 19 days, ranging from 5 to 34 days, occurred. The MLD for both specimens of plant material used was approximately 100 g/kg, administered over *ca* 10 days.

Histological examination of the myocardium of all the experimental cases except seven, showed the typical lesions[1 8]. The latter, nevertheless, developed the typical symptoms described here and *post mortem* examination revealed the other characteristic lesions.

Daily recordings of heart rate, respiratory rate, temperature and ruminal movements were made. At the same time the heart sounds were auscultated. Phonocardiograms were made on those animals that showed auscultatory changes. At the same time the application of other cardiodynamic methods were investigated on these animals.

The electrocardiograms (ECG) and phonocardiograms (PCG) were recorded simultaneously with the animals lying in the left lateral position in order to eliminate the effect of myograms due to muscle tone in the standing position. It was found that the best position for recording the phonocardiogram was in the left lateral position. The optimum position for recording the ballistocardiogram was also found to be a lateral position in order to record the acceleration of the centre of gravity of the body in the sagittal plane. The three limb leads (Einthoven I, II and III) and in

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some cases the augmented unipolar limb leads of Goldberger were recorded (aVR, aVL and aVF. Use was made of needle electrodes inserted subcutaneously on the medial side of each limb immediately above the metacarpal and metatarsal joints. The left leg was in front of the right and the two hind legs were equally placed.

The time duration of P, PQ, QRS, QT and the heart frequency were measured and the configuration of the main deflections studied. The phonocardiograms were recorded by an Elema-Schönander crystal microphone and phono-amplifier with electronic filters for recording six different frequency ranges on an Elema-Schönander Mingograf 81 jet recorder supplied with seven recording channels at a paper speed of 100-250 mm/sec and an amplification of 20 mm/mv in all leads. The microphone was fixed by a rubber band around the chest in a position which was determined by auscultation. The frequency response of the recorder was tested just prior to use by means of an impulse generator and was found to be reliable up to 750 cycles/sec. The following time durations were measured : from Q on the ECG to the beginning of the first heart sound (QI); from Q to the second heart sound (QII); the duration of each of the two heart sounds; and the duration of the mechanical systole.

The ultra-low frequency acceleration ballistocardiographic method (UF Bcg a) as described for dogs[13], was applied to goats and sheep. The natural frequency of the platform was 0.4 c/s and the weight of the platform 2.5 kg. Acceleration was recorded from a mercury-sulphuric acid glass capillary accelerometer. The animals were anaesthetised by means of intravenous injection of pentobarbitone sodium solution and placed in the left lateral position on the Bcg-platform.

Venous and arterial blood presure and venous pulse (V.Pa) recordings were made in a few cases.

Results

A. Auscultation findings:

1. *Tachycardia*: In 50 cases of gousiekte in sheep and goats examined, 86% developed a tachycardia prior to death. The heart rates of the diseased animals exceeded the value of 120 beats/min at an average of 6 days, ranging from 1-36, exceptionally up to 54 days before death. The *mean* heart rates of all cases that died were as follow:—

   On the first experimental day the heart rate was 100/min (68-160). This is rather fast, probably due to initial anxiousness. However, on the 10th experimental day, the heart rate had settled down to 70/min (52-100). From then on a progressive increase occurred as illustrated in table 1. From the 6th day before death the *mean* heart rate exceeded the maximum individual heart rate recorded on the 10th experimental day.

Table 1.—The mean heart and respiratory rates per minute of 50 Gousiekte sheep and goats

Days	1	10	–36	–35	–34	–33	–32	–31	–30	–29
Heart rate.....	99.0	76.9	71.8	75.2	73.8	74.3	78.5	75.4	77.0	78.4
Respiratory rate.....	50.6	31.0	30.0	32.1	31.3	31.1	36.6	32.5	33.2	33.1
Days	–28	–27	–26	–25	–24	–23	–22	–21	–20	–19
Heart rate.....	80.7	81.8	75.9	76.7	77.0	77.0	75.5	80.6	81.2	81.4
Respiratory rate.....	35.4	33.0	32.6	36.5	32.7	33.3	31.2	33.1	32.7	32.1
Days	–18	–17	–16	–15	–14	–13	–12	–11	–10	–9
Heart rate.....	82.6	82.8	83.8	85.3	84.9	86.2	89.2	88.5	89.6	95.6
Respiratory rate.....	34.3	33.2	33.8	34.0	37.3	35.1	36.2	36.8	40.0	40.1
Days	–8	–7	–6	–5	–4	–3	–2	–1	0	
Heart rate.....	88.9	94.2	100.9	103.7	102.9	108.5	114.7	120.3	131.1	
Respiratory rate.....	39.8	41.6	43.3	44.1	43.5	49.7	47.2	51.1	59.9	

2. *Hyperpnoea*: The mean respiratory rate of these animals was 50 per minute (range 24-60) on the first experimental day and settled to 30 per minute (range 20-60) on the 10th day. In 72% of cases the respiratory rate exceeded the value of 50/min at an average of 10 days (1-21) prior to death, while a progressive increase in the *mean* respiratory rates occurred as shown in table 1.

3. *Dyspnoea*: In 24% of cases a dyspnoea due to oedema of the lungs developed at an average of 1.5 days (1-4) prior to death. This was characterised by a rapid breathing and forced expiration accompanied by a groan.

4. *Systolic murmur*: In 66% of cases a systolic murmur developed at an average of 11 (1-43, exceptionally 59) days before death. On auscultation the murmur could be heard as a relatively low pitched sound. This usually started off as an early systolic murmur with or without the first heart sound audible (fig. 1 b) later becoming a pansystolic murmur (fig. 1 c) with or without the first heart sound audible.

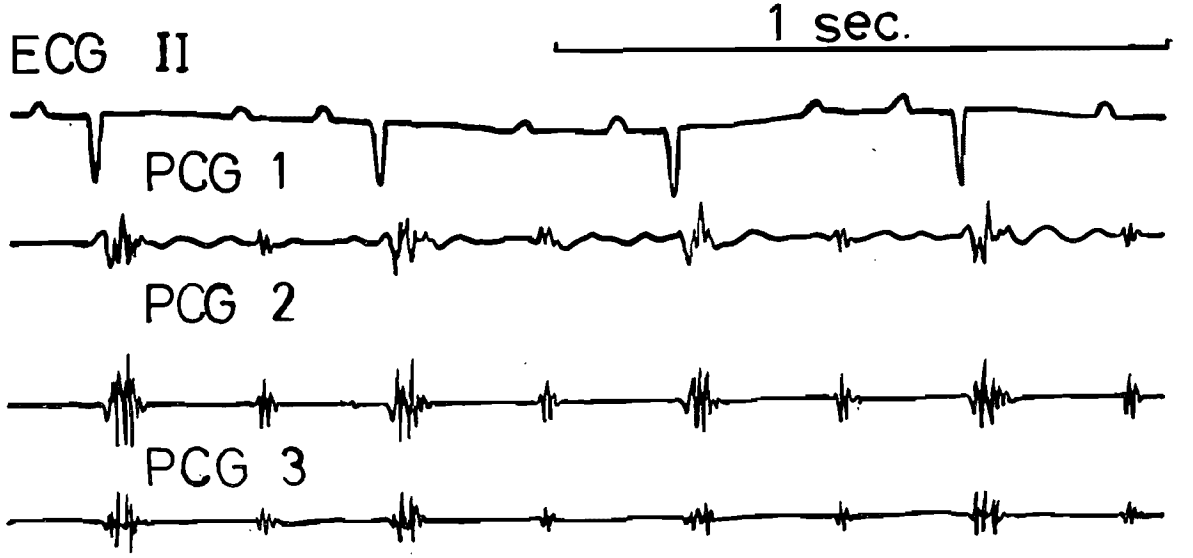


Fig. 1a — The Phonocardiogram (PCG) of a normal sheep.

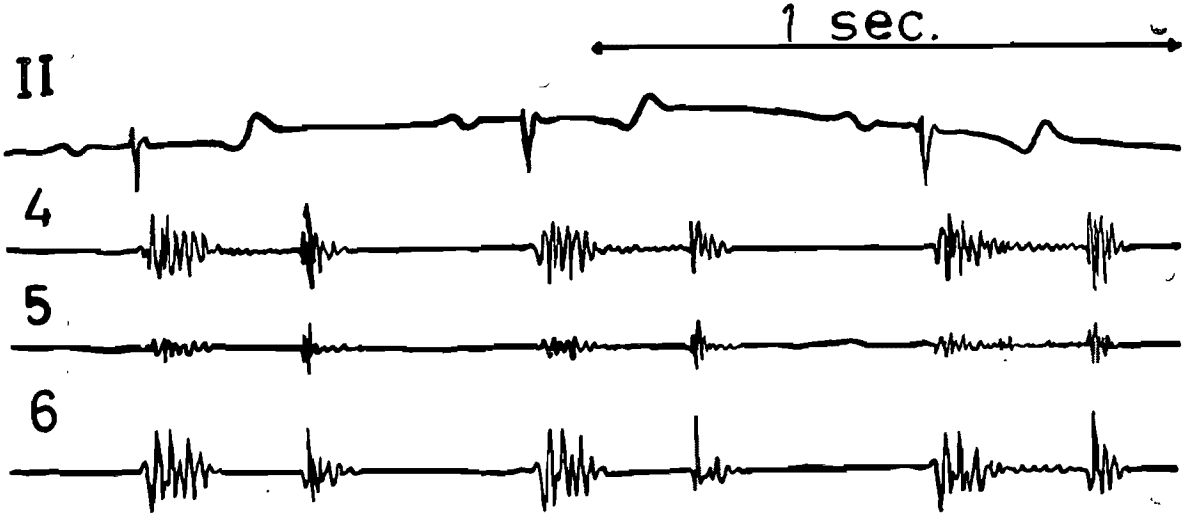


Fig. 1b — The PCG of a gousiekte sheep showing an early systolic murmur.

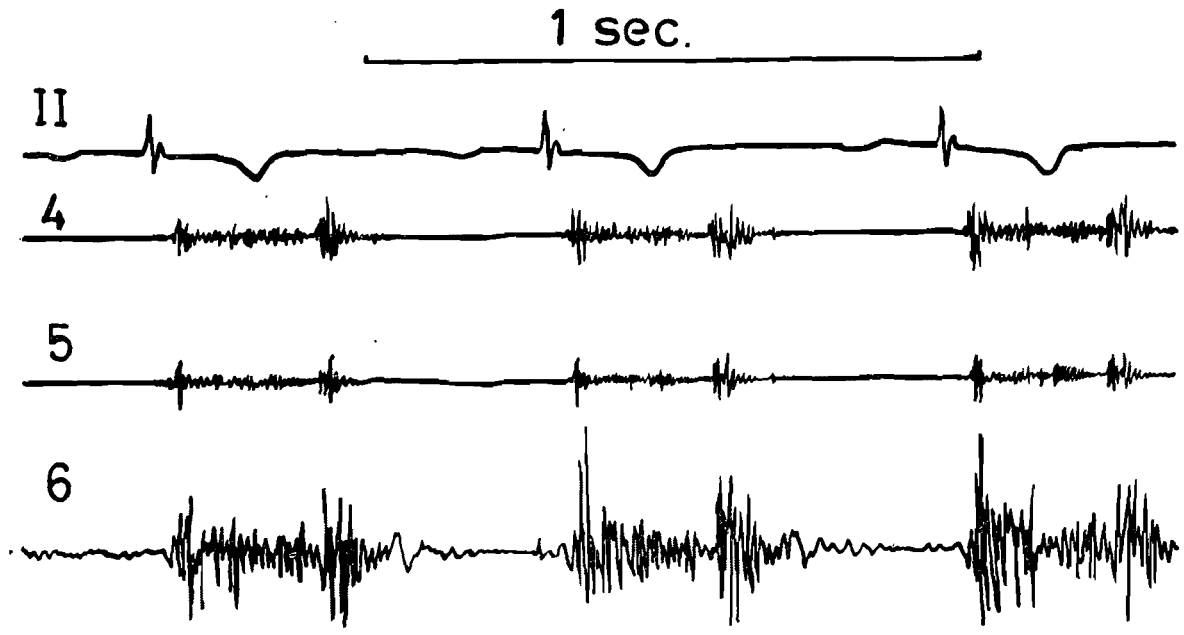


Fig. 1c — The PCG of a gousiekte sheep showing a pansystolic murmur.

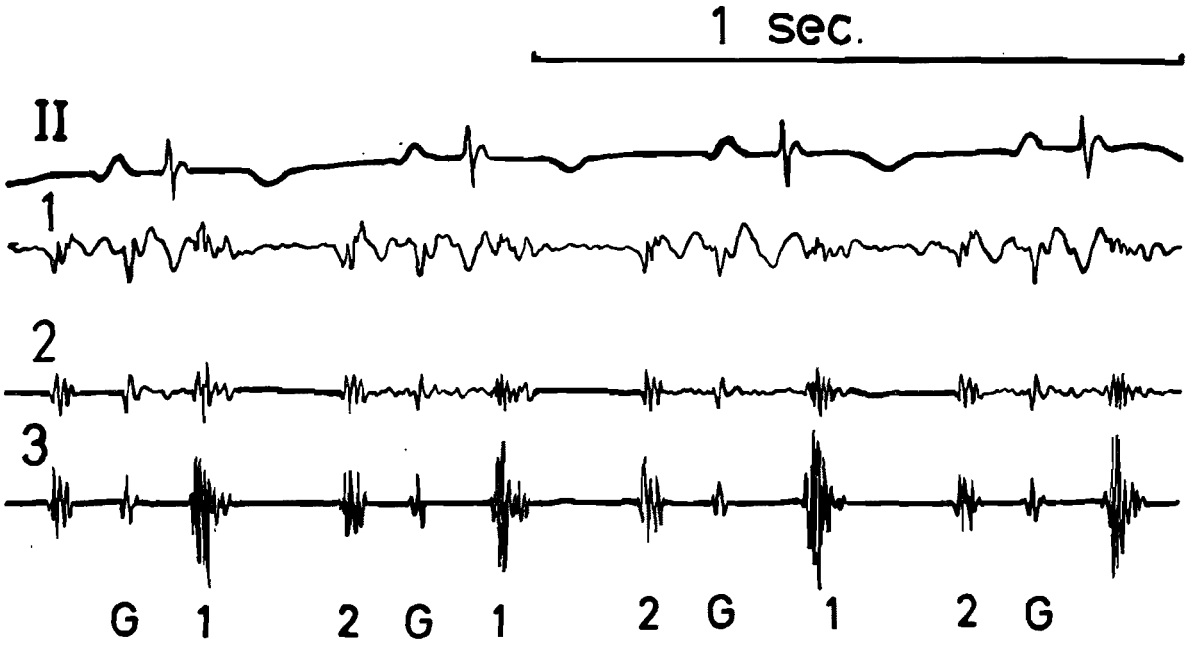


Fig. 2 — The PCG of a gousiekte goat showing a gallop rhythm. i.e. a third heart sound.

One case had a late systolic murmur. Phonocardiographically these murmurs were best seen in the relatively high frequency ranges of *ca* 800 cycles/sec.

5. *Gallop or Triple Rhythm*: In 48% of cases a gallop rhythm developed at an average of 7 (1-21) days before death. On auscultation three heart sounds could be distinguished. Usually the gallop rhythm was associated with a tachycardia. In these cases an extra heart sound could not be distinguished but the characteristic galloping rhythm was unmistakable.

   In the phonocardiograms of all these cases an extra heart sound particularly in the low frequency ranges 3, 2 and 1 were recorded. (See fig. 2).

In order to determine the genesis of this galloping sound the following time intervals were measured : Cycle length (RR), from Q on the ECG to the beginning of the gallop sound (QG), from Q to the beginning of the first heart sound (QI) as well as the second heart sound (QII) and from P on the following cardiac cycle on the ECG to the gallop sound (P_2G) (see figure 3). From this it became apparent that the gallop sound was related to the diastolic phase of the previous cycle because it occurred at a constant time interval after the previous electrical systole (QG).

During a gradual increase in the cycle length (RR interval) as seen in fig. 3, the QG time remained constant, showing the same time relationship to Q than did the first (QI) and second (QII) heart sounds. During this increase of cycle lengths (RR) the (P_2G) time did not remain constant but was inversely proportional to the cycle lengths (fig. 3). This gallop sound, therefore, appeared to be a third heart sound, which is a sound associated with abnormal ventricular relaxation.

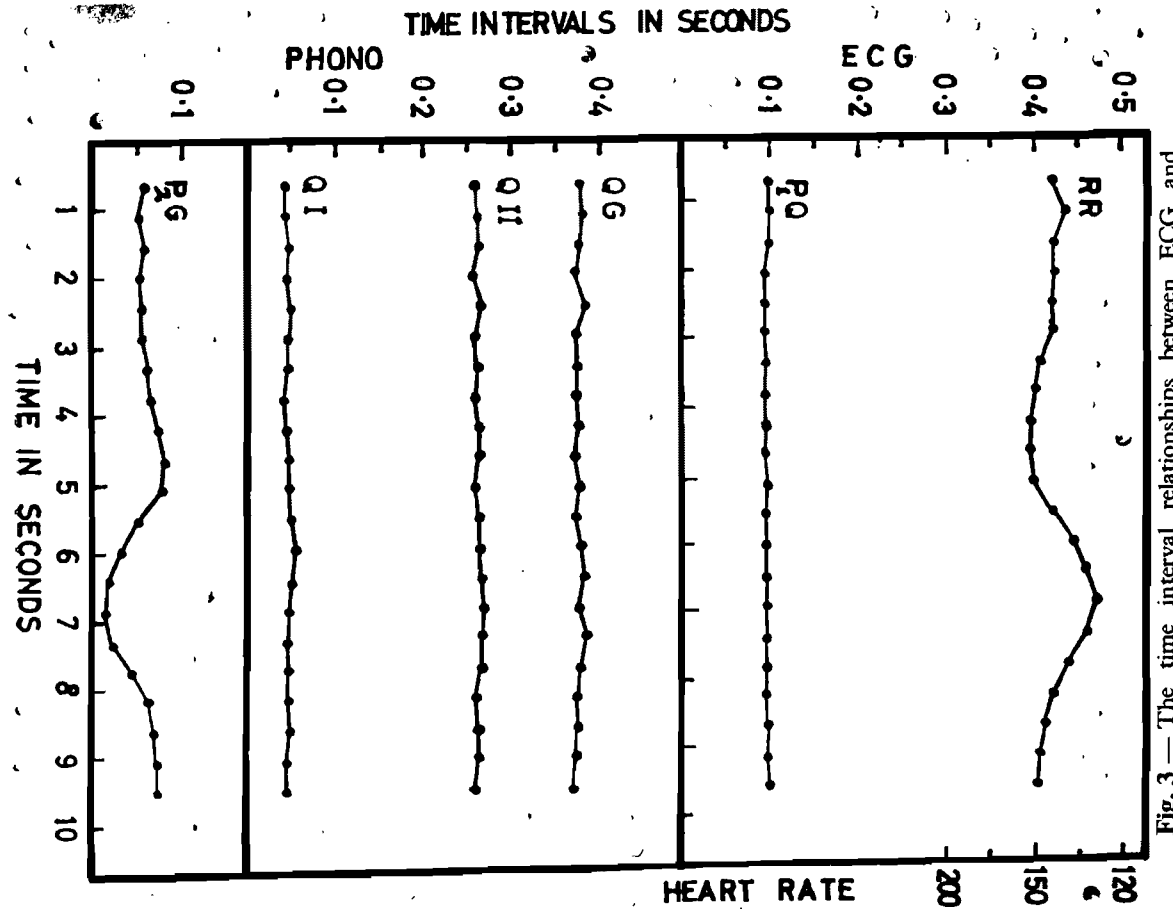


Fig. 3 — The time interval relationships between ECG and PCG during a gallop rhythm in a gousiekte sheep.

The position of the gallop sound on the PCG appears very close to the position of the fourth heart sound, which is a sound associated with abnormal atrial contraction, and can easily be confused with the latter.

During sinus arrhythmia the cycle length was suddenly increased e.g. from 0.44 to 0.56 sec in figure 4. During this increased cycle length the gallop sound clearly precedes the following P wave and is, therefore, unmistakably a third heart sound.

Various combinations of these symptoms were recorded : e.g. a pansystolic murmur plus a gallop rhythm (see fig. 5) or an early systolic murmur plus a gallop rhythm (see fig. 6).

6. *Splitting of the first heart sound*:

It was found that in phonocardiograms from sheep and goats, the first heart sound normally consisted of two vibrational complexes with main peaks 0.024 seconds (0.016-0.032) apart in goats and 0.024 seconds (0.012-0.033) apart in sheep.

In 67% of cases of gousiekte a splitting of the first heart sound could be auscultated at an average of 10 (1-31) occasionally up to 43 days before death. In goats the peaks were 0.032 (0.028-0.040) and in sheep 0.032 (0.020-0.047) seconds apart. In fig. 7 such a splitting of the first heart sound is illustrated. In this case the main peaks were 0.040 seconds apart.

From about 27 days (14-40) before death a first heart sound of longer duration and lower intensity could be recognised by an experienced observer in 45% of cases on auscultation. The duration of the first heart sound on the PCG was measured in 71 normal sheep and 78 normal goats and found to be 0.052 (0.032-0.068) and 0.060 (0.038-0.079) seconds respectively. In twelve gousiekte sheep it was found to last 0.073 (0.052-0.096) and in eleven gousiekte goats 0.068 (0.047-0.088) seconds, which confirms the auscultation finding. No definite opinion can be expressed concerning the intensity changes on the PCG at this stage.

7. *Arrhythmia*: In 56% of cases an arrhythmia could be detected on auscultation. It appeared at an average of 11 (1-28) days occasionally up to 56 days before death and the following types could be distinguished:—

(i) *Runs of tachycardia*: This was characterised by sudden increases in heart rate up to twice the normal rate for a few seconds, followed by a decrease back to normal.

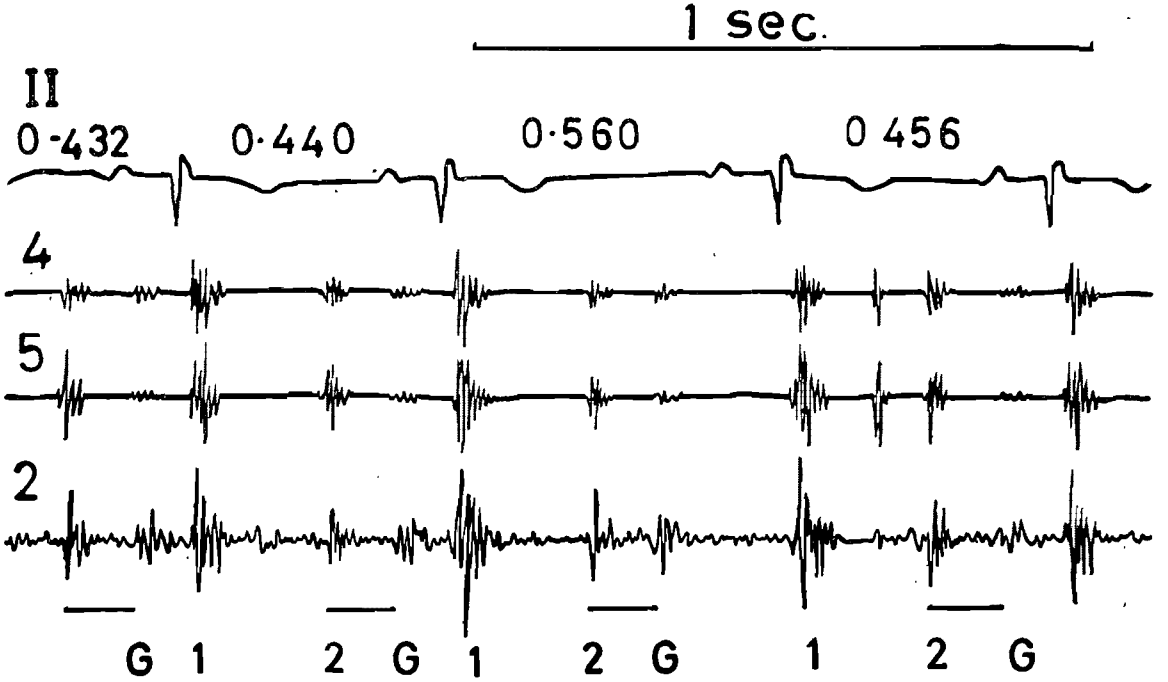


Fig. 4 — A sinus arrythmia during a gallop rhythm in a gousiekte sheep.

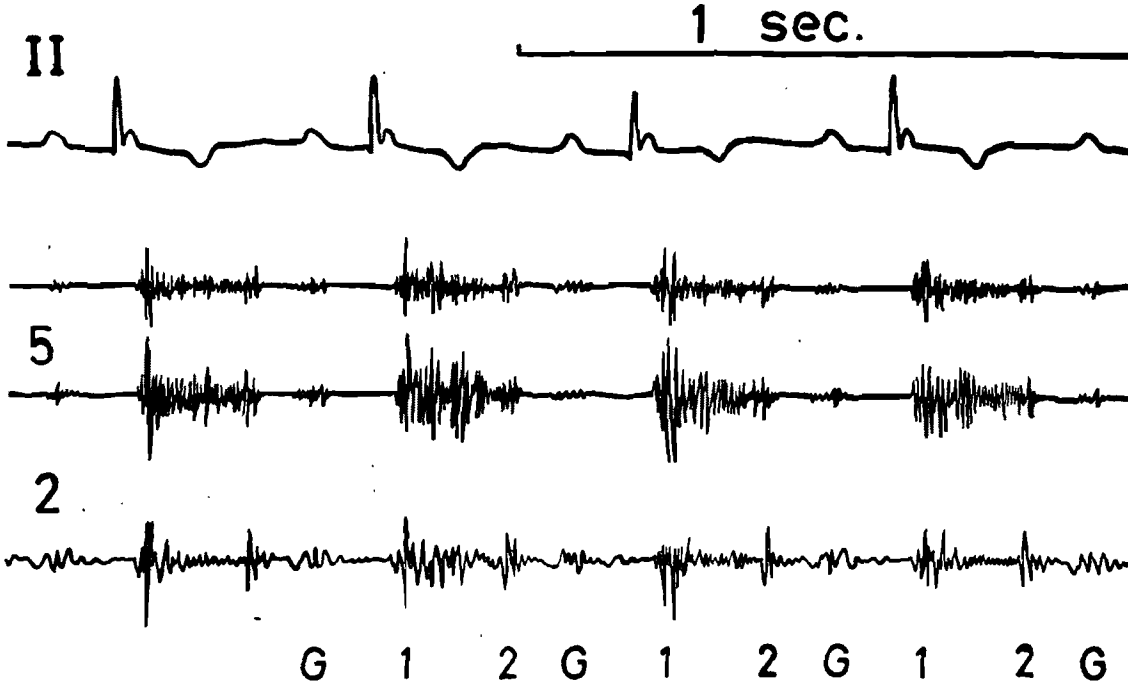


Fig. 5 — The PCG of a gousiekte sheep with a combination of pansystolic murmur and a gallop rhythm.

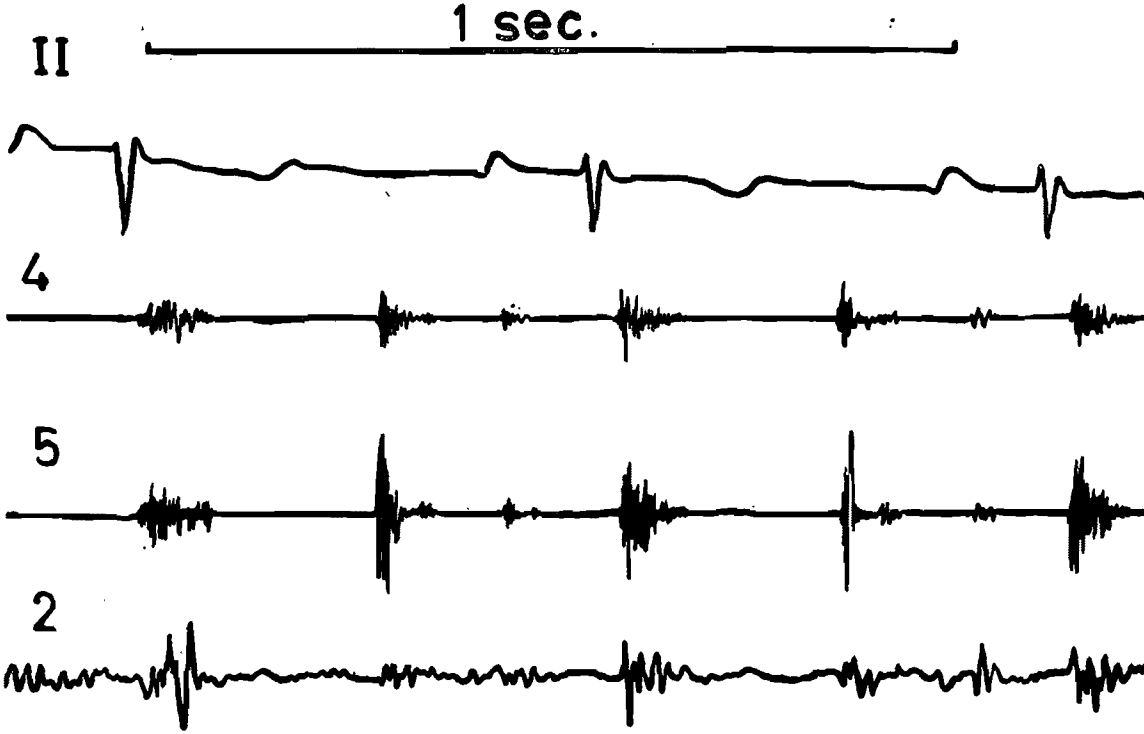


Fig. 6 — The PCG of a gousiekte sheep showing a combination of early systolic murmur and gallop rhythm.

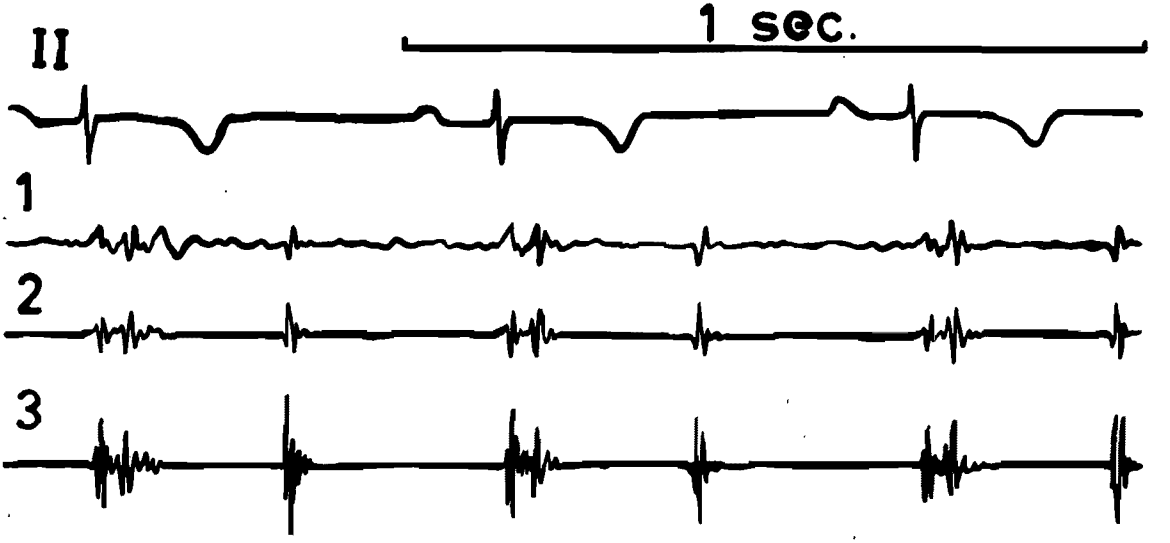


Fig. 7 — The PCG of a gousiekte goat showing a splitting of the first heart sound.

(ii) *The dropped beat rhythm*: This was characterised by combinations of 2,3,4 etc. normal beats which were followed by a short pause as if a beat were dropped.

(iii) *Irregular rhythm*: Normal heartbeats were suddenly interrupted by an irregular rhythm during which it was impossible to identify the different sounds.

The genesis of the arrhythmias was investigated by means of electrocardiography.

B. *The Electrocardiographic changes*:

I. *The Time intervals of the ECG.*

Different time intervals of the ECG Lead II measured in 71 normal sheep, 78 normal goats, and compared to 12 gousiekte sheep and 11 gousiekte goats, are summarised in table 2.

The duration of the P wave showed a small increase which may indicate atrial dilatation. The PQ interval showed inconspicuous changes which indicated normal AV-conduction. The QRS time intervals measured in the normal sheep compared very well with that given by Hamlin[10] of 0.035 sec (0.028-0.045) for lead aVF. In comparing the normals with that found in gousiekte a slight increase of 0.006 in the sick sheep and 0.008 in the sick goats was noted. This may indicate partial bundle branch block.

When the QT time interval was corrected by means of Bazett's formula an increase of 0.06 sec was found in gousiekte sheep and a decrease of 0.04 sec was found in gousiekte goats.

An increase in heart rate was found on measuring the cycle lengths on the ECG. This was due

TABLE 2.—THE DIFFERENT ECG TIME INTERVALS IN NORMAL AND GOUSIEKTE SHEEP AND GOATS

Sheep	P			PQ			QRS			QT			Heart Frequency		
	Mean	Min.	Max.	Mean	Min.	Max.	Mean	Min.	Max.	Mean	Min.	Max.	Mean	Min.	Max.
Normal.....	.034	.03	.05	.109	.08	.12	.034	.02	.06	.302	.26	.34	98	79	115
Gousiekte.....	.041	.04	.05	.093	.08	.10	.04	.03	.04	.226	.19	.25	131	107	167
Goats															
Normal.....	.037	.02	.05	.098	.08	.12	.032	.02	.04	.263	.23	.29	100	60	136
Gousiekte.....	.046	.043	.056	.105	.100	.108	.041	.040	.042	.276	.20	.31	114	94	139

to a sinus tachycardia. On measuring the RR (or PP) time intervals the following changes were observed:

(i) The runs of tachycardia found on auscultation were due to a gradual decrease in cycle length. This was not a respiratory sinus arrhythmia.

(ii) Sudden increases in the PP time intervals occurred. In fig. 4, for example, succeeding cycle lengths of 0.424, 0.432 and 0.440 seconds are suddenly interrupted by an increased cycle length of 0.560 seconds. This was again followed by a "normal" cycle of 0.456 seconds. To illustrate this change, the time intervals were plotted graphically for each experimental animal as shown in fig. 8.

This might have corresponded with the "dropped beat rhythm" heard on auscultation. However, in normal animals only 30% showed a regular PP time interval for the period during which recordings were made, 20% showed only slight irregularities, i.e. one "increased" PP interval for each 10-70 heart beats, 27% showed moderate irregularities, i.e. 1 for 9-4 beats, and 23% showed one increased PP interval during less than four normal beats, this was termed severe PP irregularity.

In the sick animals the irregularities increased. In the twelve sick sheep only 8.3% showed a constant PP time, 33.2% showed moderate irregularities while 25% showed severe irregularities.

The duration of the increased PP intervals were calculated as percentage of the mean PP interval for each individual. In normal animals the maximum PP increase had a mean value of 22.0% with a range of 0.6 to 40%. In sick animals the PP increase had a mean value of 21.9% with a range of 1.9 to 40%. Only one showed a 100% increase during heart block.

(iii) It was also observed that in some sheep every second beat was followed by an increase in cycle length. This was called PP time alternation (see fig. 9). PP time alternation was ob-

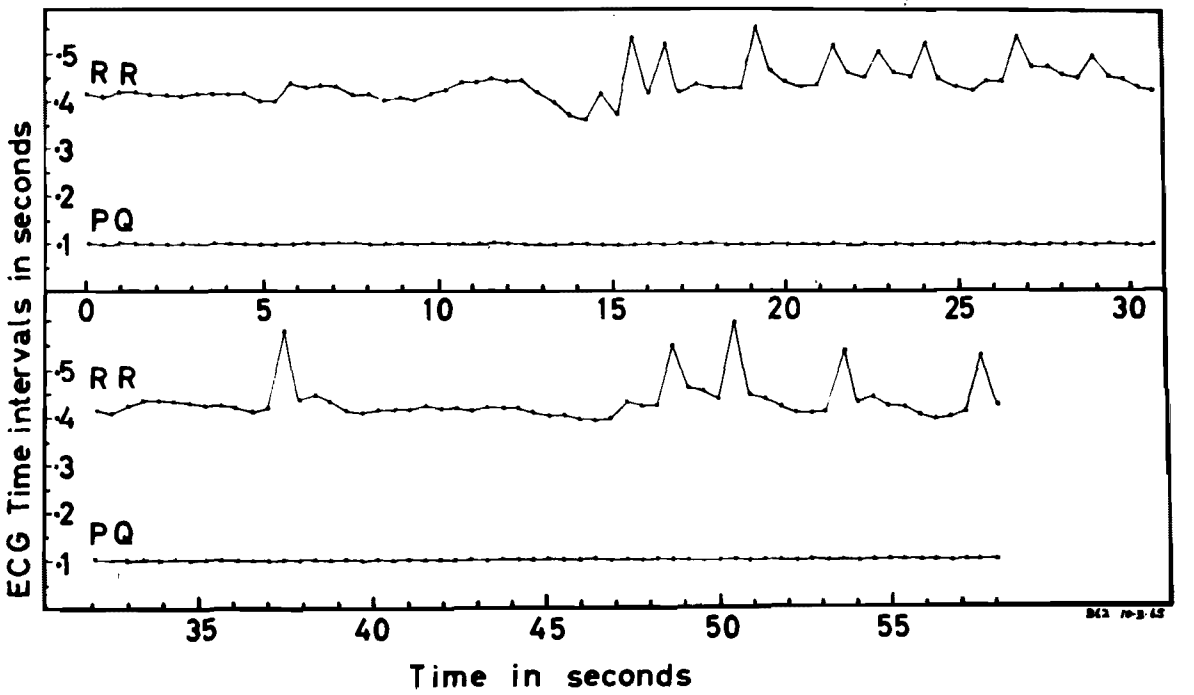


Fig. 8 — The cycle length (RR or PP-time) changes in a gousiekte sheep.

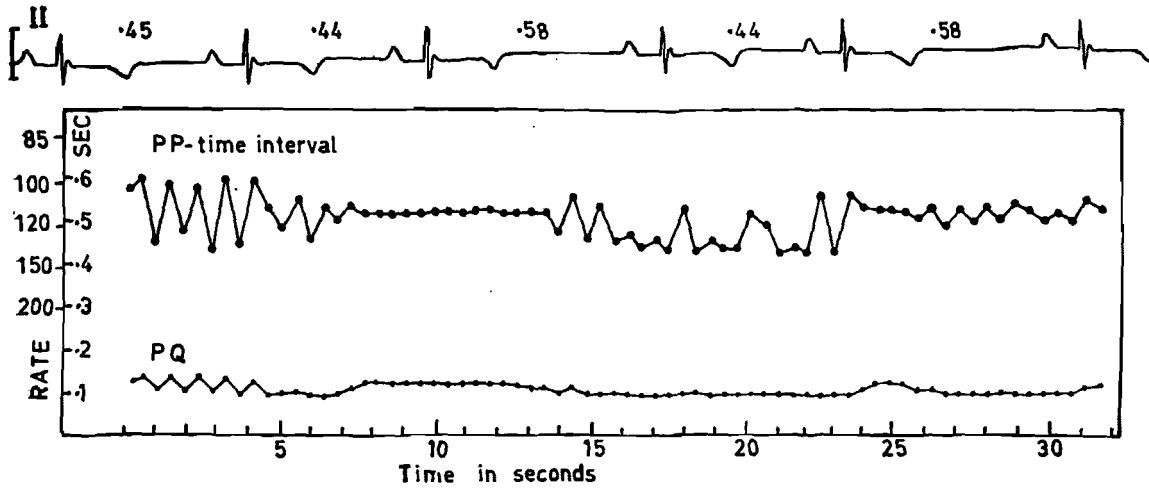


Fig. 9 — PP-time interval alternation in a gousiekte sheep.

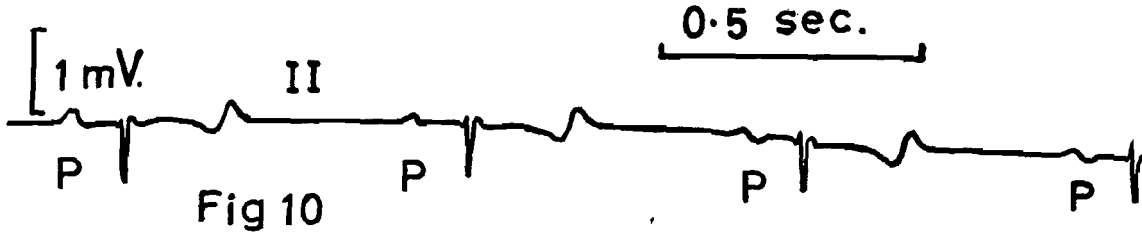


Fig 10

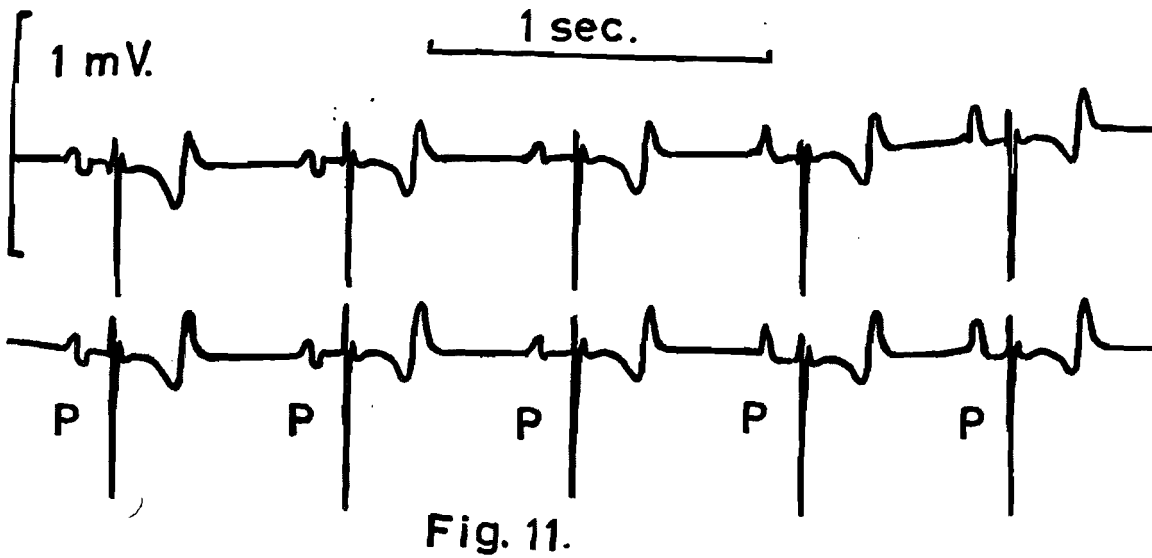


Fig. 11.

Fig. 10 — A wandering pacemaker. A gradual change in the P-wave from monophasic positive to biphasic (+/−).

Fig. 11 — A wandering pacemaker. A sudden change in the P-wave from biphasic (+/−) to monophasic positive.

served in 23.8% of normal sheep while 50% of the sick sheep showed this phenomenon. PP interval alternation from beat to beat occurred in a sporadic pattern in normal and abnormal animals, i.e. it was recorded for only about 10-20 heart beats at a time before it disappeared. After about 40-50 beats it reappeared again.

II. *The Configuration of the ECG waves.*

(i) *P-wave* changes or Wandering Pacemaker.

Changes in the configuration of the P-wave were observed during gousiekte (see table 3). In the aVR lead, abnormal biphasic (+/−) P-waves occurred in 58% of gousiekte sheep, while none were observed in 71 normal records of healthy sheep.

The aVR lead also showed the least variation in the normal sheep. Sixty four cases showed a negative (−) P-wave and only one a positive (+) P-wave while the other six showed non-measurable (0) P-wave. There is, therefore, a tendency for a biphasic P-wave to develop in the aVR lead during gousiekte. A conspicuous increase in biphasic (−/+) type P-wave was also observed in lead II. In normal sheep (−/+) type P-wave was recorded in only three out of 71 normal animals

TABLE 3.—THE CONFIGURATION OF THE P-WAVE *In normal sheep*

Lead I	Lead II	Lead III	aVR	Number of Cases
+	+	+	−	24
−	+	+	−	11
0	+	+	−	15
−	+	+	0	4
+/+	+/+	+/+	−	4
+	+	+	0	2
+	+/+	+/+	−	2
0	+/+	+	−	1
−/+	+/+	+/+	−/−	1
+	−/+	−/+	−	1
−	+	−/+	−	1
−/−	−/+	+	−	1
+/−	−/+	−/+	−/−	1
+	−	+	−	1
+/−	+	+	−	1
+	+/+	+/+	+	1
+ : = 31 − : = 96 +/+: = 4 0 : = 16 −/−: = 1 +/−: = 2 −/+: = 1	+ : = 58 +/+: = 9 −/+: = 3 − : = 1	+ : = 60 +/+: = 8 −/−: = 3	− : = 62 0 : = 6 −/−: = 2 + : = 1	71

TABLE 3.—THE CONFIGURATION OF THE P-WAVE *In Gousiekte Sheep*

Lead I	Lead II	Lead III	aVR	No. of Cases
−	+	+	+/−	3
+	+	+	−	1
−	+	+	−	1
+	+	+	+/−	1
−/+	−/+	+	−	1
−/+	−/+	+	+/−	2
−	−/+	+	+/−	1
+	+/+	+/+	−	1
−	−/++	−/++	++/−	1
3 : +	6 : +	10 : +	7 : +/−	12
6 : −	1 : ++	1 : ++	4 : −	
3 : −/+	4 : −/+	1 : −/++		
	1 : −/++			

TABLE 4.—THE CONFIGURATION OF THE DIFFERENT ECG WAVES IN GOATS

Normal Goats

LEAD II				LEAD III			
P	QRS	T	No. of cases	P	QRS	T	No. of cases
+	−	+	17	+	−	+	17
+	+/−	+	15	+	+/−	+	16
+	−/+/−	+	11	+	−/+/−	+	13
+	−/+	+	8	+	−/−	+	11
+	−/−	+	8	+	−/+	+	6
+	+	+	7	+	+	+	4
+	+/−	−/+	3	+	+/+/−	+	2
+	−	−	3	+	+/+	+	2
+	+/+/−	+	2	+	+/−	−/+	2
+	+	−	1	+	−	−	2
+	−/+/−/+	+	1	+	+	−	1
+	+/+	+	1	+	−/−	−	1
+	−/−	−	1	+	−/+/−/+	+	1
+78	− 20	+70	78	+78	− 19	+72	78
	−/− 9	− 5			−/− 12	− 4	
	−/+ 8	−/+ 3			−/+ 6	−/+ 2	
	(+)/− 18				+/− 18		
	† 55				55		
	+ 8				+ 5		
	+/+ 1				+/+ 2		
	−/+/− 11				−/+/− 13		
	+/+/− 2				+/+/− 2		
	−/+/−/+ 1				−/+/−/+ 1		
	†† 23				23		

† Mainly a negative deflection.
†† Mainly a positive deflection.

Gousiekte Goats

LEAD II

P	QRS	T	Number of cases
−/+	−/+/(+)	+	3
+	+/−/+	+	2
+	+/−	−/+	1
+	−/+/−	−/+	1
−/+	−/+/+	−/+	1
−/+	−/+/(+)	−/+	1
+	−/+	−	1
+	−/+	(−) +	1
+ 6	+/− 1	−/+ 5	11
−/+ 5	−/+/− 1	+ 5	
	+/−/+ 2	− 1	
	−/+/+ 5		
	−/+ 2		

while in the sick sheep seven out of 12 showed this type of P-wave. The same observations were made in lead II in goats (see table 4).

A change in P-wave configuration was seen during the course of a series of succeeding heart beats. This consisted of a gradual change from a (+) to a (+/−) in lead II and eventually back to a (+) P-wave (see fig. 10), or a sudden change from biphasic to positive (see fig. 11) in lead II and III and negative in lead aVR.

P-wave changes of varying degree were observed in 10 out of 12 sick sheep.

The vectorial analyses of P-wave changes in 5 animals showed that a shift of between 6° to 45° to the right occurred in two and 3° to 28° shift to the left in three animals.

The abnormal beats often persisted for a maximum of 16 successive beats before the normal P-wave configuration returned. These P-wave changes are typical of a "wandering pacemaker".

Administration of oxygen at two different occasions abolished the occurrence of the abnormal P-wave. For example, in one case (a goat) the ratio of abnormal to normal P-wave was 1:0.9. After four minutes of oxygen administration, this improved to 1:1.9 and after 10 minutes to 1:11.3.

Slight P-wave changes were also observed in one of the 71 normal sheep.

(ii) *QRS changes*:

The normal configurations of the QRS complex of 78 goats and 71 sheep are summarised in tables 4 and 5. The most conspicuous characteristics of the QRS in sheep is a definite tendency to be negative in leads II and III and positive in lead aVR. Lead I shows a variable pattern of low amplitude. A change in QRS deflection was observed during gousiekte (see table 5). The main deflection of leads II and III tend to change to positive and aVR to negative. If the QRS vector of a typical normal sheep is compared

TABLE 5.—THE CONFIGURATION OF THE ECG WAVE, QRS, IN SHEEP

Normal Sheep

Lead I	Lead II	Lead III	VR	No. of Cases
+	−	−	+	30
−	−	−	+	19
−/+	−	−	+	11
+	+/−	+/−	+	5
+	+	+	−/+	2
+/+	+/+	+/+	−/+	2
+	+	+/−	+	1
−	+/−	+/−	+	1
− : 20 + : 40 −/+ : 11	− : 60 + : 5 +/− : 6	− : 60 + : 4 +/− : 7	+ : 67 −/+ : 4	71

Gousiekte Sheep

Lead I	Lead II	Lead III	aVR	No. of Cases
+	+/−	+/−	−/+	3
−	+/−	+	+	1
+	+	+/−	−	1
+	+/+	−/+	−/−	1
+	+	−/+	+/−	1
+/−	+	+/+	−	1
+	+	+	−	1
+	−/+	−	+/−	1
+/+	−	−	−/+	1
−/+	−	−	+	1
+ : 9 − : 1 +/− : 1 −/+ : 1	+ : 5 +/− : 4 − : 2 −/+ : 1	+ : 3 +/− : 4 −/+ : 2 − : 3	+ : 2 +/− : 2 − : 4 −/+ : 4	12

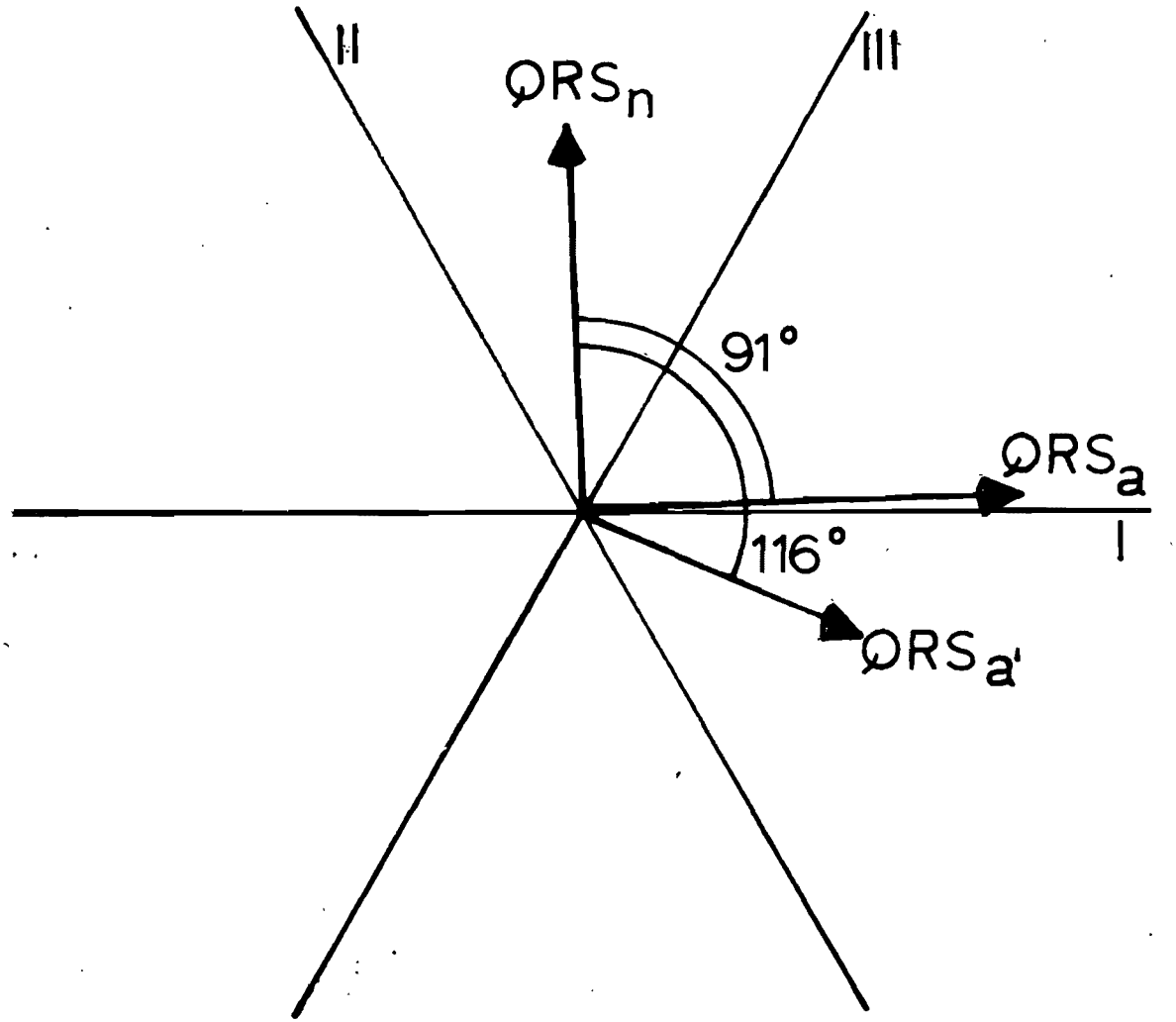


Fig. 12 — Typical QRS – axis deviation during gousiekte. Two cases in sheep are illustrated. QRSn = normal, QRSa = abnormal.

to that of two affected animals, shifts of 91° and 116° respectively, are seen (see fig. 12). These changes in the configuration of the QRS complex may be an indication of a change in the direction of ventricular depolarisation. This may be caused by partial bundle branch block. The QRS complex in normal goats showed much more variation (see table 4). In all the sick ones, however, the QRS waves were diphasic which may also indicate partial bundle branch block.

Much variation was seen in the normal T-wave and thus no conclusions for possible T-wave changes in sick sheep could be made. In goats the T-wave showed a tendency to become diphasic during gousiekte (see table 4).

(iii) *Auriculo-ventricular* block occurred in only one sheep during gousiekte (see fig. 13). Second degree incomplete AV-block with a 1:1, 2:1 and 3:1 rhythm persisted for about one minute. After this period an AV-nodal rhythm developed (see fig. 13).

(iv) *Extra systolic beats*: Premature ventricular beats were recorded in one sheep and four goats during gousiekte. A typical example is represented in fig. 14 where six different

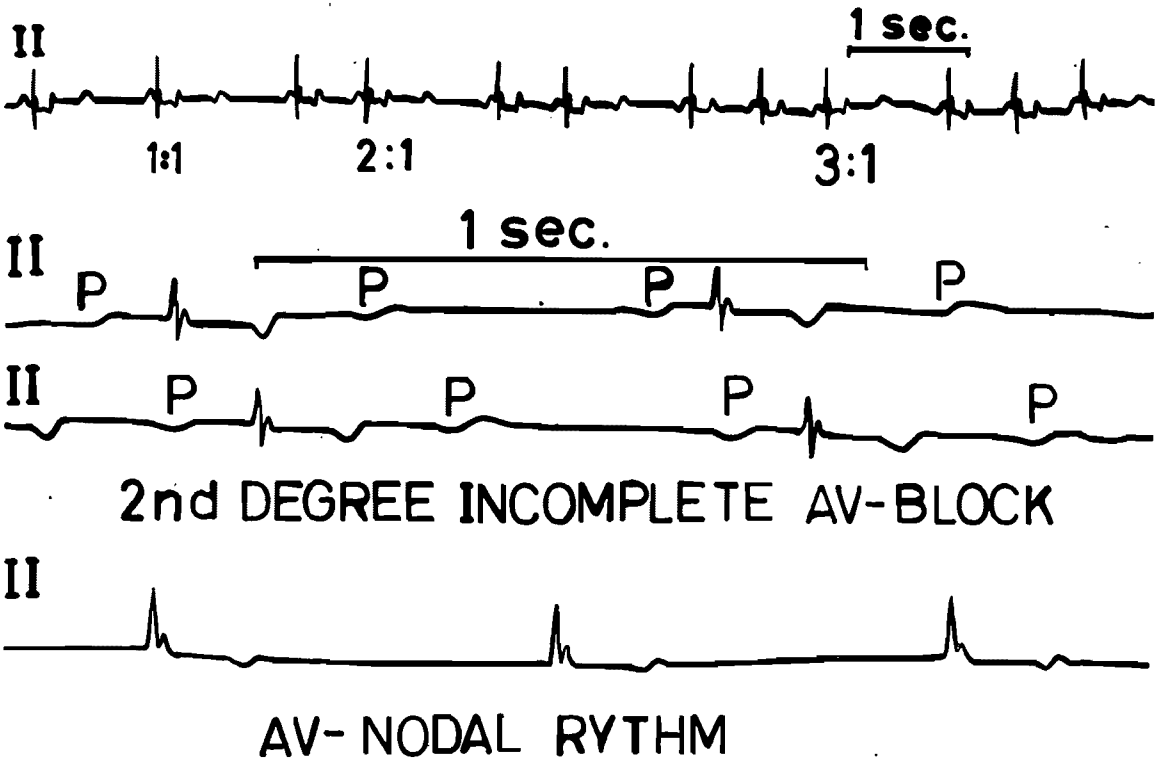


Fig. 13 — Second degree incomplete AV– block (Wenkebach-type) with dropped ventricular beats in a gousiekte sheep.

ectopic foci occurred. The QRS time interval for these premature beats ranged from 0.034 sec. to 0.064 sec. The mean increase was 46%. (Hamlin[10] found a mean increase for experimentally evoked premature beats of 145% for lead aVF). An increase in ectopic foci occurred in two goats after exercise during gousiekte.

(v) *QRS alternation*: Five sheep and one goat showed alternation of the QRS amplitude. A typical example is shown in fig. 15.

Ballistocardiographic changes (Ultra low frequency): It was found that the longitudinal body axis was not the optimum axis for recording the acceleration of the centre of gravity of the body. When the body acceleration was recorded in a direction at an angle of approximately 45° to the longitudinal axis of the body, satisfactory results were obtained (see fig. 16).

The 45° angle pointed ventro-caudally and the axis of recording was more or less parallel to the direction of blood flow during the ejection phase in the ascending aorta proximally to the bifurcation.

In order to record the ballistocardiogram, the animals were anaesthetised with "Sagatal" 64 mgm/cc sodium pentobarbitone). The effect of administration of an additional small dose of 1.5 cc Sagatal was investigated in three goats. It was found that the Bcg was conspicuously decreased for a period lasting a few minutes after administration (see fig. 17). In interpreting the results therefore, the effect of anaesthesia should be kept in mind.

In three goats recordings were made prior to dosing with *Pavetta harborii* and thereafter at regular intervals. The results obtained are illustrated by showing the changes in one goat in fig. 18. The first recording was made 13 days before dosing with *Pavetta harborii*. The second recording was made 4 days after dosing was commenced, which was 90 days prior to death. A gradual decrease especially from the 80th day before death, in the slope, amplitude and general configuration of the HI and IJ segments of the Bcg developed. At 70 days, changes in the Bcg pattern in succeeding heart beats were seen. At 66 days prior to death there were signs of improvement of the Bcg,

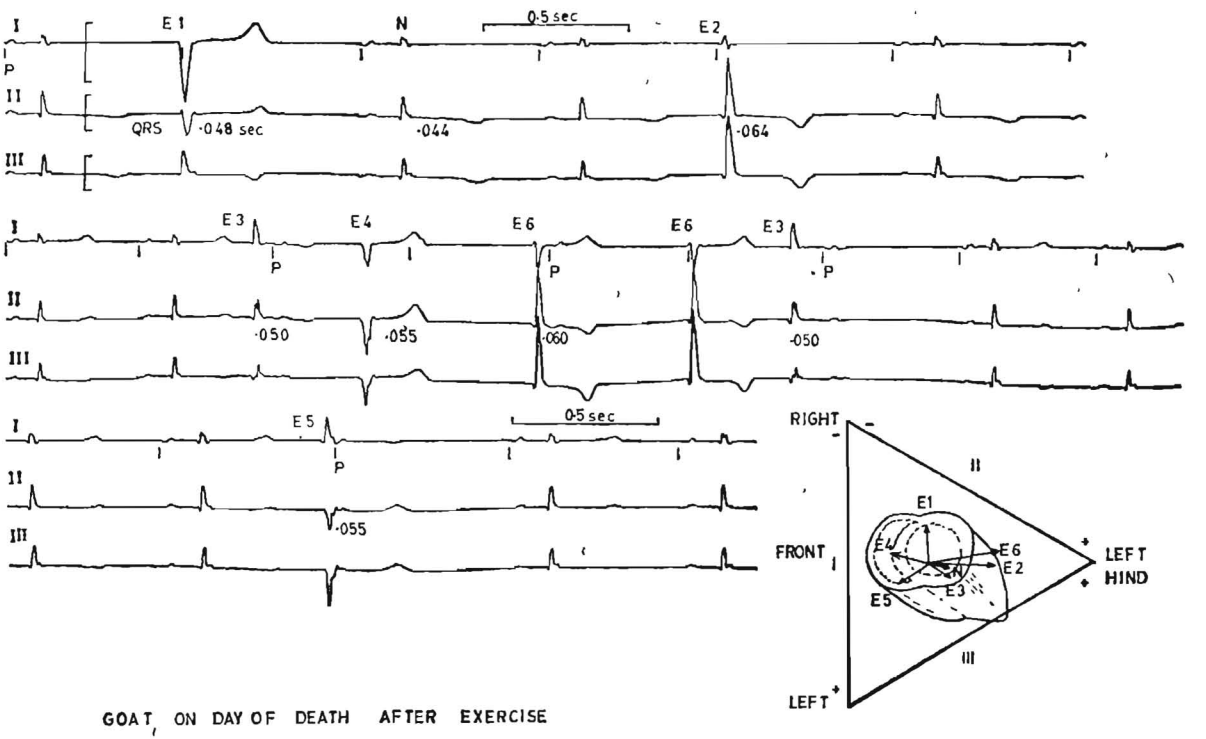


Fig. 14 — Multi-focal ventricular premature beats which developed after exercise. The QRS time intervals of 6 ectopic beats as well as their configuration and vectors are shown.

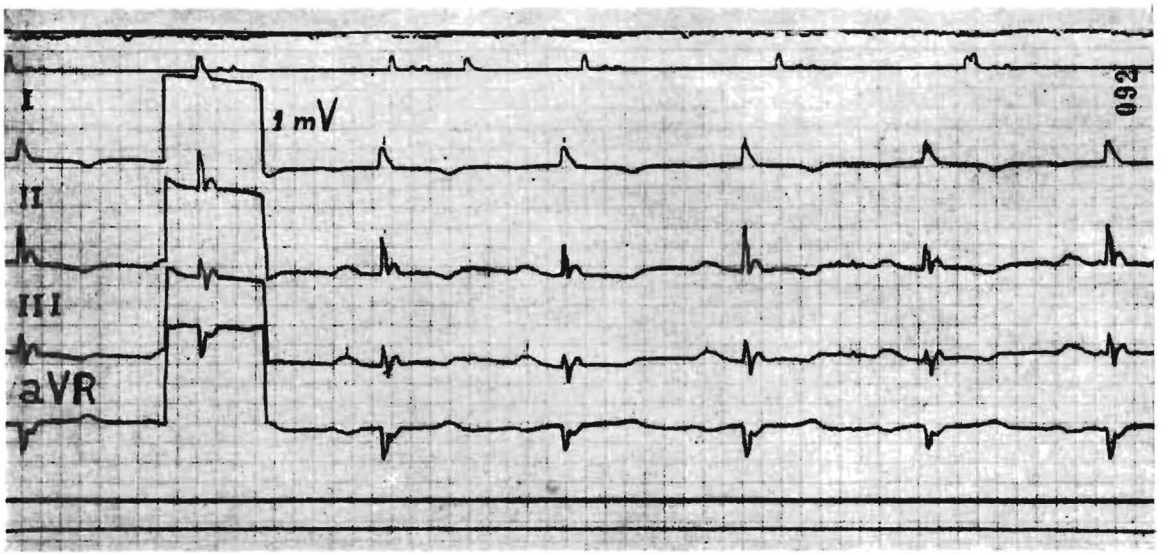


Fig. 15 — QRS alternation: Note the alternating size or amplitude of the QRS complex.

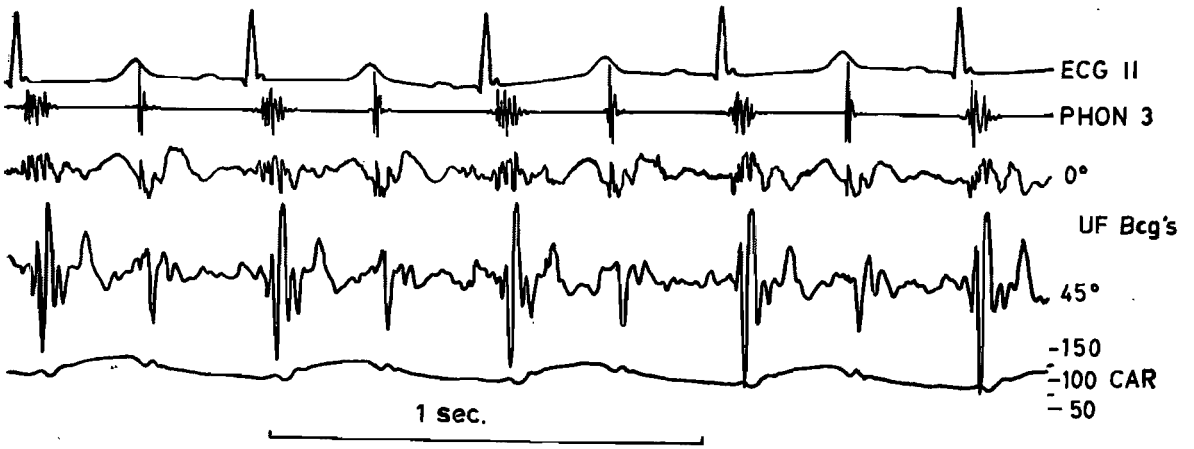


Fig. 16 — A comparison of the UF Bcga in the longitudinal body axis (0°) with that of a recording made in the dorso-ventral axis, approximately parallel to the ascending aorta (45°).

but at 55 days a marked deterioration again occurred. At 30 days ectopic (E) or premature heart beats of ventricular origin occurred (see ECG II in fig. 18 at 30 days). Improvement of the Bcg after a heart beat following an ectopic beat was noticed. This is clearly illustrated in fig. 18 at days 15 and 7. This heart beat was usually preceded by a long compensatory pause. The Bcg then showed a marked improvement of amplitude, configuration and slope of the HI and IJ segments. An improvement in the H wave was seen during some ectopic beats (see days 15, 7 and 0). From the 15th day before death severe changes in the Bcg were seen and the occurrence of ectopic beats as well as ectopic foci increased. Note the different configurations of the different ectopic beats in fig. 18. At seven days before death, three different ectopic foci were active and, on the day of death, four *foci* were generating impulses.

During ectopic beats which succeeded the previous normal beat after a short diastolic interval, only a weak first heart sound was recorded and the second sound was absent, thus indicating an insufficient beat and absence of ejection (see day 0). During these beats the Bcg was further decreased and hardly any segments could be detected. If a P-wave followed an ectopic beat while the AV valves were closed, a conspicuous venous pulse (VPa) was recorded (see fig. 18 days 7 and 0).

In one gousiekte goat in which the Bcg pattern had already deteriorated, the effect of adrenaline administration was investigated. The results are represented in fig. 19.

The maximum increase in amplitude was recorded 16 seconds after i.v. administration of 5ug/kg adrenaline. The Bcg pattern showed marked improvement as indicated by the increase in slopes of HI and IJ segments. Fifty-five seconds after adrenaline administration, the Bcg deteriorated again. The same procedure was repeated several times on this animal, as well as on a normal goat. In the normal animal, the same dose caused a much more marked increase in the amplitude of the HI and IJ segments even 8 seconds after administration (see fig. 20). These results indicate an early decrease in cardiac contractibility.

Discussion

There was a great variation in the time of onset of the different symptoms observed on auscultation of animals suffering from gousiekte. Bearing this in mind, the following table was drawn up from the mean figures:—

Day		*%*		*Symptom*
1.5	—	24	—	dyspnoea.
6	—	86	—	tachycardia.
7	—	48	—	gallop rhythm.
10	—	72	—	hyperpnoea.
	—	67	—	splitting first heart sound.
11	—	66	—	systolic murmur.
	—	56	—	arrhythmia.
27	—	45	—	dull first heart sound.

Various combinations of these symptoms were recorded, e.g. a pansystolic murmur plus a gallop rhythm (see fig. 5) or early systolic murmur plus a gallop rhythm (see fig. 6). A tendency to the following pattern was seen: firstly, a dull first heart sound developed. This was then followed by

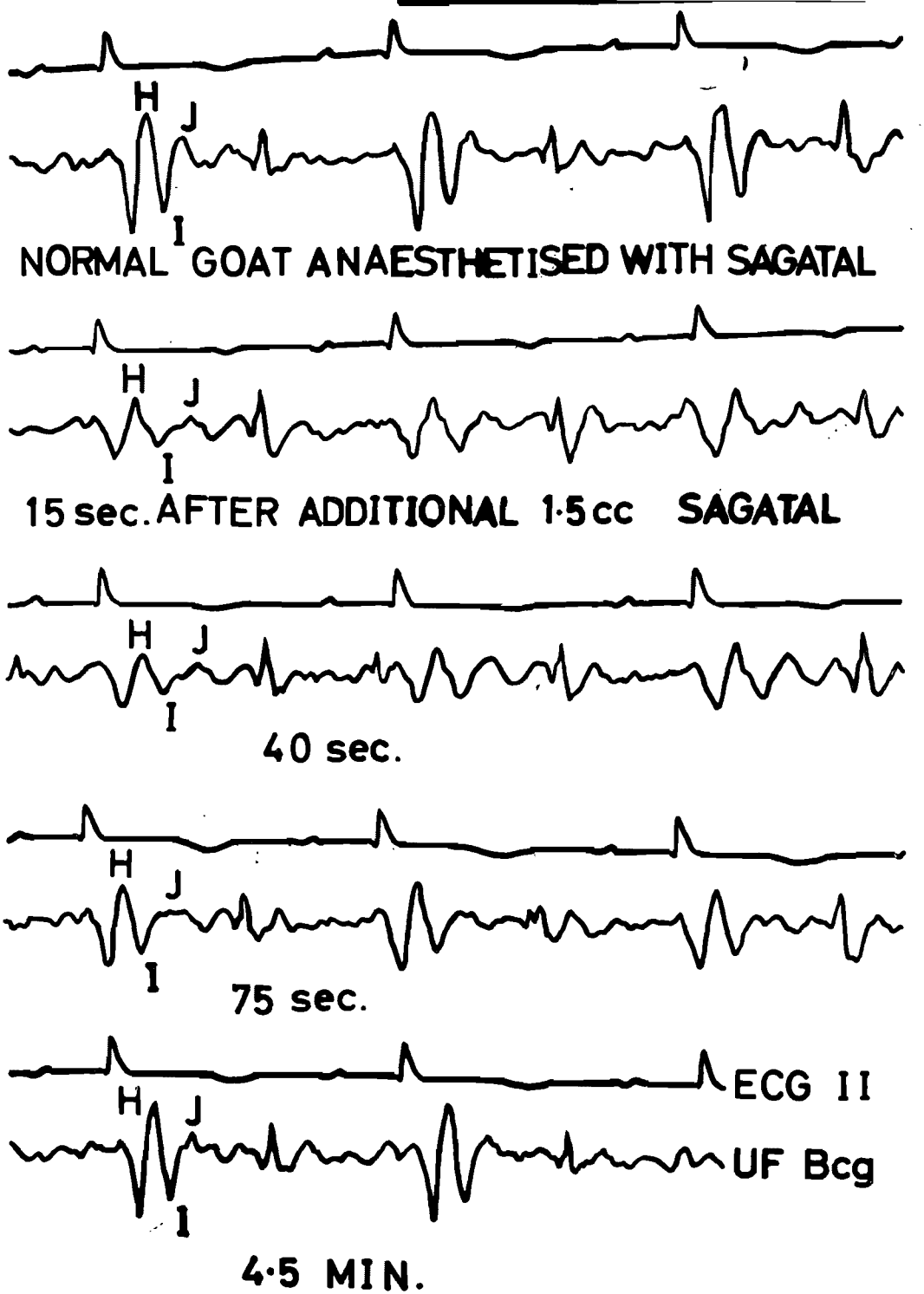
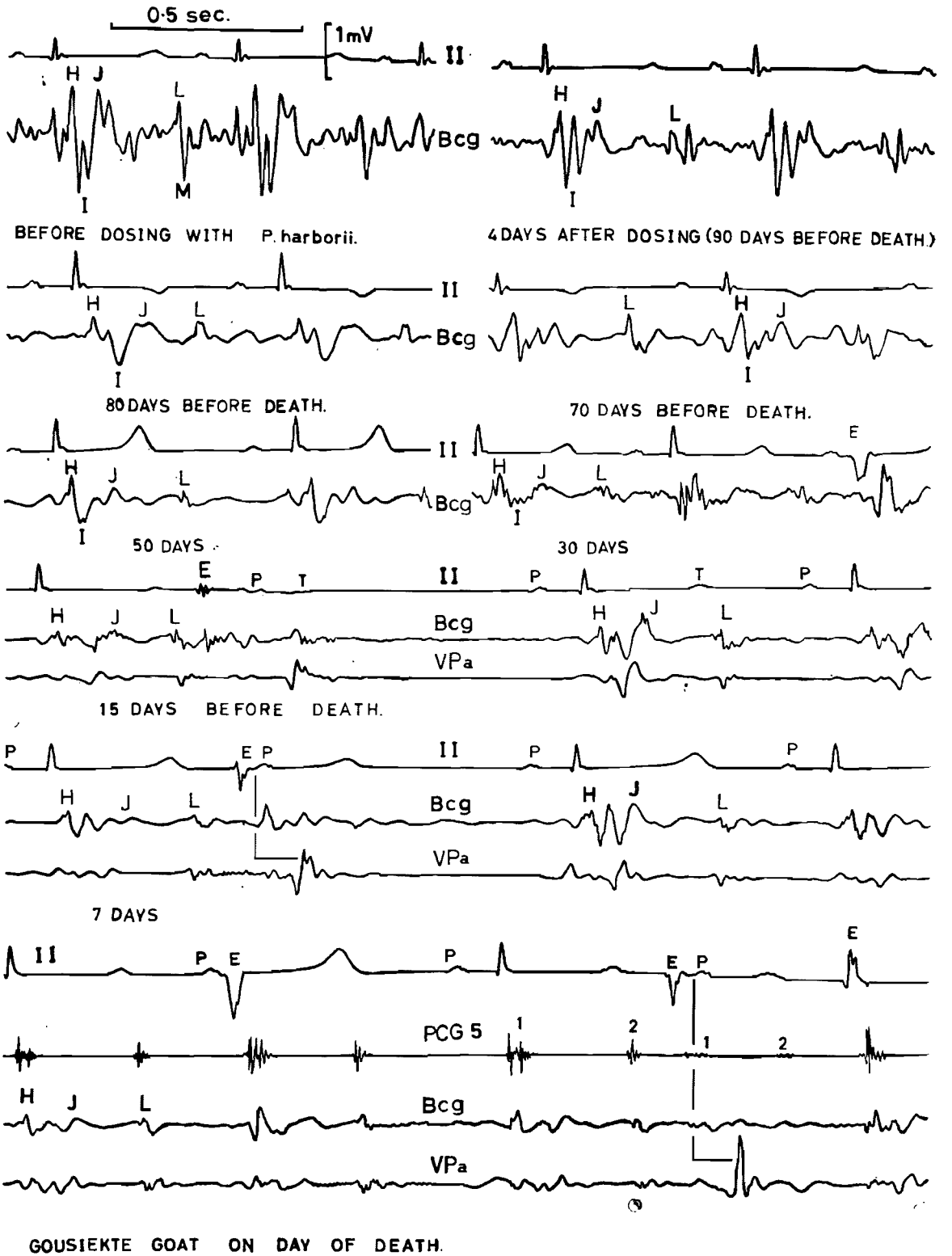


Fig. 17 — The effect of Sagatal (sodium pentobarbitone solution) on the UF Bcga of a gousiekte goat.



GOUSIEKTE GOAT ON DAY OF DEATH.

Fig. 18 — The UF Bcga changes recorded in a gousiekte goat from prior to dosing with P. harborii until the day of death.

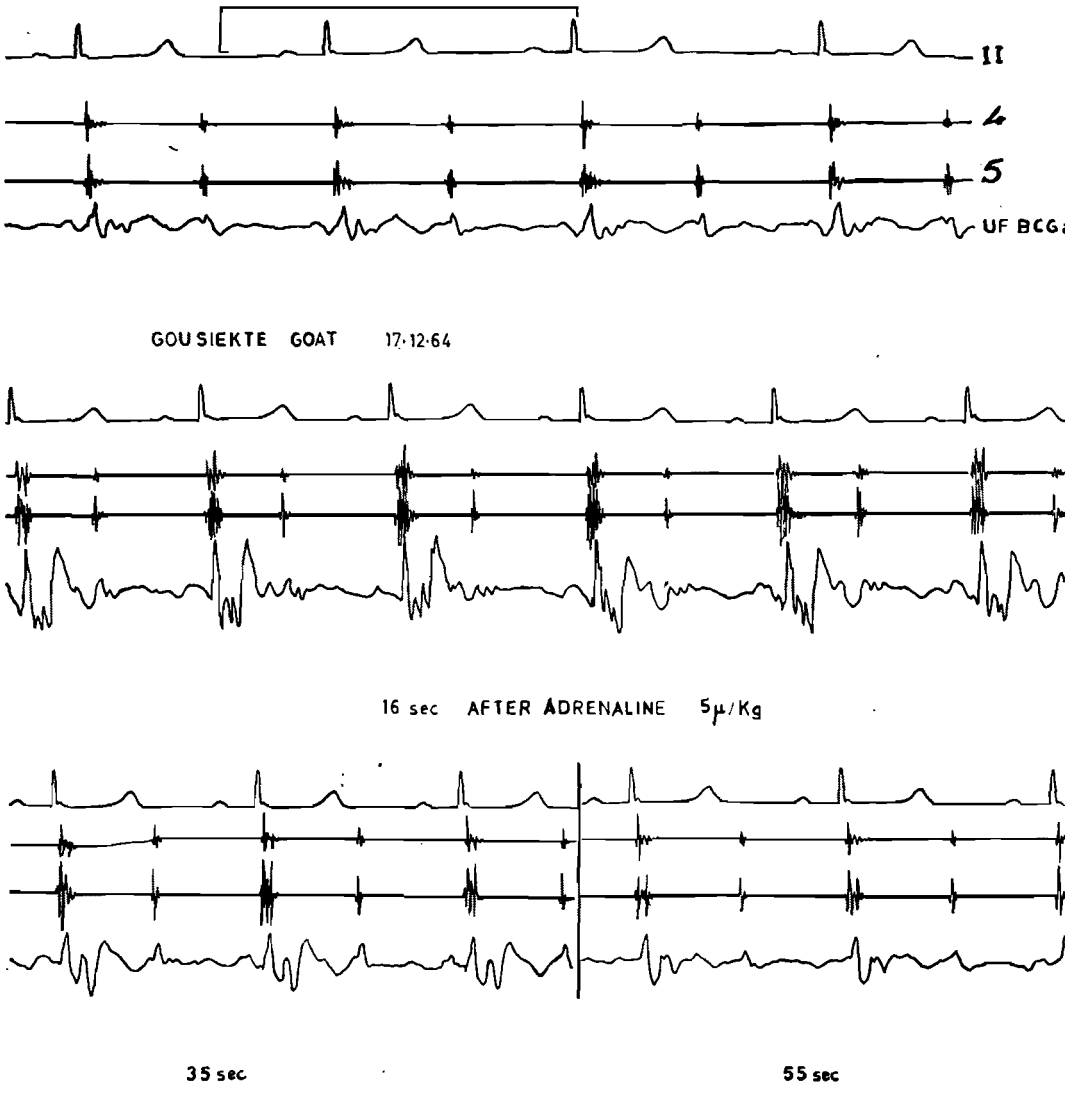


Fig. 19 — The effect of adrenaline on the UF Bcga of a gousiekte goat.

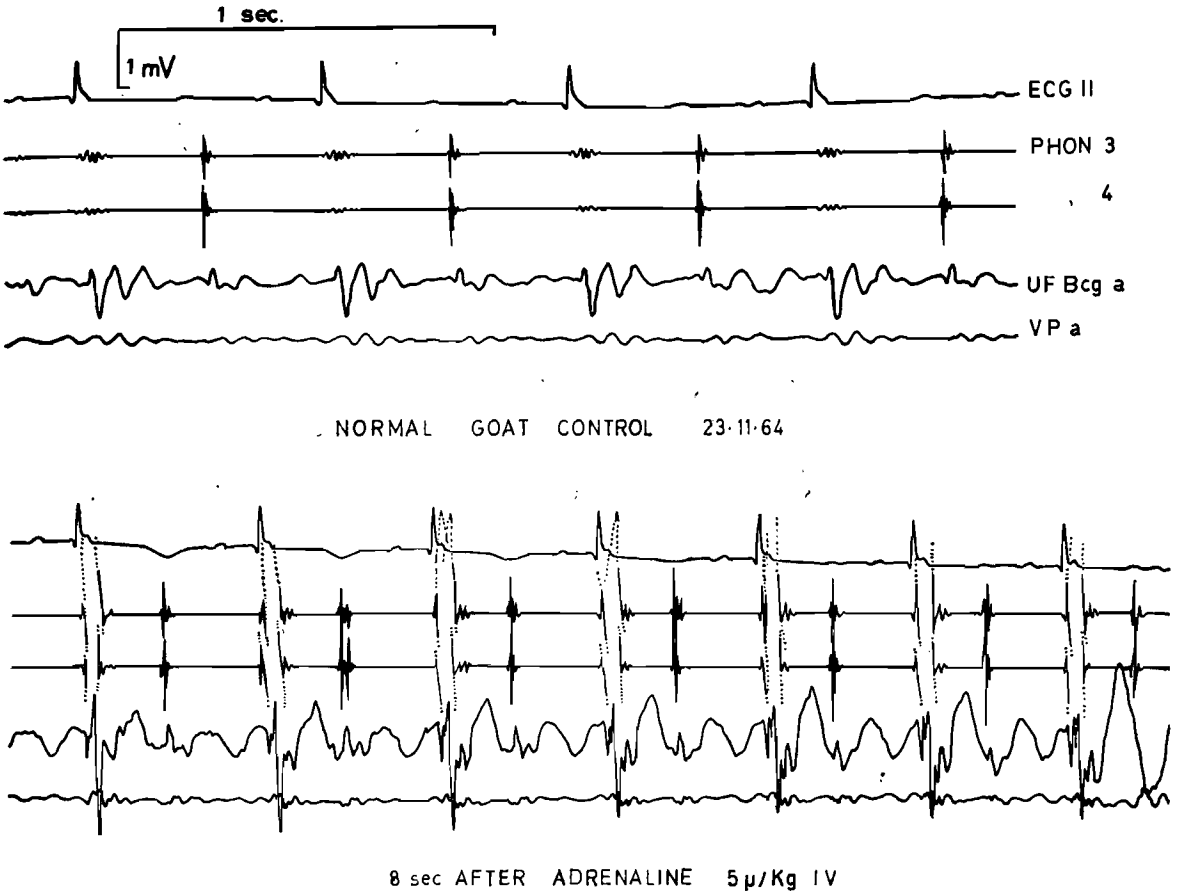


Fig. 20 — The effect of adrenaline on the UF Bcga of a normal goat.

a reduplication of this heart sound, followed by an early systolic murmur, followed by a pansystolic murmur. However, any one or more of these phenomena might be passed over, or remain static. Mid-systolic or late systolic murmurs might also be observed. Usually a tachycardia accompanied by an hyperpnoea then developed as a rule or could occur at an earlier stage. Arrhythmias appeared at any stage. Often it might even be the first change observed. A dyspnoea, when seen, usually developed terminally. In 10% of cases no symptoms could be detected on clinical examination. These animals were merely found dead in the morning. (No cardiographic studies were performed on these cases). Approximately four per cent of cases recovered, even after having developed severe symptoms.

While the dyspnoea is caused by the oedema of the lungs, the sinus tachycardia and hyperpnoea is most probably caused by the compensatory mechanisms of the body. The systolic murmur is due to AV valve insufficiency which is probably a sequel to the dilatation of the heart, as seen at necropsy. The anulus fibrosis probably dilates along with the muscular wall, thereby causing the valvular insufficiency. The gallop rhythm auscultated is probably caused by abnormal filling of the flabby ventricles. The possibility exists that some of these third heart sounds may be due to a summation of third and fourth heart sounds, consequent upon tachycardia. The splitting of the first heart sound is probably brought about by abnormal asynchronous closure of the AV valves, which in turn may be due to partial bundle branch block or to haemodynamic changes, e.g. increased pulmonary arterial pressure. The dull first heart

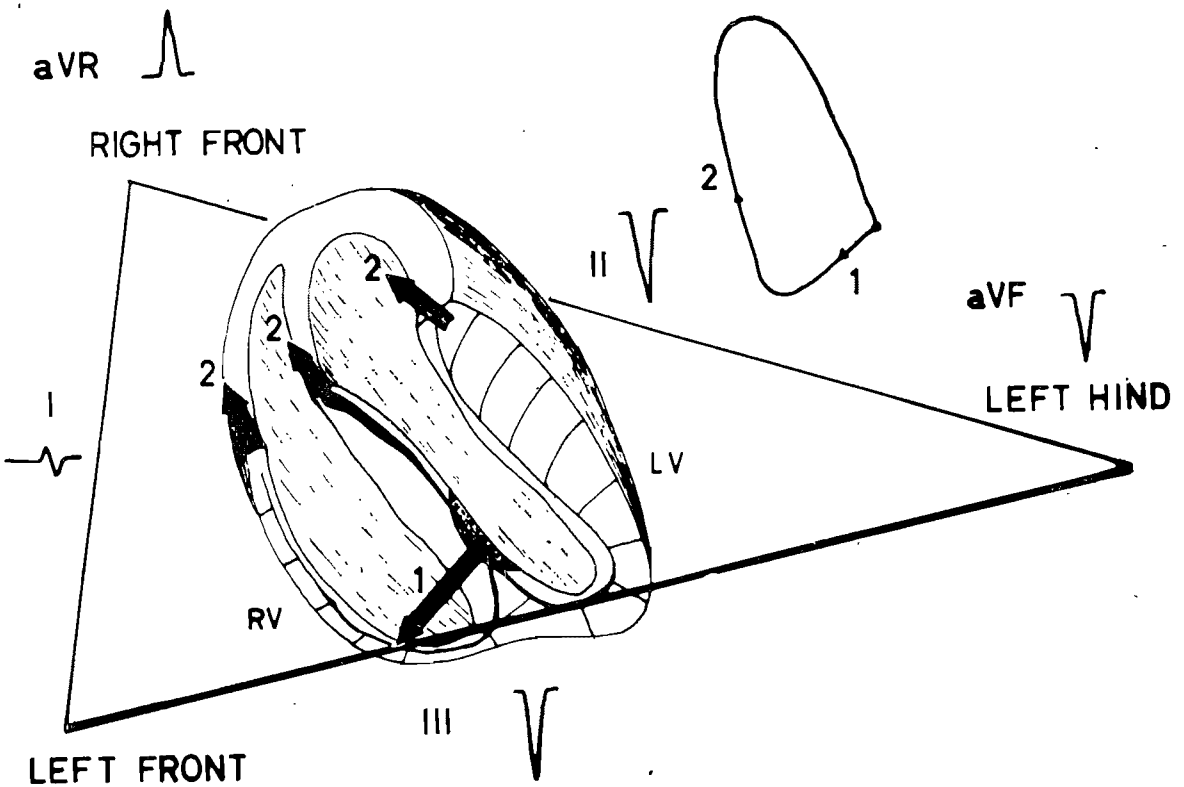


Fig. 21 — The pattern of activation of the myocard prior to ventricular contraction in the normal sheep and goat.

sound seems to be an incipient splitting of the first heart sound which may be summated with a systolic murmur.

Several factors hamper the application of human ECG findings in domestic animals. Due to the anatomical dorso-ventral placement of the heart and the attachment of the fore-limbs on both sides of the thorax over the heart, recordings made from the clissical limb leads do not record the electrical potential differences in the frontal plane as in man. Tracings thus obtained actually correspond to human records taken from the horisontal plane (see fig. 21). For this reason the position of the fore-limbs at the time of recording is critical.

Secondly physiological differences also play an important role. The activation of the ventricular muscle fibres in the sheep and goat occurs much more rapidly and with less magnitude than that of the dog and human being[10 11]. According to Hamlin[11], the activation of the ventricles in the goat starts as a cup shaped zone surrounding the apex of the left ventricle, including endocardial portions of the left septum and the free wall. During the next 10 sec of the QRS, the apical third of the interventricular septum is excited simultaneously from both endocardial surfaces, and both walls of the ventricles are suddenly activated. During the following 3-5 msec the ventricular base and rest of the left ventricular walls as well as the middle third of the interventricular septum are depolarized; this corresponds to the peak of the R-wave, as RS is inscribed during excitation of the basilar third of the interventricular septum. In recording surface potentials, this would mean that the main deflection in leads II, III, aVF and aVL will be directed negatively while aVR will show a positive QRS, which is paradoxical to what is found in the human (see fig. 21). Our records for sheep correspond well with the above (see table 5 and fig. 22), but in goats the position was not so clear at all (see table 4). Thirdly the potential difference between the fore-limbs was so small that although an amplification of 20 mm/mV was used, poor tracings were recorded in lead I

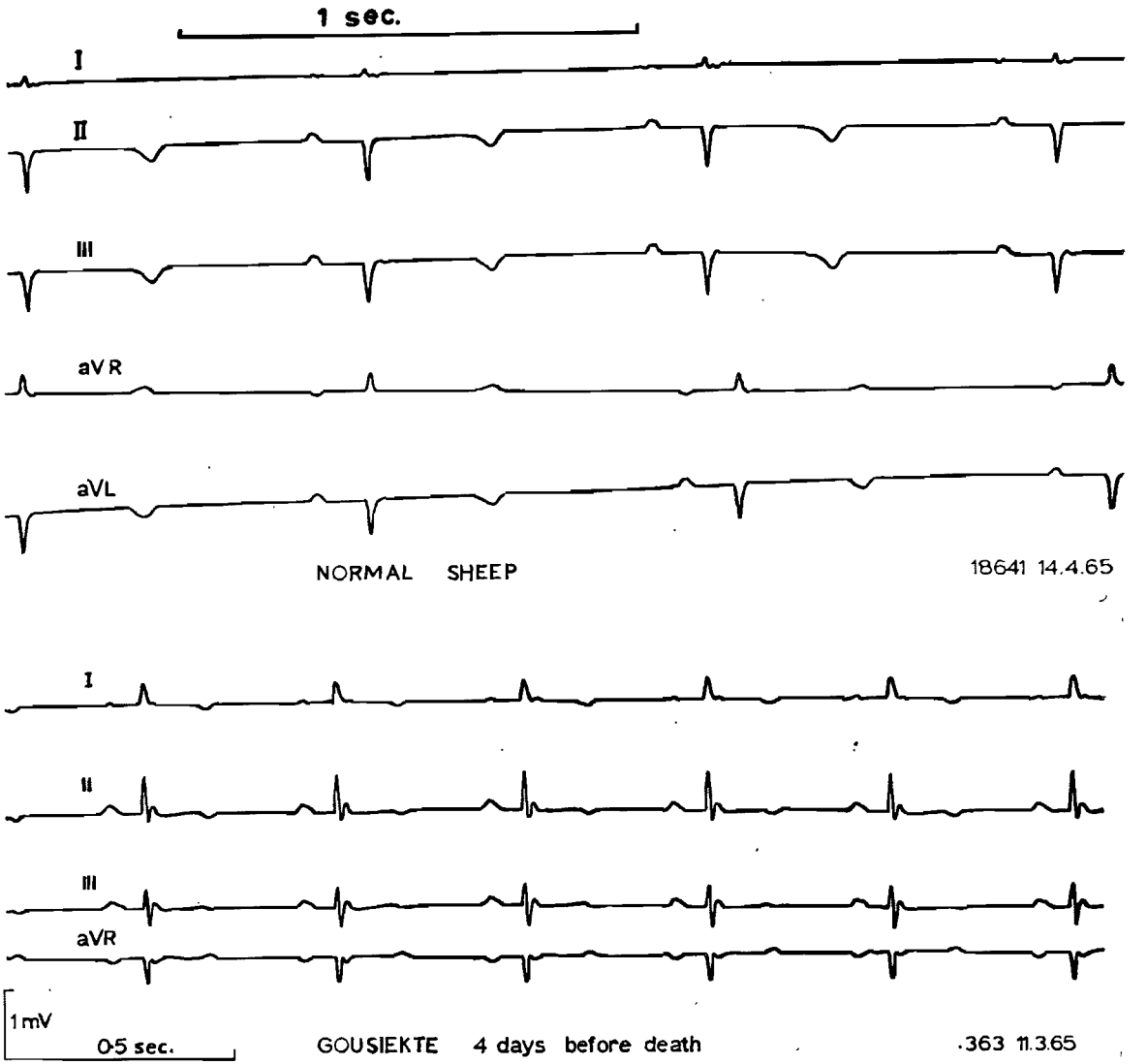


Fig. 22 — A typical example of ECG recordings in a normal and a gousiekte sheep.

and the variations so great that this lead was not used in the interpretation of results.

In the fourth instance various arrhythmias which are considered as abnormal in man occurred spontaneously in apparently healthy sheep and goats.

Sheep and goats, with gousiekte showed a definite change in QRS polarity (see figs. 12 and 22). Since no signs of ventricular hypertrophy were found on post mortem it was concluded from this that partial bundle branch block occurred. The QRS time increase during gousiekte, although small, supported this diagnosis. It was at the same time noted that most ectopic beats of ventricular origin during gousiekte or during administration of adrenaline to normal animals do not show a conspicuous time increase either. The mean increase in QRS time during ventricular premature beats in four gousiekte cases in lead II was only 156%. (Normal mean QRS was 0.032 sec while mean premature QRS time was 0.050 sec in goats). These findings are in accordance with the results of Hamlin et al[10] who found

that in sheep, when ventricular beats were evoked by pricking the heart mechanically a mean increase of 145% occurred. (Normal = 0.035 sec. while premature QRS = 0.051 sec. for lead aVF).

In dogs the increase was found to be 210%. It is therefore clear that the time increase in QRS duration above, is not of much value in diagnosing bundle branch block in sheep and goats.

No conclusions could be made from T-wave changes in sheep, because the T-wave showed a discordant pattern in 71 normal sheep. In goats the T-wave showed a positive wave in most normal animals. During gousiekte a diphasic or negative pattern predominated (see table 4). This change in T-wave may be indicative of relative cardiac ischaemia. Thus it became clear that the classical limb leads of the ECG cannot be used for individual diagnosis of gousiekte and is only of value in an experimental study.

Although the normal sheep and goats suffered peculiar sinus arrhythmias, the increase in occurrence of all these sinus arrhythmias was probably indicative of myocardial disease. It is clear that sinus arrhythmia cannot be used as a diagnostic sign in gousiekte. Again it is of value only in experimental animals subjected to examination before gousiekte is evoked.

The most conspicuous disturbance in cardiac rhythm observed during gousiekte is probably due to an increase in the occurrence of a so-called PP-time interval alternation. This is the only type of sinus arrhythmia that undergoes a marked increase.

None of the 71 normal sheep and 78 normal goats showed second degree incomplete AV block and only one case occurred among the sick animals. It is, quite clear, therefore, that the so-called dropped beat rhythm heard on auscultation cannot be ascribed to AV block as was anticipated. Second degree incomplete AV block may indicate heart disease in dogs, but is regarded as a normal finding in horses[12].

None of the normal animals showed ventricular premature beats. Therefore, the occurrence of ectopic beats in the sick animals was also considered to be indicative of myocardial disease. The fact that unifocal beats changed to multifocal premature beats after exercise may indicate a relatively ischaemic state of the myocardium during gousiekte. This might also have been the cause of the "irregular rhythm" heard on auscultation. The presence of QRS alternation may also indicate severe derangement of myocardial functioning (see fig. 15).

The changes in P-wave configuration during gousiekte are not related to changes in vagal tone and do not show any relation to the respiratory cycle. The incidence of P-wave changes in normal animals was much lower than in sick animals. As the administration of oxygen to animals showing conspicuous P-wave changes reduces the P-wave abnormalities, the conclusion was reached that the P-wave change in gousiekte was due to a wandering pacemaker caused by a relative ischaemia of the SA-node. The increase in P-wave time duration may indicate dilatation of the atria due to venous congestion.

The ballistocardiographic changes indicated early derangement of the energy releasing functions of the myocardium. The slope of the HI and IJ segments is the most sensitive indication of the ability of the left ventricle to accelerate the stroke volume during the ejection phase. This ability of the myocardium is related to contractility. The slope of all segments of the Bcg showed a marked decrease during gousiekte. This was assumed to be indicative of decreased contractility due to intoxication of the energy transformation mechanism by the active principle in the plant material. This may lead to venous congestion and dilatation. A vicious circle is started when dilatation causes AV insufficiency: the mechanical efficiency of the weakened myocardium is further lowered because more energy is required by the dilated heart to develop the normal pressure according to the law of Laplace.

Conclusions

In gousiekte the animals develop a cardiac insufficiency which is characterised by:—

(i) Functional cardiac dilatation, which causes symptoms of AV-valve insufficiency, gallop rhythm, bundle branch block, and an increase in P-wave duration.

(ii) Cardiac ischaemia, which causes symptoms of wandering pacemaker, bundle branch block and ectopic ventricular beats.

(iii) Decreased myocardial contractibility, which causes symptoms of generalised congestion, lung oedema, hydrothorax, hydropericardium and ascites.

It appears as if the primary lesion in gousiekte is an inhibition of the contractile mechanism of the entire myocardium, probably on a biochemical level, by the toxic principle in the plant.

The genesis of the well known histological lesion[1],[8] in the myocardium was not studied simultaneously, but it is anticipated that it is either a sequel to the cardiac ischaemia or that during the inhibition of the biochemical process of contractibility other vital metabolic processes are also inhibited, and that this is then followed by the productive myocarditis.

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The Chief of the Veterinary Research Institute, Onderstepoort and the Rector of the University of Potchefstroom for C.H.E. are thanked for permission to publish this paper. The assistance of Mr. J. J. van der Walt, lecturer in the Department of Physiology of the latter university, in handling the recording equipment is acknowledged. Part of the apparatus used was obtained by the University of Potchefstroom through a grant from the C.S.I.R.

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BOOK REVIEW

Animal Nutrition by P. McDonald, R. A. Edwards and J. F. D. Greenhalgh

Publishers: Oliver & Boyd Ltd. (1966). Edinburgh 1, Great Britain. pp. 407. Price 57/6d

Animal Nutrition consists of 22 chapters. A third of the book deals with basic facts of carbohydrates, lipids, proteins, vitamins, minerals, enzymes, digestion and metabolism. Feed evaluation and feeding standards cover one quarter of the volume. The remaining portion discusses some aspects of ensilage, grass, hays, roots, grains and their byproducts, protein concentrates and growth stimulators.

This book has been prepared for agricultural students in Britain. For veterinary students one would have liked to see the chapters on vitamins and minerals in particular, strengthened considerably. For example very little is said about the recent developments on selenium responsive conditions or the required levels. All the emphasis is placed on toxicity.

The authors prepared the book as a general approach to animal nutrition and insufficient emphasis is placed on the species approach. Furthermore it is considered that the various concepts discussed could in several cases have been presented with a more direct approach, so that the junior student for whom the book is intended may get a clearer understanding.

It is felt that the publishers have made a good presentation, errors have been carefully avoided, and that this book can be of considerable value to the nutrition student in his junior years.

PHILIP A. BOYAZOGLU.

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BOOK NEWS

It is no secret that many valuable microscopes have been ruined and many wrong diagnoses made purely as the result of a lack of proper knowledge of the construction and operation of this delicate instrument.

We have thus no hesitation in recommending **INTRODUCTION TO MICROSCOPY** by G. W. White, which aims at providing a basic knowledge of the main principles and techniques of microscopy. It is a book that will be instructive and of interest to every user of a microscope. 264 pages; many illustrations; R3.00.

MICROBIOLOGICAL METHODS by C. H. Collins is essentially a practical bench book for all microbiologists, and has been described as "An excellent introduction to the many aspects of a very large subject"; 340 pages; many illustrations; R5.00.

Of interest to mycologists, physiologists, biochemists, cytologists and geneticists is **THE FUNGUS SPORE,** Proceedings of the Eighteenth Symposium of the Colston Research Society held in Bristol in March 1966; 354 pages; illustrated; R12.53.

SURGERY OF THE DOG AND CAT, by A. Noel Ormond, is a practical handbook on the operative surgery of small animals by a veterinarian in general practice, which sets out to provide in a single volume descriptions of the surgical methods and essential equipment which will give consistently good results in day-to-day practice. It is a thoroughly practical guide, and will be of great value to the student and small animal practitioner. 242 pages; 158 drawings; 16 plates; R5.50.

HAEMATOLOGICAL TECHNIQUES FOR USE ON ANIMALS by R. K. Archer is a small book which aims at gathering together techniques for obtaining and examining blood from a number of different species. It also gives a table of some normal haematological figures in different animals, and makes brief mention of aspiration biopsy techniques and of blood transfusion, 135 pages; 24 figures; many tables; R2.20.

The third edition of **NEWSOM'S SHEEP DISEASES** completely revised by H. Marsh is now available. As a reference book for veterinarians, students and research workers this popular publication needs no recommendation. 456 pages; 113 figures; R8.85.

CANINE PEDIATRICS: DEVELOPMENT, NEONATAL AND CONGENITAL DISEASES by M. W. Fox brings together for the first time diverse aspects of canine medicine, nutrition, physiology and genetics. The text is orientated towards dog breeder and clinician, and various aspects of nutrition and management of the pregnant dog and of the offspring are discussed. 148 pages; illustrated; R6.35.

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While no sodium retention or potassium depletion has been observed at the doses recommended in animals receiving Predef 2X Liquid, as with all corticoids, animals should be under close observation for possible untoward effects. If symptoms of hypopotassemia should occur, corticoid therapy should be discontinued and 5% potassium chloride solution administered by intravenous drip.

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ACACIA NILOTICA (L.) DEL. SUBSP. *KRAUSSIANA* (BENTH.) BRENAN. AS A POISONOUS PLANT IN SOUTH AFRICA

M. TERBLANCE*, J. G. PIENAAR*, R. BIGALKE*, J. VAHRMEYER**.

SUMMARY

ng among goats with the ripe pods of *lotica* (L.) Del. subsp. *kraussiana* (Benth.) reported, and confirmed experimentally. ıppears to be the first proven case, the toxicity, symptoms and the chemical and morphological pathology are described. The main features of the disease are methaemoglobinaemia, abortion, dyspnoea, tachycardia, ruminal-atony, anaemia and slight hyperglycaemia. Liver damage and kidney dysfunction were often also encountered.

Fig. 1

INTRODUCTION

Acacia nilotica (L.) Del. subsp. *kraussiana* (Benth.) Brenan is a thorn tree which is widely distributed in the northern and eastern Transvaal, Natal and South West Africa. (See botanical description in appendix 1.)

The plant is rich in tannins and is extensively used for tanning and as a livestock fodder[1].

Although Watt[1] states that Greshoff found the sap from the seeds poisonous and Kobert[2] reported the isolation of a saponin from it, this plant has not been regarded as toxic to domestic animals in this country to date.

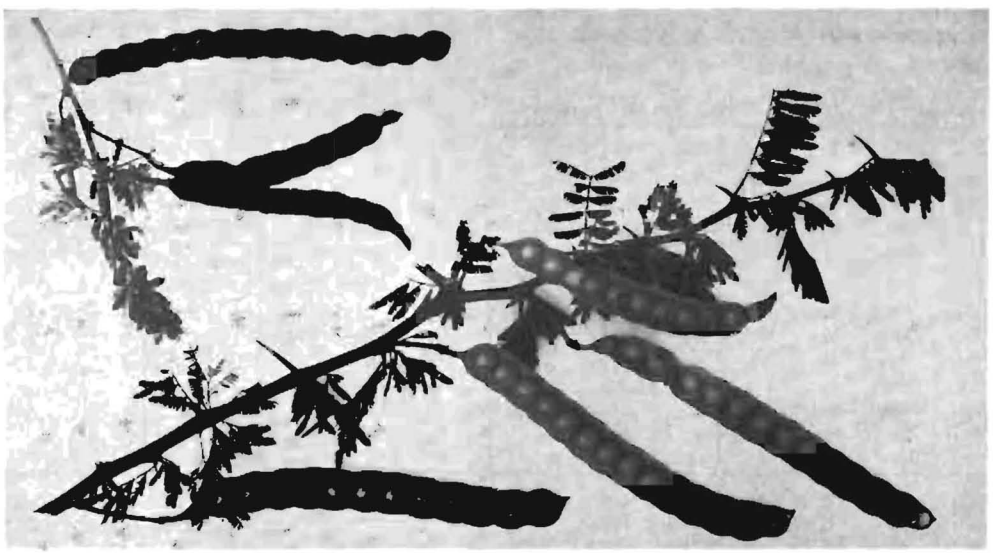

Fig. 2

* Veterinary Research Institute, Onderstepoort.
**Botanical Research Institute, Pretoria.

In July 1963, 19 goats (improved native breed) out of a flock of 160 became ill in Estcourt, Natal, and seven died. They showed listlessness, fever and a marked diarrhoea just prior to death. On post mortem, tumor splenis and gastro-enteritis (but no methaemoglobinaemia) were seen. The owner had felled a large number of *Acacia nilotica* trees, and the goats had probably consumed more of the pods than usual. A sample of ripe pods was forwarded to this laboratory by the State Veterinarian, Estcourt, Natal and proved toxic to a goat at a high dosage. The outstanding lesion in this animal was a marked methaemoglobinaemia. (See appendix[2], goat no. 1.) The pods were negative for nitrates, nitrites or oxidising agents with the diphenylamine reaction described by Feigl[3].

During June 1965, a farmer near Brits, Transvaal, reported abortions in his goats. Attempts to isolate any causal organisms from foetal and placental material failed. Some of the aborting animals died and showed anaemia, icterus and a slight methaemoglobinaemia. When mortality spread to castrated male goats, which showed marked methaemoglobinaemia, poisoning was suspected. On local investigation no evidence of nitrate or nitrite poisoning could be found. Examination of the camp in which the animals were grazing revealed the presence of *Acacia nilotica* (L.) Del. subsp. *kraussiana* (Benth.) Brenan trees in full seed (unripe and ripe pods). According to the owner, abortions and deaths occurred shortly after the animals were allowed to graze on the area of the farm on which these trees occurred. The area on which the animals had been grazing previously contained few of these trees with no seed.

Affected animals were treated symptomatically with methylene blue i.v. at 4 mg/kg. and dosed a solution of sugar and vinegar for atony of the fore-stomachs. They were removed from further exposure to the plant. Most of these animals recovered and no further cases occurred. Pods were collected from the trees and the results of tests performed on them are presented here.

Two of the affected goats were brought back to the laboratory for observation. Apart from methaemoglobinaemia, these animals showed a marked macrocytic hypochromic anaemia and a slight increase in serum glutamic-oxaloacetic transaminase and bilirubin levels, indicating some liver damage. A slight hyperglycaemia was also present, but no indications of kidney dysfunction. These abnormalities gradually disappeared. The relevant figures are given in Table 1.

Citrated blood drawn from two of the goats was injected into a splenectomized goat and *Eperythrozoon ovis*[4] was isolated. Inoculation of non-splenectomized animals however only produced a mild transient anaemia which showed no resemblance to the severe disease seen on the farm. It was therefore concluded that *E. ovis* was not the aetiological agent in the disease.

The pods again showed no nitrates, nitrites or other strong oxidising agents. It was noticed that

TABLE 1.—THE CHEMICO-PATHOLOGICAL EXAMINATION OF A NATURAL CASE OF *A. Nilotica* POISONING IN A GOAT.

Days	1	3	8	13	17	23	31	41
SR mm/h.....	14	14	14	9	12	10	8	7
PCV %.....	10	11	12	15	14	15	15	21
Haemoglobin g%.....	2.8	3.8	4.0	4.3	4.3	6.0	5.5	7.3
Red cell count × 10^6/ cumm.....	2.1	2.6	—	—	4.4	—	6.1	—
MCV $^3\mu$.....	47.6	42.3	—	—	31.8	—	25	—
MCHC %.....	28	34.5	—	—	30.7	—	36.7	—
SGOT K.U.....	174	200	80	139	89	86	74	68
SGPT K.U.....	67	79	50	52	50	42	48	36
UBR mg%.....	0.4	0.1	0.1	0.1	0	0.1	0.3	0
CBR mg%.....	0.2	0.1	0	0	0	0	0	0
Glucose mg%.....	83	72	66	76	57	59	45	41

SR = Sedimentation rate.
PCV = Packed cell volume.
MCV = Mean corpuscular volume.
MCHC = Mean corpuscular haemoconcentration.
SGOT = Serum glutamic oxaloacetic acid transaminase (King units).
SGPT = Serum glutamic pyruvic acid transaminase (King units).
UBR = Unconjugated bilirubin.
CBR = Conjugated bilirubin.

they contained large amounts of tannins in accordance with the findings quoted by Watt[1].

Toxicity Trials

Experiment 1. The dry ripe pods received from Estcourt, were chopped and dosed through a rumen fistula to an adult female goat (no. 1) in increasing amounts over 10 days (see appendix 2). The animal was found dead on the 11th day. No significant clinical or chemico-pathological changes had been observed. On post mortem a marked chocolate coloured discolouration of the carcass, blood and all the organs was found. A specimen of blood-coagulum from the ventricles was extracted in water and examined under a Hartridge Reversion Spectroscope. The typical absorption bands at 500, 540, 580 and 630 m μ for methaemoglobinaemia[5] were found. Furthermore, a slight emphysema and oedema of the lungs, congestion of liver and spleen and a degree of ruminal atony were seen. The ruminal contents had the typical sweet smell of the ground pods. Histologically, marked nephrosis with pigment casts causing a dilatation of the kidney tubules was seen.

Experiment 2. The dry ripe pods collected on two occasions at Brits were ground and dosed through a stomach tube to six adult female goats of which five were heavily pregnant. (See appendix 2, goats nos. 2 to 7.)

Acute Toxicity: Goat no. 2 (pregnant) received increasing dosages from 5.0 g/kg to 20.0 g/kg over 7 days. On the 7th day clinical symptoms developed and dosing was stopped. On the 12th day the animal aborted and appeared very ill. (See table 2 and appendix 2.) It died on day 14. The remainder of this batch of material was dosed to goats 6 and 7.

The second batch of material was dosed to goat no. 3 which was non-pregnant. It received increasing dosages from 10 to 30 g/kg over 3 days. On the 4th day symptoms developed and dosing was stopped. The goat was treated with 4.3 cc of a 4% solution of methylene blue once on the 6th day and on two occasions with an interval of 5 hours, on the 7th day. In spite of the treatment it showed marked symptoms (see appendix 2 and table 4) and died on the 8th day before further treatment could be applied.

Goats nos. 4 and 5 received single dosages of 20 and 30 g/kg respectively. No. 4 gave birth to two healthy kids the following day and apart from showing slight anorexia on that and the next day, came to no harm. Goat no. 5, however, developed symptoms on the second day which worsened (see appendix 2 and table 3) until she aborted twins on days 7 and 8 and died a few hours later.

Chronic toxicity: Goats nos. 6 and 7 received a daily dose of 5.0 g/kg over 31 days. No. 6 which received 23 doses (dosing was not performed on Saturdays and Sundays), showed slight symptoms but no methaemoglobinaemia between days 7 and 22 and gave birth to a healthy kid on day 24. It was discharged from the experiment on day 36. No. 7 showed slight symptoms from day 2, aborted on day 4 and showed dirty discoloured mucous membranes from day 8. A true methaemoglobinaemia did not develop and the animal was discharged on day 42 after receiving a total of 22 doses over 31 days.

Symptoms

Acute toxicity: Two of the three pregnant goats receiving high dosages over a short time, aborted. The dosage in the third (goat no. 4) was probably subtoxic. Four of the five goats developed a methaemoglobinaemia (only no. 4 did not). Most of the cases developed a tachycardia, hyperpnoea (see tables 2 and 3) ruminal stasis, anorexia, listlessness, muscular weakness and soft faeces. The goats acquired a sweet putrid (garlic-like) smell and showed no fever. Terminally, inability to

Table 2.—The results of the Clinical and Chemico-Pathological Examination of Goat No. 2.

Days	1	2	3	5	6	7	8	9	10	11	12	13	14
Pulse/Min	96	92	96	92	96	124	124	120	124	116	162	132	96
Resp/Min	32	32	36	32	28	44	40	44	44	40	42	36	24
SR in mm/hour.....	3	3	—	3	3	—	3	—	—	—	2	—	9
Packed cell volume %	42	36	—	35	34	—	36	—	—	—	31	—	16
Haemoglobin in g%...	12	11	—	11	10	—	11	—	—	—	10	—	6
SGOT in K U	99	92	—	119	112	—	82	—	—	—	319	—	305
SGPT in K U	24	36	—	48	48	—	42	—	—	—	202	—	131
Creatinine in mg%....	0.3	1.4	—	0.9	1.7	—	—	—	—	—	1.7	—	5.8
BUN in mg%.....	18	15	—	13	13	—	18	—	—	—	48	—	105
Glucose mg%.....	45	53	—	53	44	—	64	—	—	—	108	—	44

BUN = Blood Urea Nitrogen.

stand, putrid diarrhoea and dyspnoea were seen in some cases. These animals breathed with their mouths wide open and the necks stretched forward making gasping movements which might be accompanied by short anxious bleats on expiration. Occasionally a cyanosis could be discerned together with marked methaemoglobinaemia.

Chronic toxicity: Of the two animals dosed with a relatively small dosage over a long period, none died and only one aborted. Only occasional spells of tachycardia and hyperpnoea were seen. There was very slight methaemoglobinaemia, as indicated by dirty discoloured mucous membranes but this could not be confirmed by spectroscopic examination of the blood. No significant chemico-pathological changes were found.

Chemico-pathology: The methods used are described in a previous paper[6]. A definite increase in serum glutamic-oxaloacetic and glutamic-pyruvic transaminase were observed in goats nos. 2 (see table 2), and 5 (see table 3). Goat no. 5 also showed an increase of serum bilirubin which was mainly due to conjugated bilirubin (see table 3). This was interpreted as liver damage occurring in the later stages of the disease. Simultaneously an increase of serum creatinine and blood urea nitrogen was observed in goats nos. 2, 3 and 5 (see tables 2, 3 and 4) which was interpreted as kidney dysfunction.

There was also a tendency to the development of hyperglycaemia in these animals (see tables 2, 3 and 4).

In two cases where we were able to collect blood a few hours before death, a terminal anaemia was demonstrated (see tables 2 and 3). In both instances there was an increase in the sedimentation rate and a decrease in packed cell volume, haemoglobin concentration and red cell counts. In both instances the anaemia was classified as normocytic, normochromic (slightly hyperchromic). In the case of goat no. 2 the red cell count (R.C.C.) was 6.9 x 10^6/cu mm, the mean corpuscular volume (MCV) 23.2 μ^3, the mean corpuscular haemoglobin concentration (MCHC) 39% and in the case of goat no. 5 these were 7.5 x 10^6/cu mm MCV, 18.7 μ^3 and MCHC, 51.7%. It is thought at this stage that the indications of a slightly hyperchromic anaemia is an artifact following too high haemoglobin readings on the colorimeter due to the high concentration of methaemoglobinaemia. Drabkins solution only was used as a blank.

Pathology

Macroscopic Pathology.

Three natural cases from the Brits farm were available for autopsy. One, a pregnant female, killed *in extremis* for autopsy, had two autolysed foetuses *in utero*. The second animal, which ar-

Table 3.—The Result of the Clinical and Chemico-Pathological Examination of Goat No. 5.

Days	1	2	3	4	5	6	7	8
Pulse/Min.	108	88	128	168	204	208	188	—
Resp./Min.	44	36	40	40	44	36	32	—
Rumen/5 min.	7	4	5	6	0	0	0	—
SR mm/h.	5	4	5	2	3	—	—	10
PCV %	35	32	34	35	31	—	—	18
Haemoglobin g %	11	10	10	7.8	10	—	—	7.3
SGOT K.U.	129	92	132	226	267	—	—	342
SGPT K.U.	33	42	42	118	118	—	—	228
UBR mg%	0.2	0.1	0.1	0.1	0.1	—	—	0.4
CBR mg %	0.1	0.1	0	0	0	—	—	1.2
Creatinine mg%	0.6	—	2.8	2.2	1.8	—	—	4.2
BUN mg %	18	18	28	50	48	—	—	94
Glucose mg%	34	64	40	48	72	—	—	51

Table 4.—The Chemico-Pathological Examination of the Blood of Goat No. 3.

Days	1	2	3	6
Creatinine in mg %	1.1	1.1	1.1	14
BUN in mg %	20	13	9	97
Glucose in mg %	46	45	43	83

rived dead, also revealed intrauterine foetal death prior to maternal death. The third animal was a castrated male.

The main gross pathological features of the natural cases were; anaemia, slight icterus, haemoglobinaemia, haemoglobinuria, nephrosis, centrilobular necrosis of liver, diffuse focal necrosis of myocardium, slight oedema of lungs, slight hydropericardium, tumor splenis, ruminal atony and some degree of constipation. Marked necrosis of the foetal cotelydons was evident. Methaemoglobinaemia was more pronounced in the castrated male than in the females.

In the experimentally produced cases a marked methaemoglobinaemia was the most striking feature. Anaemia, icterus, haemoglobinaemia, haemoglobinuria and a hydropericardium were absent. Very slight oedema of the lungs was seen and in only one case was focal necrosis of the myocardium present.

Microscopic Pathology.

Microscopic examination of sections from various organs confirmed the gross observations.

Severe nephrosis was present in all cases. Degenerative changes in the tubular epithelium varied from cloudy swelling to fine hyaline droplet degeneration. Some tubules had undergone necrosis of the epithelium. Hyaline casts were numerous. In addition, haemoglobin casts and slight haemosiderosis of the kidney were present. A varying degree of centrilobular degeneration and necrosis were present in all the livers. In the natural cases with icterus, mild bile stasis and mild splenic haemosiderosis were evident.

Diffuse focal areas of necrosis in the myocardium of some cases were seen as groups of intensely eosinophilic muscle fibres, with pyknotic nuclei. Pulmonary oedema was mild.

Discussion

Acacia nilotica (L.) Del. subsp. *kraussiana* (Benth.) Brenan pods in large amounts are therefore toxic to goats and this should be borne in mind where these pods are fed to ruminants, especially during droughts. The MLD is approximately 30 g/kg with a tendency to a cumulative effect, as 20 g/kg can be lethal if the animal has had previous access to the pods (see appendix 2). Even 5 g/kg/day over a long period can cause symptoms. Because such a large dose is required to cause death, it is anticipated that under natural conditions these pods will cause death only in browsers. Such a natural outbreak of poisoning has occurred and has been confirmed, although all the symptoms were not exactly simulated. For example, a marked macrocytic, hypochromic anaemia was an outstanding symptom in the natural cases. In the experimental cases it was noted that an anaemia developed only terminally during the last day and that this anaemia was normocytic normochromic. It is thought that the natural cases consumed smaller amounts of pods over a longer period and that they represented long standing cases. Two goats, (nos. 6 and 7) were dosed with small amounts over a long period. However, the above phenomena were not reproduced; the dose used probably being too low. Bilirubinaemia was reproduced in one case only, while icterus was very common in the natural cases. However, most of the experimental cases did develop liver damage and at the right dosage pattern icterus can probably also be produced.

It is postulated that the toxic principle causes a methaemoglobinaemia followed by anoxia and haemolysis and the resultant anaemia exaggerates the anoxia. This anoxic state is then responsible for the symptoms and lesions of tachycardia, hyperpnoea, dyspnoea, necrosis of foetal cotyledons followed by abortion and the other changes.

As no nitrates or other strong oxidising agents could be demonstrated in the pods, the possibility of an enzyme inhibitory factor was considered. However, a preliminary investigation of the erythrocytes of some of the above cases revealed no inhibition of the methaemoglobin reductase system or triose phosphate dehydrogenase, but there was a tendency to increased red cell fragility. As saponins may cause haemolysis without methaemoglobin formation[3] the possibility of more than one active principle might have to be considered.

ACKNOWLEDGEMENTS

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Appendix 1 *A. Description of the plant:*

Name: *Acacia nilotica* (L.) Del. subsp. *kraussiana* (Benth.) Brenan[7].

Synonyms: *Acacia arabica* (Lam.) Willd. var. *kraussiana* Benth. Hook.[8].

2. *Acacia benthamii* Rochebr.[9].
3. *Acacia nilotica* L. var. *kraussiana* (Benth.) A. F. Mill[10].
4. *Acacia subalata* auct. non Vatke, as to South African species[9];.
5. *Acacia scorpioides* (W. F. Wight) A. Chev. = *Mimosa scorpioides* Linn.[11] = *Acacia arabica* (Lam.) Willd.[12].
6. *Acacia vera* Willd[13].

Family: Leguminosae.

Popular names: Stinkpeul, Ruikpeul, Lekkerruikpeul, Redheart, Swartsaadpeul, Nslangwa (Sjangaan), Tshungapandu (Venda).

There occurs, therefore, in South Africa only one plant, namely *Acacia nilotica* (L.) Del. subsp. *kraussiana* (Benth.) Brenan.

Tree up to 25 ft. high, crown often umbrella-like, occasionally rounded especially in young trees. Bark black with longitudinal fissures. *Leaves* alternate, bipinnate; pinnae in 4 to 10 pairs, pubescent; pinnules in 10 to 24 pairs. *Spines* straight or subrecurved, robust or weakly developed, 5 mm to 10 cm long, paired, often white when well developed. *Inflorescens* globose, yellow, pedunculate. *Peduncles* axillary, 3 to 4 together in the leaf axils, pubescent; involucel (bracts) situated above or below the middle of the peduncle. *Individual flowers* numerous, packed closely together in the inflorescens, sweet-scented, sessile. Calyx campanulate, 4 to 5 lobed. *Corolla:* tube $\frac{3}{4}$ of the total corolla length ($\pm$ 3 mm), lobes rounded to acuminate. *Stamens* many, exerted. *Ovary* sessile, 2 to many ovuled; style filiform; stigma terminal, small. Pod flat, linear, moniliform like a string of pearls, tomentose at first, later glabrescent, black when ripe, 2 to many seeded, usually 5 to 15 seeded. *Seeds* compressed.

Distributed in Northern and Eastern Transvaal, Natal and S.W.A. Recorded in the following districts:—

Natal: Ingwavuma, Ubombo, Hlabisa, Umvoti, Ngotshe, Mapupulo, Nkandla, Estcourt, Lions River, Lower Mfolozi.

S.W.A.: Tsumeb, Okahandja, Otjiwarongo, Grootfontein, Kaokoveld.

Transvaal: Pretoria, Rustenburg, Brits, Naboomspruit, Waterberg, Zoutpansberg, Pietersburg, Malelane, Marico, Lydenburg, Witbank.

Acacia nilotica grows in relative dry and frostfree areas, in almost every kind of soil and in a variety of habitats like on open plains, mountain slopes (not very common), or in valleys (a more common habitat) where the trees grow clumplike.

Appendix 2

Toxicity Trials in Adult Female Goats

Goat no.	Weight in kg.	Dosage g/kg	Exper. days	Day aborted	Day of death	Symptoms
1	24.5	2.0	1	—	11	
		4.0	2			
		8.0	3			
		16.0/day	4–9			
		20.0	10			No symptoms. Methaemoglobinaemia on p.m. examination.
2 (pregnant)	30.0	5.0/day	1–3	12	14	No symptoms
		10.0	5			No symptoms
		15.0	6			No symptoms
		20.0	7			Tachycardia and hyperpnoea (see table 2).
		—	8–11			Ditto plus methaemoglobinaemia, anorexia, listlessness and muscular weakness.
		—	12			Ditto plus marked muscular weakness, dyspnoea, cyanosis, liver damage, kidney dysfunction, hyperglycaemia and anaemia (see table 2).
		—	13			In addition ruminal atony developed.
3	42.7	10.0	1	—	8	No symptoms.
		20.0	2			No symptoms.
		30.0	3			No symptoms.
		—	4–5			Methaemoglobinaemia.
			6–7			Ditto plus treated with methylene blue, soft faeces, goat has sweet putrid (garlic like) smell, compare smell of pods, kidney dysfunction and hyperglycaemia (see table 4).
4 (pregnant)	35.4	20.0	1	did not abort	did not die	No symptoms.
			2			Gave birth to a healthy kid.
			3–4			Slight anorexia. Discharged on day 15.
5 (pregnant)	45.4	30.0	1	7 & 8	8	No symptoms.
			2			Methaemoglobinaemia, soft faeces.
			3–4			Ditto plus anorexia, listlessness, muscular weakness and tachycardia (see table 3).
			5–8			Ditto plus complete ruminal stasis, dyspnoea putrid swelling diarrhoea, inability to stand, liver damage, bilirubinaemia, kidney dysfunction, anaemia and hyperglycaemia (see table 3).
6 (pregnant)	30.9	5.0/day	1–31	did not abort	did not die	Received 23 doses. Showed spells of tachycardia and hyperpnoea on days 7, 9, 14, 17, 20 and 22. Dirty-coloured mucous membranes from the 11th day. Gave birth to a healthy kid on 24th. Discharged on day 36.
7 (pregnant)	40.9	5.0/day	1–31	4	did not die	Received 22 doses.
			2–3			Soft faeces, paleness of mucous membranes.
			4			Ditto plus tachycardia (124/min), and aborted.
			5–6			Paleness of mucous membranes and muscular weakness.
			8–32			Dirty colour of mucous membranes. No significant chemico-pathological changes had developed. Discharged day 42.

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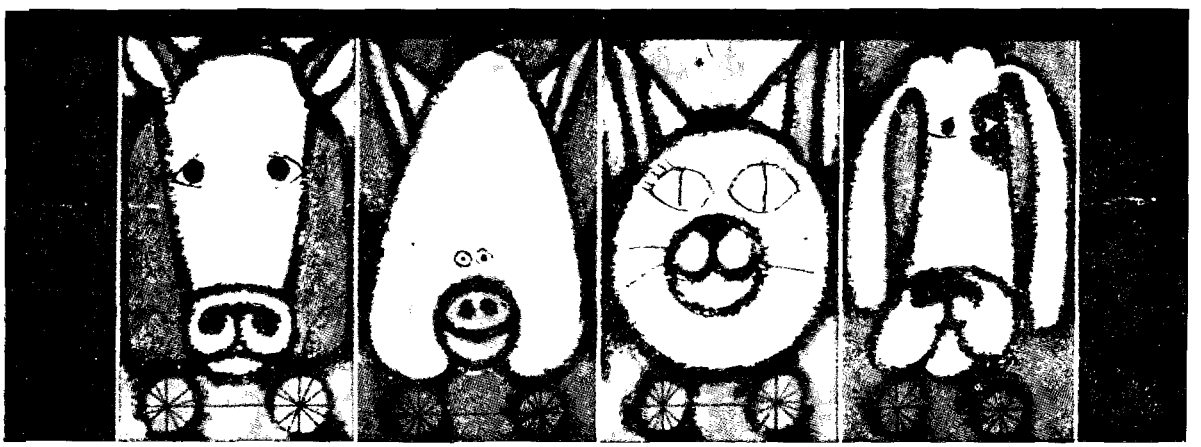
INJECTION and TABLETS



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LEPTOSPIROSIS: A BRIEF REVIEW, GENERAL CONSIDERATIONS AND INCIDENCE IN SOUTH AFRICA

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Summary

A comprehensive review is given of leptospirosis as a world problem with special reference to increase in incidence and serotypes, epidemiology and epizootiology, pathogenesis and control.

The methods of diagnosis, the interpretation of the agglutination-lysis test and its value in controlling the disease are briefly discussed.

Available data, based on a preliminary serological investigation, indicate that leptospirosis is a serious problem also in South Africa. The disease is widely distributed in the Republic, affecting dogs, pigs, cattle, horses and sheep, with *L. pomona, L. hyos, L. icterohaemorrhagiae* and *L. canicola* being the most prevalent serotypes.

Introduction

The question whether leptospirosis constitutes a threat to our animal industry, originates from the fact that serologically positive animals, particularly cattle, do not always reveal clinical manifestations and also that losses due to abortions may be minimal or even exceptional.

In spite of this, the joint W.H.O./F.A.O. Expert Committee on Zoonosis[1] stated that "leptospirosis now constitutes a major problem in cattle and a problem of undetermined size in swine."

Besides the abortion storms that can follow epidemics in cattle[1 5], and pigs[2 6 7], it must be born in mind that leptospirosis is a zoonosis[1 8], the epidemiology of which is characterised by spread from animal to animal and from animal to man, with man as the endpoint host[1 6]. These considerations more than justify implementation of strict control measures.

Considering the incidence of leptospirosis, the large number of serotypes involved, and the epidemiology and epizootiology of the disease, the question automatically arises as to whether we are able to control the disease effectively with the methods at our disposal. In formulating a standard method of control, these important aspects as well as the immunising value of vaccines all need to be considered.

1. *Incidence of leptospirosis.*

a. *General Incidence.*

Although disease conditions like "Schlamfieber" and Weil's disease had been recognised in the 1880's, the causative organism *Leptospira* was isolated only in 1915[9]. This was followed by the diagnosis of Stuttgart's disease, caused by *L. canicola*[10]. Until then the rat was considered the only possible reservoir.

In 1937, however Klarenbeeck and Winsser[10] isolated *L. icterohaemorrhagiae* from pigs showing jaundice and in the same year Wirth[11] showed that Weil's disease (*L. icterohaemorrhagiae*) was associated with leptospirosis in pigs. During the early 1940's, two additional serotypes, viz. *L. pomona* and *L. hyos*, were associated with leptospirosis in humans and pigs[12 13], bringing the number of known serotypes to four.

Since the initial establishment of leptospirosis as a disease complex in man and animals[10 14], its incidence has tended to increase[15 16] and in some countries the disease constitutes a serious problem [3 15 16]. Leptospirosis is today one of the most widely distributed of the zoonoses and has been reported from all parts of the world[1 17]. Although all serotypes have been encountered in nearly every country, specific serotypes are more prevalent in certain countries and areas. The international geographical prevalence of *Leptospira* according to Wolff[18] is given in table 1. (Page 68).

In this as well as in subsequent reports[1], no mention was made of the incidence of leptospirosis in the Republic of South Africa.

A survey conducted in the U.S.A. during 1960[2] revealed that 11.2 per cent of cattle and 22 per cent of pigs tested serologically were positive, 89 per cent of which were positive to *L. pomona*. This

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high incidence of *L. pomona* in the U.S.A. and its association with disease was confirmed by Babudieri and Gaspardis[3]. In addition these authors stated that *L. grippotyphosa* is by far the most prevalent serotype in the U.S.S.R.

In contrast to the general high incidence of *L. pomona* in the U.S.A., it had been shown in a previous survey[19] that 85 per cent of cattle tested in the State of Florida, were serologically positive to *L. sejroe*. In another area, again *L. hebdomadis* was the most prevalent serotype[3].

According to Wolff[18], *L. grippotyphosa, L. sejroe* and *L. saxkoebing* are the most prevalent serotypes in Italy. However, Babudieri and Gaspardis[3] indicated that 55 per cent of 1,000 cattle tested, were serologically positive to *L. icterohaemorrhagiae,* and that this was the most prevalent serotype in Northern Italy while in Southern Italy *L. saxkoebing* was most common. Fennestad and Borg Peterson[15 16] found 8 per cent of all cattle above the age of 12 months tested serologically positive for *Leptospira* in Denmark during 1956.

b. *Incidence in South Africa.*

Surveys conducted during 1942[20] and 1946[21] provided no evidence of the occurrence of leptospiral infection among rats in the Witwatersrand area. However, Malherbe and Kaschula in 1953[22] diagnosed *L. canicola* infection in the dog. In 1958, Gear *et al*[23] after diagnosing leptospiral-meningo-encephalitis in humans in Johannesburg, examined dogs and rats in the vicinity and found 60 rats (*Rattus rattus*) that were positive for *L. canicola,* the same serotype responsible for the human cases.

Stimulated by the occurrence of Weil's disease in the Cape[24], and the isolation of *L. icterohaemorrhagiae* from rats[25], Rademan, Steytler and Wright[26] examined a further 256 rats, 7 of which positive for *L. icterohaemorrhagiae.* This comparatively high incidence of leptospirosis in the Cape Peninsula was subsequently confirmed when 54 per cent of 100 dogs tested by Beyers[27] yielded agglutinin values ranging from 1:2000 to 1:160,000, specific to both serotypes *L. icterohaemorrhagiae* and *L. canicola.*

Before 1960 there was no direct evidence of the incidence of leptospirosis among cattle and pigs in the Republic. The first abortion storm in pigs, where leptospirosis was suspected, occurred in the Orange Free State during 1960[7]. In this particular epizootic more than 50 per cent of approximately 1,000 sows aborted. The aborted foetuses showed jaundice and subcutaneous oedema while a large percentage of piglets born alive, died days later.

This preliminary diagnosis of leptospirosis was serologically confirmed in 1965 at which time a national survey was introduced. This survey, which was originally started in the Western Cape and subsequently extended to the rest of the country, indicated that leptospirosis is widely distributed in the Republic. From April 1965 to July 1966, 3,654 pigs, 5,694 cattle, 100 horses, 8 dogs and 47 sheep, derived from 893 premises distributed throughout the Republic, were tested. The incidence of leptospirosis in terms of animals and premises found infected is summarised in table 2.

These preliminary results show that all species of domestic animals tested were found to be affected, with the highest incidence in dogs (25 per cent) followed by pigs (16.2 per cent), horses (9

TABLE 1.—INTERNATIONAL GEOGRAPHICAL DISTRIBUTION OF LEPTOSPIRA (WOLFF, 1954) AND DISEASES PRODUCED (HUMANS)

Serotype	Disease	Distribution
1. L.icterohaemorrhagiae	Weil's disease	World wide.
2. L. pomona	Swineherd's disease	
3. L. canicola	Canicola fever	
4. L. grippotyphosa	Mud fever	U.S.S.R., Germany, Italy, France, Switzerland, Netherlands, Denmark, Central Africa, Israel, U.S.A.
5. L. hebdomadis	Seven day fever	Japan, Formosa, Indo-China, Indonesia.
6. L. sejroe	Feldfieber	Denmark, Germany, Italy, Switzerland.
7. L. saxkoebing	Leptospirosis	Denmark, Northern Italy, Germany.
8. L. ballum	Laboratory infection	Denmark, Netherlands, U.S.A., Portugal.
9. L. autumnalis	Autumnal fever	Japan, Southern Asia, France, Congo.
10. L. australis	Cane fever	Australia, Switzerland, Germany, Italy, Southern Asia.
11. L. hyos	Swineherd's disease	Australia, Switzerland, Western Europe, S. America.
12. L. bataviae	Ricefield fever	Southern Asia, Denmark, Italy, Central Congo, Puerto Rico.
13. L. pyrogenes	Spirochaetosis	Indonesia, Southern Asia, Japan, Australia.

TABLE 2.—INCIDENCE OF LEPTOSPIROSIS IN SOUTH AFRICA
(March, 1965, to July, 1966)

	Transvaal and Highveld	Cape West	O.F.S.	Natal	S.W.A.	Total
PIGS						
Tested	820	2,563	29	242	—	3,654
Positive	61	518	10	3	—	592
Prem. Test	107	288	4	119	—	518
Prem. Inf.	17	62	1	3	—	83
CATTLE						
Tested	1,357	4,190	35	23	89	5,694
Positive	190	99	5	2	15	311
Prem. Test	41	320	2	4	8	375
Prem. Inf.	13	37	2	1	5	58
HORSES						
Tested	2	98	—	—	—	100
Positive	0	9	—	—	—	9
DOGS						
Tested	3	5	—	—	—	8
Positive	1	1	—	—	—	2
SHEEP						
Tested	—	47	—	—	—	47
Positive	—	3	—	—	—	3

Prem. Test. = Number of premises on which tests were done.
Prem. Inf. = Number of premises on which infection was found.

per cent), sheep (6.4 per cent) and cattle (5.4 per cent). However, in terms of numbers of animals tested, only the figures for pigs and cattle are in any way comparable and indicative of the actual incidence. The highest incidence in pigs was found in the Orange Free State (34.4 per cent) followed by the winter rainfall area (20.2 per cent) while no positive cases could be found in the Eastern Transvaal. The Orange Free State also showed the highest incidence (17.1 per cent) among cattle, followed by the Transvaal (14.4 per cent) while on various farms in the winter rainfall area leptospirosis was found in both pigs and cattle on the same premises.

Routine serological tests carried out at Onderstepoort showed that *L. pomona* (48 per cent) was by far the most prevalent serotype followed by *L. hyos* (24 per cent), while *L. icterohaemorrhagiae* and *L. canicola* were less frequently encountered, except in the dog.

2. *Leptospira serotypes*:

In many instances leptospirosis, in the different animal species, is confined to infection by a specific serotype or a limited number of serotypes. Although certain serotypes maintain a fair degree of host-specificity it is generally accepted that all the pathogenic serotypes can cause leptospirosis in any of the animal species and in humans. However, it would appear that certain host-specific serotypes produce clinical manifestations, including abortion storms, while infections by other serotypes are usually asymptomatic[3 6].

During the past decade great prominence was given to the large number of serotypes associated with leptospirosis in humans and livestock. The original association of *L. icterohaemorrhagiae* with leptospirosis in pigs and humans[10 13], was subsequently confirmed[30 33] and found to extend to cattle[4]. Subsequently *L. pomona* was described as the most prevalent serotype in cattle and pigs[2] and also as a very prominent serotype in the dog[6]. In addition, other serotypes also play very prominent roles in livestock, the most important of which is *L. hyos*, which has been described as the cause of leptospirosis in pigs[6], humans, cattle and dogs[34] and also in a large variety of wild animals[35 36 37]. Similarly, *L. canicola*, one of the most common serotypes in dogs, has been encountered in leptospirosis of cattle, pigs and humans all over the world[23 38 42].

In humans, Weil's disease is caused by *L. icterohaemorrhagiae* but a large variety of other sero-

types have often also been encountered. This also applies to cattle and pigs[6].

It would therefore appear that humans, cattle and pigs are susceptible to a large variety of pathogenic serotypes if not to all, the pig being the most important reservoir[6].

The list of 32 Leptospira serotypes described during 1954[18] increased to 60 during 1959[1]. In collaboration with the sub-committee on leptospirosis of the International Committee on Bacterial Nomenclature, the joint W.H.O./F.A.O. Expert Committee on Zoonosis[17] revised the classification of *Leptospira.* In this classification the genus *Leptospira* is divided into *L. biflexa* (saprophytes) and *L. interrogans* (parasites). *L. interrogans* (*L. icterohaemorrhagiae*) again is divided into 14 groups containing more than 100 serotypes and subserotypes. These are shown in table 3.

3. *Epidemiology and Epizootiology.*

In the epidemiology of leptospirosis, active carriers among cattle, pigs, dogs and wild animals, play a prominent role. A large variety of pathogenic *Leptospira* serotypes have on numerous occasions been isolated from dogs, rodents, bats, mongoose, hedgehogs, jackals and foxes, racoons, skunks and wild cats[6 35 36 43 46]. *L. pomona* alone has been recovered from 25 wild life species[6 47], while *L. icterohaemorrhagiae* has been found the most predominant serotype in rats[6]. In these animals *Leptospira* is excreted in the urine for periods as long as six months after infection[48]. Continuous excretion of Leptospira by carriers for such long periods, is the most important source of infection to livestock and man[6].

Of the domestic animals, dogs and pigs are the most important sources of infection. Although *L. canicola* and *L. icterohaemorrhagiae* are generally considered the most prevalent serotypes in dogs, *L. pomona* has on numerous occasions been encountered in carnivorous animals[6 44 45 49].

The pig is a natural host for a large variety of Leptospira serotypes. Besides *L. pomona*[2 28 50], *L. hyos*[51] and *L. icterohaemorrhagiae,* the following serotypes have been associated with the pig: *canicola, ballum, sejroe, bataviae, autumnalis, grippotyphosa, hebdomadis, pyrogenes, javanica* and *australis*[6]. Alexander, Yager and Keefe[6] produced evidence indicating that *L. canicola* has a universal distribution among pigs which also serve as natural hosts for this serotype.

L. hyos is an important serotype not only for

TABLE 3.—CLASSIFICATION OF LEPTOSPIRA INTERROGANS (1965)

Group	Serotype
1. *icterohaemorrhagiae*	*icterohaemorrhagiae* (3); *mankarso; naam* (3); *sarmin; budapest; birkini* (2); *weaveri; ndambani*
2. *javanica*	*javanica; poi; coxus; sofia; celledoni* (2).
3. *canicola*	*canicola; schueffueni; benjamin; jonsis; sumneri; malaya; kamituga; bafani; kahendo; broomi; bindjei.*
4. *ballum*	*ballum* (3).
5. *pyrogenes*	*pyrogenes; zanoni* (2); *abramis; biggis; hamptoni; alexi; robinsoni; manilae.*
6. *cynopteri*	*cynopteri; butembo.*
7. *autumnalis*	*autumnalis* (4); *bangkinang; erina cei-auriti; mooris; louisiana; sentot; orleans; djasiman* (2).
8. *australis*	*australis; lora; bangkok; muenchen; jolna; bratislava; fugis.*
9. *pomona*	*pomona* (3).
10. *grippotyphosa*	*grippotyphosa.*
11. *hebdomadis*	*hebdomadis* (2); *kambale; kremastos; worsfoldi; jules; borincana; kabura; mini* (3); *hardjo; wolffii; medanensis; sejroe* (2); *maru; saxboeking* (2); *haemolyticus; perameles; polonica.*
12. *bataviae*	*bataviae; paidjan; djatzi; kobbe; balboa.*
13. *hyos*	*hyos* (3); *atlantae; kisuba; bravo; atchefalaya.*
14. *panama*	*panama.*

The figures in parenthesis indicate the number of sub-serotypes.

cattle and pigs[51], but has been recovered from rats[34], other wild animals[35 36 37], humans, cattle and dogs[34].

Transmission occurs either by direct contact or by urine contaminated bedding, feedstuffs or water. Kenzy, Gillespie and Lee[52] described an epizootic of bovine leptospirosis where transmission occurred by means of a running stream. Leptospirosis has also been transmitted by semen of infected bulls [47 53 54 55].

The main source of infection, however, is urine. Leptospiruria occurs 3 weeks after infection[6] after which time excretion continues for an indefinite period[56 59], continuously for at least 3 months and intermittently for periods as long as 12 months [56 60 61 62]. Once established in an area, it therefore becomes extremely difficult to control the disease mainly as a result of formation of reservoirs continuously excreting the infection[6].

Leptospiruria not only results in transmission within but also between species[38 52 63]. For example, numerous cases have been reported where leptospiral abortion storms in pigs originated from dogs[39 40 64 65], while epizootics in pigs were followed by high serum agglutinin values against that specific serotype in cattle in close vicinity[66].

4. *Pathogenesis.*

Infection is followed by leptospiraemia two days to two weeks later[56 61 67], during which time clinical symptoms may be noted. Symptoms, however, are not always noticeable as leptospirosis is usually subclinical and may readily be overlooked [6 68 69 70]. Symptoms, when present, include fever (up to 106.4°F), depression, inappetence, anaemia, jaundice, haemoglobinuria, a sudden drop in milk production and interstitial nephritis[69 71]. Leptospirosis may show a close resemblance to Babesiosis.

The leptospiraemia stage is terminated by the appearance of serum agglutinins[6 56 59 61 71 72], and followed by concentration of the organism in the kidneys for which organ it has a predilection[6]. Circulating antibodies apparently have no direct effect on the persistence of *Leptospira* in the kidneys as this can persist despite the presence of serum agglutinin levels varying from 1 : 1,000 to 1 : 100,000[72]. In pregnant animals, abortion is a characteristic but not very constant feature[6 15 70 71 73 76]. In fact, the percentage of abortions is usually very low, especially in cattle [6 13 77]. Abortions usually occur during late pregnancy and two to five weeks after the leptospiraemia stage[6 52 69 70 78]. In this respect the disease resembles Brucellosis very closely. Maximum serum agglutinin values are usually evident at the time of abortion or shortly thereafter[6 15 16 43 79].

Available evidence indicates that the foetus dies off during the acute leptospiraemia stage but expulsion occurs only 2 to 5 weeks later[52]. The aborted foetus shows characteristic lesions, viz. subcutaneous oedema, hydrothorax and ascites (dark red stained fluid), subserous haemorrhages and interstitial nephritis. The placenta is oedematous with greyish yellow cotyledons[13 15 16 78]. Abortion-storms show a close relation to the serotypic infection. In pigs *L. pomona, hyos* and *canicola* are known to cause abortions but epizootics due to other serotypes cannot be excluded[6].

In non-pregnant, adult animals infection is mostly inapparent but infection in young animals shortly after birth is followed by severe disease manifestations. These include, fever, jaundice, inappetence, weakness, haemoglobinuria and even haematuria and central nervous involvement resulting in paralysis[6]. In epidemics of this nature in pigs, *L. icterohaemorrhagiae, L. pomona* and *L. hyos* have been incriminated[30 51 80]. In calves, haemoglobinuria is a characteristic symptom.

5. *Diagnosis.*

As clinical manifestations are not a reliable diagnostic feature, other methods are resorted to. These include serology (microscopical agglutination-lysis, complement fixation and haemagglutination tests), isolation (direct or in conjunction with animal inoculation) and microscopy (direct or fluorescent antibody detection)[17].

The agglutination-lysis test is most commonly used[17] but has practical as well as technical disadvantages. In the first instance a large variety of live antigens, representing all the pathogenic serotypes known, must be included in a single test. Although grouping of related serotypes has reduced the number of individual tests considerably [50 81], a large number of tests are still required. In its most recent report W.H.O.[15] recommended 34 representative serotypes for routine serological testing by diagnostic laboratories. Being relatively serotype-specific, the agglutination-lysis test is not only laborious but necessitates maintenance and regular subculturing of a large number of subcultures[15 82]. There is divergence of opinion as to the interpretation of test results. W.H.O.[1] recommended a screening test at a single dilution of 1 : 100; a positive reaction at this dilution must be considered evidence of a past infection. Similarly Babudieri and Gaspardis[3] consider titres below 1 : 100 as insignificant.

In evaluating serological results two basic ques-

tions of great importance must be considered, namely.

a. of what significance are low titres especially as most cattle do not as a rule develop clinical symptoms?
b. what relation exists between serum agglutinin values and leptospiruria?

In the evaluation of serum agglutinin values the time interval between infection and the test is of great importance. There is general agreement that serum agglutinins are not demonstrable during the early leptospiraemia stage and that titres attain their maximum at the time of abortion or shortly thereafter[6 52 71]. This is followed by a fairly rapid decline in titre, within 3 to 4 weeks of reaching the maximum values[3 15 16 71 83 84]. On the other hand significantly high titres may persist for periods longer than 12 months[68].

This decline is directly related to the serum agglutinin levels originally stimulated. For example, it has been shown[15 16 71] that cattle with abortion titres of 1:3000 and higher showed levels of 1:100 to 1:1000 when tested 3 weeks to 3 months later. During the same time interval original titres of 1:100 had declined to 1:30 and even less.

The maximum titre at the time of abortion need not be very high. Fennestad and Borg Petersen[16] showed that cows, which yielded a negative test at the time of abortion, had titres varying from 1:300 to 1:100 three weeks later, while no less than 29 per cent of tested cows which had aborted showed maximum titres of only 1:30. In fact, a large percentage of aborting cows dit not react to tests for both Brucellosis and leptospirosis even when 16 serotypes were used.

Complete absence of serological reactions and these low titres could be attributed to infections by heterologous *Leptospira* serotypes. The mere probability that these reactions could have been due to heterologous or antigenically distantly-related serotypes, emphasises the necessity for including as many as possible of the known pathogenic serotypes in a single test. The possibility that these low titres which are frequently encountered, could be non-specific in nature was convincingly disproved, as Babudieri and Gaspardis[3] provided conclusive evidence indicating that such titres were in fact specific and due to true antibodies which could have been stimulated only by a specific serotype.

These findings strongly suggest that high titres are indicative of a past infection and such reactors must be considered active renal carriers. Low titres cannot be assumed to exclude recent infection or a carrier state in view of the possible rapid decline in titre value and the chronic nature of the infection[6].

It has been proved that leptospiruria can persist even in the absence of demonstrable serum agglutinin[56]. This is of great significance and needs special consideration. It is not unjustified to postulate that in such cases leptospiruria continued long after the diagnostic serum agglutinins had completely disappeared.

Isolation of *Leptospira* in conjunction with the serological test therefore appears to be most reliable method of diagnosis[18 50 85]. Isolation is of special value in detecting urinary carriers that reveal low titres and is even more important in those animals that are serologically negative. Isolation is done either directly in Kortoff-medium or by animal inoculation in conjunction with culture in Kortoff-medium. The culture of suspected material directly into Kortoff-medium has, as a major disadvantage, namely, contamination with other bacteria which sometimes renders diagnosis very difficult if not impossible. Guinea-pig inoculation on the other hand is extremely effective and completely reliable[6 85].

Leptospira is readily isolated from the blood of an infected animal during the leptospiraemic stage and from urine during the acute leptospiruric period. Irregular excretion of *Leptospira* in the urine of chronic carriers must not be overlooked. In these cases a large number of specimens must be examined over a long period and at regular intervals.

Direct microscopical examinations have limited application[17] as it is impossible to differentiate between *L. biflexa* and *L. interrogans* by this method.

The fluorescent antibody technique as a standard method of diagnosis may have value but its usefulness is limited to the leptospiraemic stage or to cases where detection of urinary carriers[17], following serological evidence, is required.

6. *Control.*

In the control of leptospirosis the epidemiology, epizootiology and prophylactic immunization have to be considered.

Elimination of the shedder in livestock and rodents is important. Detection of livestock shedders is possible by serological and isolation methods but combatting rodent spreaders is difficult[1]. Rodent control should nevertheless be encouraged, especially in artificial insemination centres and in studs.

In view of the contradictory results obtained from chemoprophylaxis and chemotherapy[1], immunization would appear to be the most effective method of control.

Numerous immunity studies in which monovalent bacterins were used, have been conducted on experimental animals as well as in natural outbreaks in the U.S.A., U.S.S.R., Italy, Japan, Spain and also to a lesser extent in South Africa[1 64 86 96].

In general, these investigations have proved the efficacy of inactivated monovalent vaccines against homologous but not against heterologous serotypes[6 97 98]. Cross-immunity has been obtained only where the serotypes concerned were antigenically closely related[99]. In these as well as other studies[53 100 101 102] evaluation of immunity was based on serum agglutinin response, protection against artificial exposure or prevention of further clinical manifestations in natural epidemics. Although vaccination prevents abortion storms, it apparently does not prevent leptospiruria in all vaccinated animals[1 94 103]. An effective vaccine should not only render satisfactory protection against specific infection, including leptospiruria but also against the most prevalent serotypes. Polyvalent vaccines composed of two or more serotypes have been claimed successful in humans[104 105] as well as in animals[3 103 106]. The polyvalent bovine vaccine at present used in the U.S.S.R.[3 106] consists of four serotypes, viz: *L. grippotyphosa, icterohaemorrhagiae, pomona* and *canicola*.

The duration of immunity stimulated by bacterins is not known but Gillespie and Kenzy[93], York, Johnstone and Robinson[107] and Lubashenko [106] provided evidence indicating effectivity for periods as long as 12 months.

A disadvantage in the use of bacterins, is the stimulation of serum agglutinins which is directly related to the agglutinogenicity of the specific strain[103]. The titres stimulated by inactivated vaccines have been shown to range from 1:10 to 1:100 persisting for six months or longer[100 101]. This was subsequently confirmed when Garifallou[96] showed that pigs vaccinated with inactivated *L. pomona* developed titres as high as 1:200 and when artificially exposed to virulent organisms the exposure dose acted as a booster, causing an increase in titre to levels as high as 1:3200. These vaccinal titres as well as their marked increase after exposure, constitute a serious drawback, especially where control measures are dependent on serological evidence of the disease.

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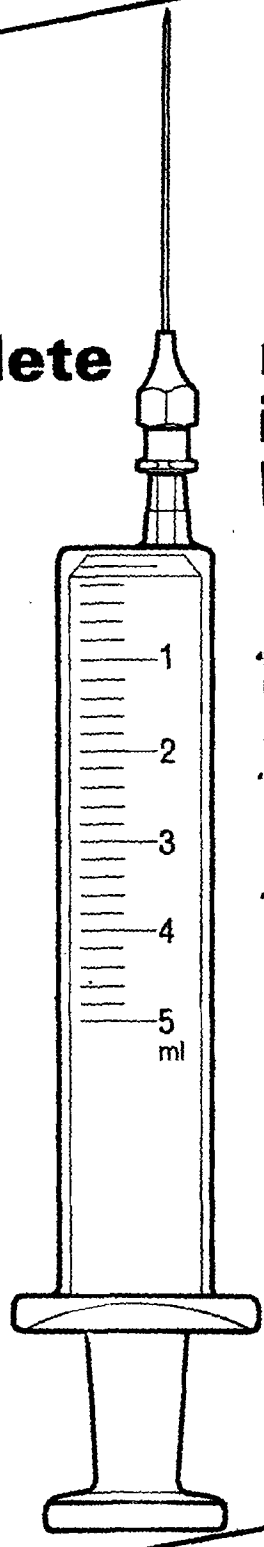

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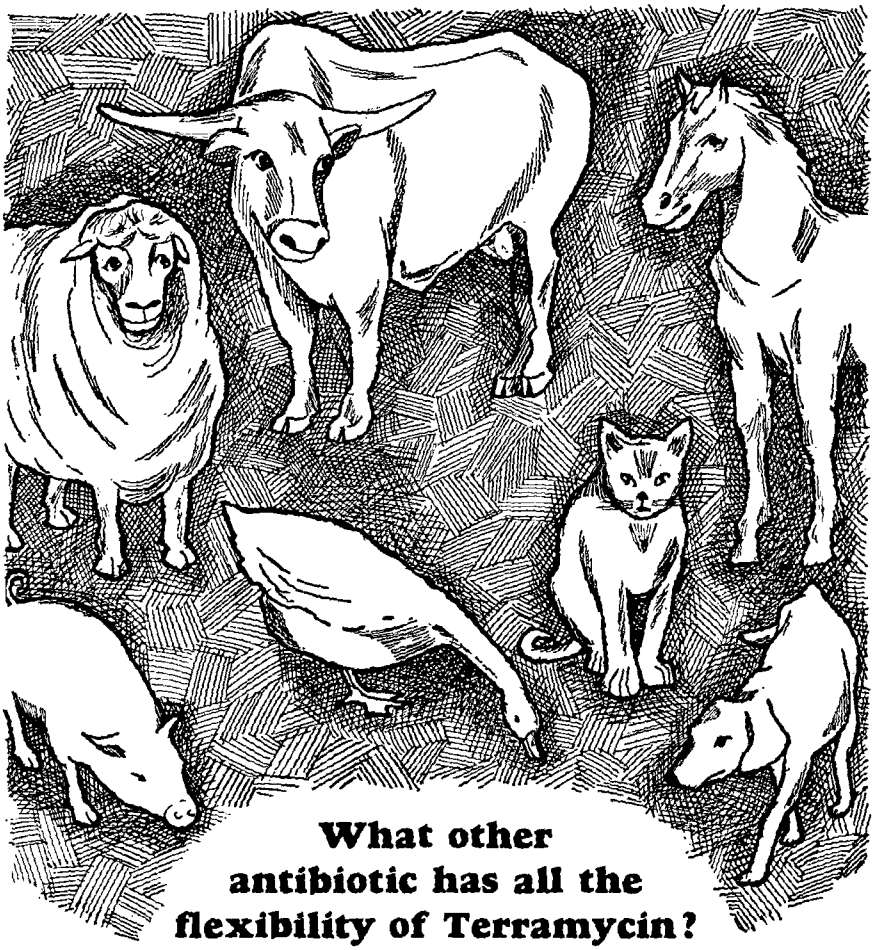

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AN OUTBREAK OF TOXOPLASMOSIS IN CHINCHILLAS IN SOUTH AFRICA

J. L. DU PLESSIS†, R. D. BIGALKE† AND T. O. GURNELL*

SUMMARY

The first outbreak of toxoplasmosis in chinchillas in the Republic of South Africa is reported. With an estimated mortality rate of 17 per cent it is also the first record of the disease in minor epizootic form in this country.

In naturally infected chinchillas the histopathology, characterised by the participation of macrophages, varied considerably depending on the course of the disease. Pneumonia, manifested by marked activation of alveolar macrophages and oedema in an acute case, and by focal perivascular and peribronchiolar accumulations of macrophages and plasma cells in subacute cases was seen consistently.

In acute cases these lesions were associated with the presence of intra- and extracellular proliferative forms of *Toxoplasma gondii* (Nicolle & Manceaux, 1908) but in subacute cases the parasites were frequently not demonstrable. In all subacute and chronic cases *Toxoplasma* cysts were found in the brain. Other common lesions were focal necrosis of the liver and myocardium, and focal gliosis and perivascular infiltration of the brain by plasma cells.

T. gondii was isolated in laboratory mice from the brain and viscera respectively of a chinchilla that had survived natural infection. Cross-immunity was found to be present between this strain, the RH strain and a ferret strain of *T. gondii*.

INTRODUCTION

In a recent report on the isolation of *T. gondii* from ferrets, the published evidence on the occurrence of toxoplasmosis in man and animals in South Africa as revealed by histopathological and serological methods was briefly reviewed[1]. The articles reviewed dealt with isolated cases of the disease and incidental discovery or serological detection of occult infection. No mention was made of extensive mortality.

The purpose of this paper is to report the first major outbreak of toxoplasmosis in South Africa, which occurred in chinchillas in the Somerset West district of the Cape Province during March, April and May, 1966.

HISTORY OF OUTBREAK

Initial mortality which occurred during March, 1966 in a chinchilla colony of some 600 animals, the majority of which were kept on the burrow system, was attributed to infection by *Pseudomonas aeruginosa* isolated in pure culture from the organs of several animals. Sulphadimidine * administered at a level of one per cent in the drinking water for 14 days usually prevented further mortality. It was found on several occasions, however, that further deaths occurred within eight days of withdrawal of this drug. Toxoplasmosis was first diagnosed histologically in mid-April in an animal that died after withdrawal of medication. In spite of continued sulphadimidine treatment an estimated total number of 102 chinchillas died from toxoplasmosis during this outbreak, which lasted until May, 1966.

Chinchillas affected with toxoplasmosis showed symptoms of lethargy, anorexia and a profuse mucoid diarrhoea; a pronounced dyspnoea was invariably present and the animals sat huddled in a corner of their cages. A muco-purulent nasal and ocular discharge was observed in the majority of animals, but at no stage were any nervous symptoms noticed.

MATERIALS AND METHODS

Chinchillas.

Formalin-preserved specimens of liver, spleen, lung, brain, kidney, adrenal, testis and myocardium from five chinchillas that had died were available for examination; four were natural cases and one had been infected artificially. In addition an apparently healthy adult male, which was said to have recovered from symptoms similar to those shown by the fatal cases was killed with ether. Portions of liver, lung, spleen, myocardium and brain were removed aseptically for transmis-

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sion purposes (*vide infra*), and representative specimens, including skeletal muscle, adrenal, thymus and mesenteric lymph nodes fixed in 10 per cent formalin. Haematoxylin-eosin and Giemsa stained sections were prepared from these tissues by standard methods.

Inoculation of mice.

Separate suspensions were prepared from half of the brain and portions of the viscera (i.e. spleen, liver, lung and myocardium) of the apparently healthy chinchilla referred to above in 5.0 ml volumes of Hanks' solution. The number of cysts in the brain suspension was calculated[1]. Using Hanks' solution as diluent, 1:10 dilutions were subsequently prepared from both brain and viscera suspensions.

Four groups of eight mice were inoculated with the diluted and undiluted suspensions of chinchilla brain and viscera respectively, each mouse receiving 0.5 ml by the intraperitoneal route. The four groups were housed in separate cages and two uninoculated mice were placed in each cage as controls.

Seven days later peritoneal fluid was aspirated from mice of the groups that had received undiluted suspensions of brain and viscera and examined for proliferative forms of *T. gondii* as described previously[1]. Approximately six weeks after injection, the mice of the two groups inoculated with diluted organ suspensions were killed and their brains removed. One half of each brain was examined for *Toxoplasma* cysts by the suspension technique[1], while the other half was fixed in formalin and five haematoxylin-eosin stained sections, cut at 10 micron intervals, prepared from each according to standard methods.

Cross-immunity tests.

The immunity of the eight mice inoculated with undiluted suspension of chinchilla viscera was challenged approximately six weeks after infection with a strain of *T. gondii* isolated from ferrets[1] and maintained in mice for six serial passages. Each mouse received approximately 50 cysts in 0.5 ml of brain suspension intraperitoneally. Eight mice were inoculated concurrently as controls.

The five mice that had survived infection with undiluted chinchilla brain suspension were challenged after six weeks with the RH strain of *T. gondii**, each mouse receiving approximately 36,000 extracellular organisms in 0.2 ml appropriately diluted peritoneal exudate by the intraperitoneal route. Eight mice were infected similarly as controls.

Results

Macroscopic pathology in chinchillas.

Significant gross lesions were seen regularly in the lungs. The lobes were all increased in size and consistency, and uniformly whitish-grey in colour. In cases where the course of the disease was somewhat prolonged, numerous white foci, one to two mm in diameter, could be seen evenly distributed throughout the lung in a reddish-grey background. A copious, slightly opaque exudate was present in the pleural cavity of some of the chinchillas but was seen less frequently in the peritoneal cavity, and then only in smaller quantities. Marked splenomegaly, with prominent Malphigian corpuscles on section, was another regular finding. Emaciation was observed in several animals later in the outbreak.

Histopathology in chinchillas.

Microscopic lesions were commonly present in the lungs, liver, myocardium, spleen and brain, and varied in severity and nature according to the duration of the disease.

In the acute case the characteristic feature in the lungs was the lining of the alveolar walls by a single row of closely packed alveolar macrophages, creating an impression of epithelization of the alveoli (Fig. 1, A & B). The alveoli were filled with a serous exudate which contained numerous free macrophages. These cells, in contrast to those lining the alveolar septa, had an abundant, frothy, sometimes vacuolated, cytoplasm (Fig. 1 D). Groups of organisms lying free in the alveolar exudate and readily distinguishable as toxoplasmas (Fig. 1 C) were demonstrable in the haematoxylin-eosin sections and somewhat more distinct in sections stained with Giemsa. Single or small numbers of organisms were present in the cytoplasm of free macrophages (Fig. 1, C & D) but could not be detected in macrophages in close apposition to alveolar walls. The majority of free macrophages contained what were presumed to be non-viable, partly disintegrated organisms or their remnants (Fig. 1 D, arrows). These are conveniently designated as "ghost" forms of *Toxoplasma.*

The lungs of subacute cases contained poorly circumscribed foci consisting of aggregations of macrophages and plasma cells in close proximity to bronchioles and blood vessels (Fig. 1 E). Distinct organisms as well as "ghost" forms were abundant in the cytoplasm of macrophages in these foci (Fig. 1 F), in which the alveolar architecture was markedly disturbed. In more chronic cases peri-

* Kindly supplied by Dr. J. Gear, South African Institute for Medical Research, Johannesburg.

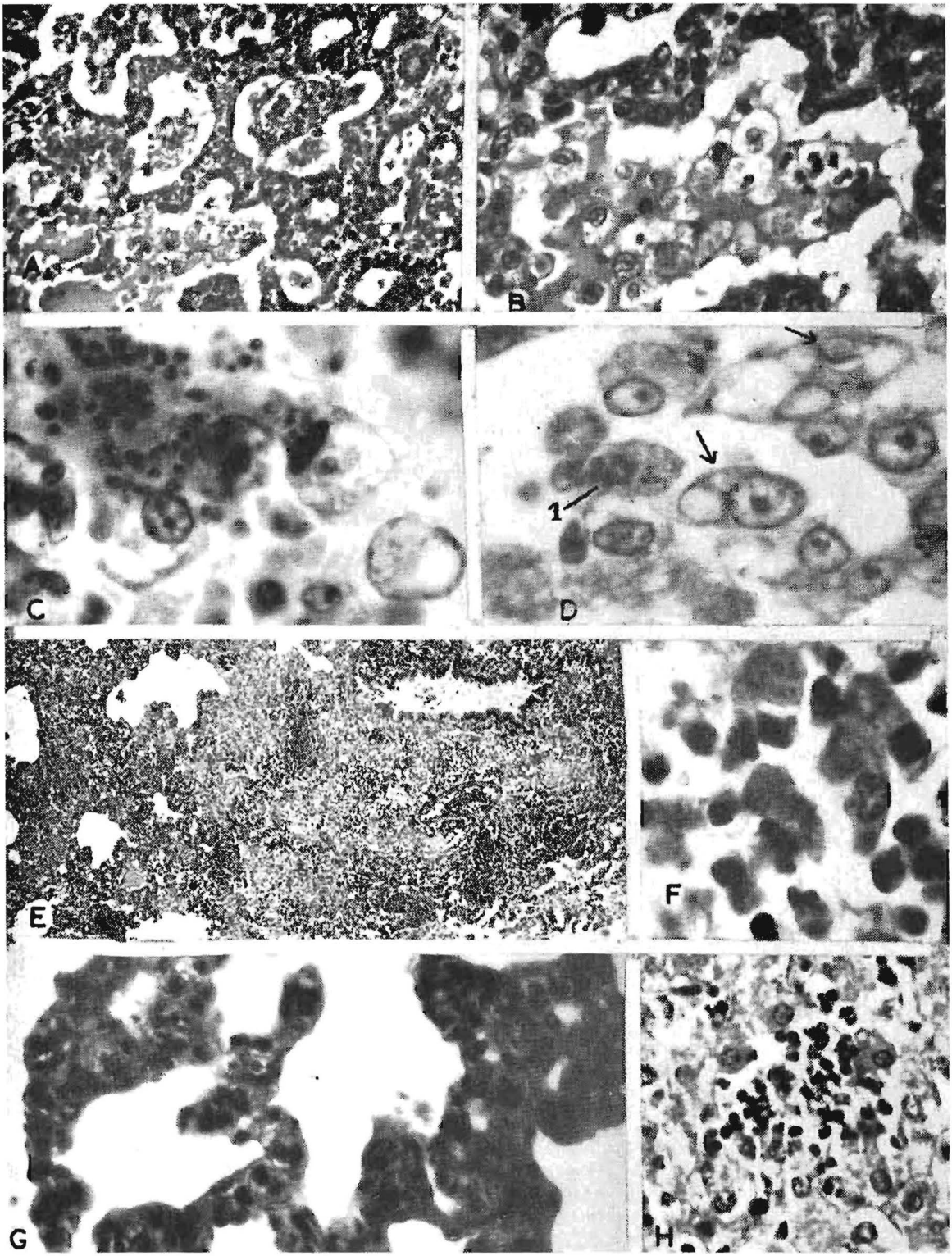

Fig. 1

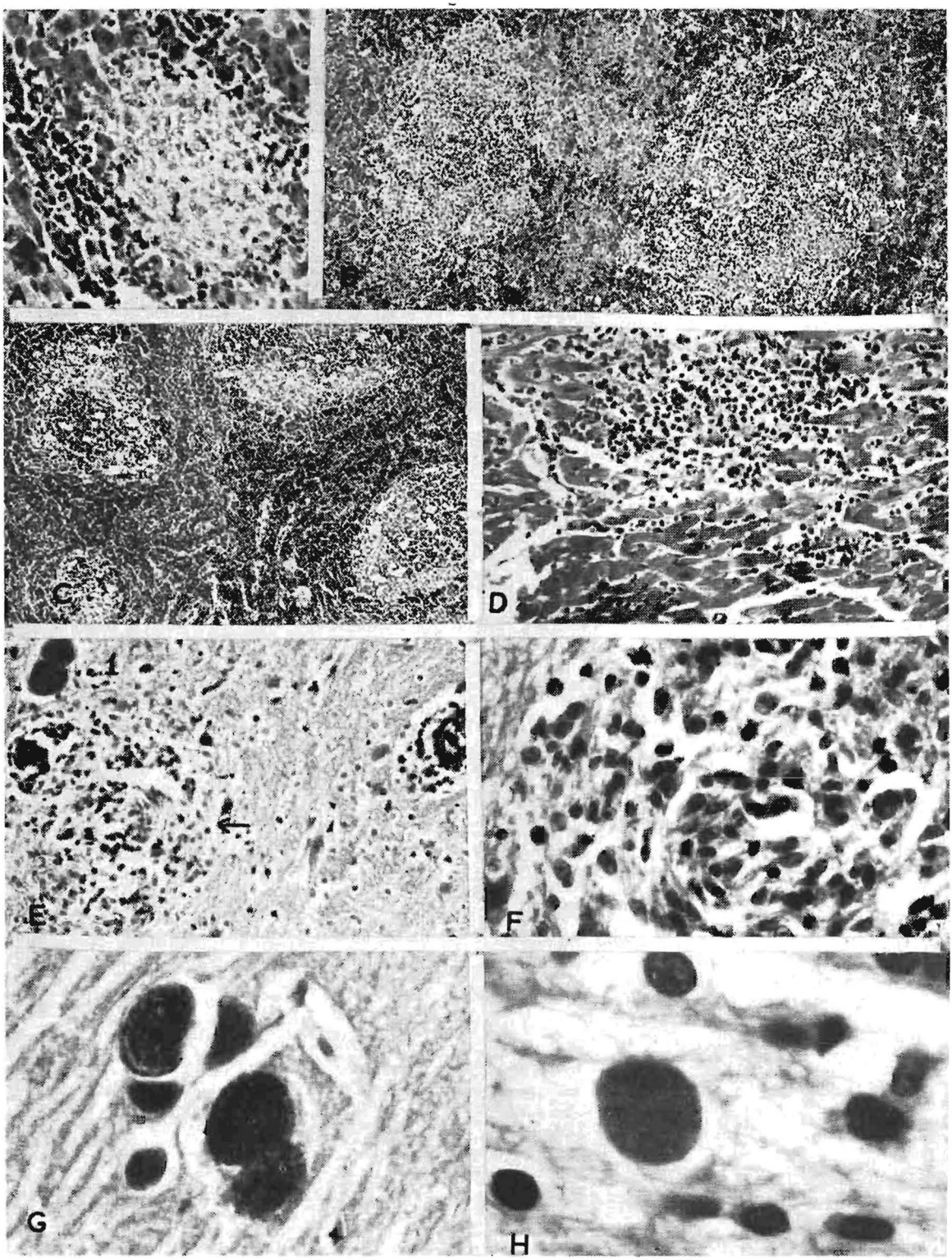

Fig. 2

vascular and peribronchiolar accumulation of predominantly plasma cells and a few macrophages, in addition to thickening of the alveolar walls by these cells, was observed throughout the lung.

In chronic cases that had apparently recovered, including the animal from which *T. gondii* was isolated, an interstitial pneumonia characterised by an increased cellularity of the alveolar walls was the only lesion suggestive of toxoplasmosis (Fig. 1 G).

There were marked differences in the severity and character of the liver lesions of the more acute cases. They varied from small disseminated foci consisting of a few necrotic hepatocytes surrounded by a number of round cells (Fig. 1 H) to extensive areas of necrosis, sometimes in confluence with one another, which affected large portions of lobuli (Fig. 2, A and B). Generally toxoplasmas were much more plentiful in the large than in the small necrotic foci, where a few "ghost" forms were detected with difficulty. The presence of large numbers of plasma cells and small lymphocytes in the sinusoids between necrotic foci, was observed in some cases (Fig. 2 B). Microgranulomata consisting of plasma cells and a few macrophages were demonstrable in the livers of chronic and recovered cases.

Disseminated foci of necrosis with infiltration by macrophages and plasma cells were consistently found in the myocardium (Fig. 2 D). Readily distinguishable organisms could not be detected.

The lymphoid follicles in the spleen were hyperplastic and prominent (Fig. 2 C) with karyorrhexis of the cellular elements in their germinal centres. "Ghost" forms of organisms were found, but with difficulty.

Lesions attributed to toxoplasmosis were found in the brains of the five cases in which this organ was available. In subacute and recovered cases large numbers of cysts of *T. gondii* were found (Fig. 2, E and G). Foci of gliosis and perivascular infiltration of plasma cells were also frequently seen (Fig. 2, E and F). The cysts and gliomata had no specific topographical distribution. Groups of cysts, unaccompanied by any sign of host-reaction, were often encountered (Fig. 2 G).

The only other site where *Toxoplasma* cysts were detected was the adrenal medulla of the recovered case used for isolation purposes (Fig. 2 H). In addition, a few microgranulomata consisting of plasma cells were present in this organ. No lesions, *Toxoplasma* cysts or phagocytised organisms were found in the skeletal muscle, thymus and mesenteric lymph nodes of this animal.

Inoculated mice.

It was calculated that the brain of the chinchilla used as donor contained approximately 11,500 *Toxoplasma* cysts and that the mice into which 0.5 ml of undiluted brain suspension had been injected, had received approximately 525 cysts each. No cysts were found in the viscera suspension.

Seven days after infection proliferative forms of *T. gondii* were found in small numbers in the peritoneal fluid of the mice of the two groups inoculated with undiluted suspensions of chinchilla brain and viscera. Six weeks after infection *Toxoplasma* cysts were demonstrated by direct microscopical examination in six of the eight mice into which the diluted viscera suspension had been injected. No cysts were found in the four uninoculated control mice assigned to these two groups.

Histological examination of serial sections of the brains of the eight mice inoculated with diluted viscera suspensions revealed *Toxoplasma* cysts in three, accompanied by perivascular round cell infiltration and focal gliosis in one case only. In one mouse these changes were detected in the absence of cysts. The brains of the eight mice inoculated with diluted brain suspensions all contained *Toxoplasma* cysts. In six of the eight, either microgranulomata or perivascular infiltrations, or both, were present.

Cross-immunity tests.

The eight mice inoculated with the undiluted suspension of chinchilla viscera and challenged with a ferret strain of *T. gondii* were all alive six weeks later, whereas only three of the eight controls survived infection for that period (Table).

In the case of the five mice infected with undiluted chinchilla brain suspension, two died 13 and one 28 days after being challenged with the RH strain. The other two survived until they were killed six weeks after the challenging infection. The eight control mice all died within a week of infection. (See Table next page).

Discussion

In the outbreak of toxoplasmosis recorded here, the initial histological diagnosis was based on the typical pneumonia, lesions of focal necrosis in the liver and myocardium and accumulations of large numbers of organisms indistinguishable from *T. gondii*. This was substantiated by the isolation of the parasite in laboratory mice and by showing that a cross-immunity existed between this, the RH and a ferret strain of *T. gondii*. Hence all the requirements which have been recommended for an unequivocal diagnosis of *T. gondii* infec-

TABLE.—CROSS-IMMUNITY TESTS ON 2 GROUPS OF MICE INOCULATED SIX WEEKS PREVIOUSLY WITH CHINCHILLA ORGANS

Challenging strain	Original inoculum	No. of mice	Survivors after	
			1 week (%)	6 weeks (%)
Ferret	Chinchilla viscera	8	8 (100)	8 (100)
	Uninoculated controls	8	8 (100)	3 (37.5)
RH	Chinchilla brain	5	5 (100)	2 (40)
	Uninoculated controls	8	0 (0)	0 (0)

tion have been fulfilled[2 3]. It is interesting to note that although the mice challenged with the RH strain enjoyed a significant degree of protection as compared to the controls, some of them eventually did succumb to the rather severe challenge. Although no organisms or cysts could be demonstrated in the viscera other than the adrenal of the chinchilla from which the strain was isolated, mice inoculated with a suspension of certain viscera became infected, indicating that parasites must have been present.

Hitherto all the recorded cases of toxoplasmosis in man and animals in South Africa have been of sporadic nature. This epizootic, in which an estimated 17 per cent of a chinchilla colony consisting of some 600 animals died, represents the first major outbreak of the disease in this country. Chinchillas are apparently highly susceptible to toxoplasmosis as suggested by McAllister[4] who recorded 44 deaths out of a colony of 55 animals from an acute form of the disease in Canada. He also cites reports on four other colonies in which mortality rates due to toxoplasmosis were in the vicinity of 50 per cent.

In this investigation the evidence obtained from histopathological studies of the lungs in particular, confirmed McAllister's observations that toxoplasmosis often runs an acute course in chinchillas. However, subacute and chronic cases were also encountered. Large numbers of proliferative forms of *T. gondii* were demonstrable in histological preparations of the acute case thus facilitating a rapid diagnosis. In subacute and chronic cases, however, organisms were rare and often only the atypical "ghost" forms were found in phagocytes. In such cases the pathologist must rely solely on the histopathological picture and the lesions described in this article may be of value in this respect. A useful aid in subacute and chronic cases is the fact that cysts usually can be demonstrated in the brain. The presence of necrotic foci in the liver, in the absence of identifiable toxoplasmas, has limited diagnostic value in chinchillas, as focal necrotic hepatitis is a common finding in listeriosis[5] as well as in *Pasteurella pseudotuberculosis*[6 7] and *Pseudomonas aeruginosa* infections[8]. Distinctive features that could be of assistance in the differential diagnosis, are the presence of large numbers of Gram-positive bacteria in listeriosis, the predominantly polymorphonuclear cellular reaction in *P. aeruginosa* infections, and the presence of macrophages and plasma cells in toxoplasmosis. The reticulo-endotheliosis seen in the liver of some cases is not specific for toxoplasmosis, as it has also been encountered in the diseases mentioned and in salmonellosis[8].

The epizootiology of the outbreak of toxoplasmosis is relevant and justifies some speculation. The chinchilla colony was established in May 1964 and the majority of the animals are housed in sheds under the burrow-system of management. Theoretically, opportunities for the introduction of infection from outside do exist; field mice, for instance, have been seen in the sheds on a few occasions. The chinchillas themselves, however, present a much more likely source of infection. As in other animal species, a carrier state develops in ostensibly recovered animals and these may be responsible for the production of new cases, e.g. by congenital transmission, or in some unknown acquired manner. Once acute cases occur, it is not inconceivable that under suitable conditions, as in a colony of this type where the animals are separated from one another by a single wire-mesh partition only, droplet transmission could take place on a wide scale resulting in major outbreaks of the disease. This possibility is not so far-fetched if one recalls the histopathological picture of intra- and extracellular organisms lying free in the exudate of the alveoli of the lungs. The moist and cool climate of the western Cape would favour

this type of transmission. In retrospect, it is impossible to determine when mortality due to *P. aeruginosa* infection stopped and that due to toxoplasmosis started. Concurrent infection with the former pathogen may have contributed towards the outbreak of toxoplasmosis by reducing the resistance of the animals, or vice versa.

Another interesting aspect was the probable influence of sulphadimidine therapy on the course of the outbreak. The inhibitory effect of sulphonamides on the proliferation of *T. gondii* is well-known[2]. From the history it is evident that no deaths occured whilst the drug was being given. On three different occasions, however, fatal cases reappeared within eight days of cessation of treatment. Obviously the treatment prolonged the course of the disease and must have been responsible for at least some of the less acute cases, thus influencing the histopathological picture.

The chinchilla industry has made rapid strides during the past few years in South Africa. The number of animals is conservatively estimated at 40,000 of which *15,000* are breeding females. The explosive nature of this outbreak of toxoplasmosis accentuates the marked susceptibility of chinchillas to the disease and draws attention to the role that frequent handling of these fur-bearing animals by human beings may play in transmitting this important zoonosis.

ACKNOWLEDGEMENTS

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FIG. 1

Fig. 1, A–G, lung.

A. Lining of alveoli by activated alveolar macrophages and filling of alveoli by free macrophages and oedema. H & E, X200.
B. Higher magnification of A. H & E, X480.
C. Free and intracellular *Toxoplasma* organisms in alveolus. Giemsa, X1200.
D. Clump of organisms (1) and "ghost" forms (arrows) in free macrophages. Giemsa, X1200.
E. Subacute focal pneumonia. H & E, X75.
F. Higher magnification of parasitized macrophages in E. H & E, X1200.
G. Thickening of alveolar walls in interstitial pneumonia of chronic (recovered) case. H & E, X480.
H. Small necrotic focus in liver. H & E, X480.

FIG. 2

A. Medium-sized necrotic focus surrounded by plasma cells in liver. H & E, X200.
B. Extensive focal necrosis of liver, with marked reticulo-endotheliosis in sinusoids between foci. H & E, X75.
C. Hyperplasia of lymphoid follicles in spleen. H & E, X75.
D. Focal necrosis with round cell infiltration in myocardium. H & E, X200.
E. *Toxoplasma* cysts (1), glioma (arrow) and perivascular infiltration by plasma cells (upper right) in brain. H & E, X200.
F. Higher magnification of focal gliosis in E. H & E, X480.
G. Several *Toxoplasma* cysts in brain unaccompanied by host reaction. H & E, X480.
H. *Toxoplasma* cyst in adrenal medulla. H & E, X480.

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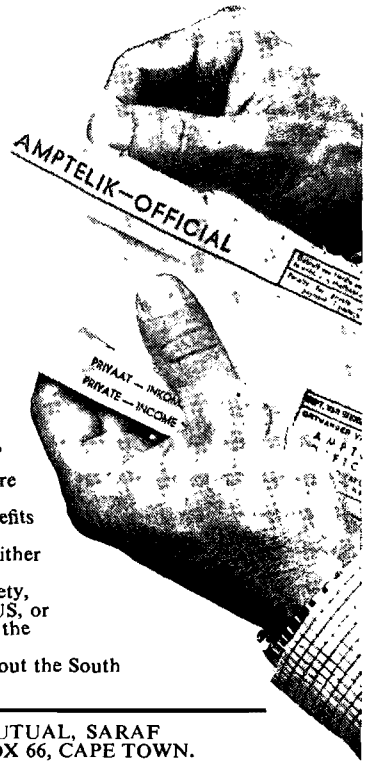

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DIE WENSLIKHEID VAN VRUGBAARHEIDSERTIFISERING BY RAMME EN DIE VEEARTSENYKUNDIGE BENADERING DAARVAN*

W. DU T. MALAN

Vrede O.V.S.

OPSOMMING:

Vrugbaarheidstoetsing en -sertifisering van skaapramme in Suid-Afrika is 'n noodsaaklike diens wat deur die veterinêre professie aangebied word. Die wetenskaplike waarde daarvan word egter nie ten volle begryp en benut deur die skaapboer nie, en daarom behoort die veeartsberoep meer aandag daaraan te wy.

INLEIDING:

Hoë wol- en vleisproduksie én goeie vrugbaarheid is die twee kardinale kwaliteite vir die ideale wol- of vleisram. Om hierdie twee funksies na wense te kan verwesenlik, moet die ram die genetiese potensiaal inherent besit, en dit moet ondersteun word deur goeie voeding, goeie beheer en behandeling. Fundamenteel is elke skaapboer 'n produsent en is die hoogste doeltreffendheid van sy teeldiere van absolute belang. Alleen deur verantwoordelike teling kan die produsent tred hou met die hoë vereistes wat gestel word in die voorsiening van die voedings- en kledingsbehoeftes van die mens.

GESKIEDENIS:

In 1657, slegs vyf jaar na die volksplanting aan die Kaap, het Kommissaris van Goens tien skape van die woltipe ingevoer, waaronder ook twee ramme was.

Die eerste doelbewuste invoer van merinoramme na Suid-Afrika het in 1789 geskied deur die toedoen van Kolonel R. J. Gordon, militêre bevelvoerder van die Kompanjie aan die Kaap. Uit hierdie skape het ook die wolbedryf in Australië sy oorsprong gehad. Van hulle vrugbaarheid is egter niks spesifieks te vind nie, behalwe dat die vinnige getalsvermeerdering genoegsame bewys was dat hulle wel vrugbaar was.

Vrugbaarheidstoetsing en -sertifisering van ramme, soos tans bekend, is egter van resente datum. Dit word algemeen aanvaar dat hierdie tipe werk eers op sy vroegste ongeveer 22 jaar gelede begin is.

BETROKKE KOMPONENTE:

1. *Die Ramkoper*: By ramkopers het toetsing en sertifisering van ramme vir vrugbaarheid beslis inslag begin vind en die koper dring vandag almeer daarop aan. Ter stawing van bogenoemde feit kan genoem word dat in September 1965 'n resolusie aanvaar is op die S.A.N.W.K.V.-kongres, dat vrugbaarheidstoetse en -sertifisering 'n voorvereiste moet wees by die verkoop van 'n ram *én* dat die tegniese en praktiese probleme wat daarmee gepaard gaan, ondersoek moet word.

   Die moderne ramkoper vereis hierdie sertifisering veral ten opsigte van twee aspekte:
   (1) omdat hy geslagsiektes in sy kudde graag wil voorkom en
   (2) omdat die produsent ook 'n waarborg wil hê dat geld, belê in 'n ram, die grootste moontlike dividend sal afwerp.

   Om hierdie redes beskou georganiseerde landbou dit as baie wenslik om ramme te laat toets en sertifiseer, nie net vir telingdoeleindes alleen nie, maar ook met oog op die beheer en voorkoming van aansteeklike geslagsiektes.

   Hierdie produsentegroep voel hom genoodsaak om 'n beroep op veeartsenykundige professie te doen om nie net voorbehoedend met entstowwe op te tree nie, maar om ook waarborgend op te tree met betrekking tot hoë fertiliteit.

2. *Die Ramteler* as tweede komponent is die teelrigtingbepaler; dit word van hom verwag om toe te sien dat die teeldier voldoen aan die doeltreffendste telingsvereistes. Dit is 'n taak wat deur hierdie groep produsente tot 'n groot mate vervul en vervolmaak is.

   Geslagsiektes het egter ook op die terrein van die teler te voorskyn getree en alreeds groot afmetings aangeneem. Statistiese opnames van die omvang van hierdie siektes onder skape is tot nog toe nie onderneem nie. Die veeartsenykundige professie behoort dit as 'n uitdaging en taak te aanvaar om hierdie informasie in te win, die afmetings

*Voordrag gelewer op die 61ste Kongres van die S.A.V.M.V. September 1966.

daarvan te bepaal en die bekamping van genoemde siektes meer positief te benader en langs dié weg 'n positiewe bydrae te lewer aan 'n bedryf soos veral die wolbedryf, wat, naas goud, die meeste valuta vir ons land verdien.

Anders as by die ramkoper is die stoetteler vanweë wanbegrip nie baie entoesiasties met betrekking tot sertifisering van ramme nie. Hierdie groep produsente is baie sensitiewe besigheidsmense, en hulle is uiters versigtig wanneer daar gepraat word van vrugbaarheidstoetse of die gebruik van entstowwe soos Rev. I. wat enigsins aanleiding kan gee tot suspisie dat daar moontlik geslagsiektes of onvrugbaarheidsprobleme onder hulle kuddes kan wees.

Die vrees vir ekonomiese verliese is die kern van hierdie verkeerde benadering en die noodsaaklikheid van 'n regstelling deur die professie moet beklemtoon word.

3. *Veterinêre Benadering*: Die veearts, as derde komponent, is die wetenskaplike vertroueling, en hy neem uit die aard van die hele aangeleentheid 'n uiters vername plek in, wat hom in staat stel om die saak baie te bevorder.
Die vertroue in ons as wetenskaplikes word geensins betwyfel deur sowel koper as teler nie, máár die feit dat die veearts nie bereid is om sertifisering van vrugbaarheid vir 'n lang periode te waarborg nie, skep die indruk dat die hele aangeleentheid nie veel waarde inhou nie.

Omdat sertifisering van ramme nog nie 'n lang geskiedenis het nie, is die betrokke partye nog nie ten volle vertroud met die doel wat beoog word nie, en word die waarde nie altyd verstaan en reg geïnterpreteer nie.

Die ramkoper word al meer bewus van die gevare wat dit vir hom inhou om by die aankoop van ramme net oppervlakkig na die geslagsorgane van die ram te kyk en dit te bevoel. Dit het 'n toenemende behoefte geskep dat die teler 'n vrugbaarheidsertifikaat as waarborg vir sy produk moet gee. So 'n sertifikaat verminder die risiko van die koper met betrekking tot sy belegging.

Die aanvraag van so 'n veterinêre sertifikaat het eintlik begin met die styging van wolpryse in die vroeë vyftigerjare. Dit het meegebring dat die aanvraag na goeie ramme gestyg het en die rampryse dienooreenkomstig die lug in geskiet het. Hierdie taamlike skerp styging van rampryse het die versigtige koper nog meer versigtig gemaak en het 'n natuurlike begeerte by hierdie produsente laat ontstaan om vooraf sekerheid oor die potensiaal van die ram te hê, des te meer omdat 'n ram wat klinies normaal voorkom, dikwels teen 'n hoë prys aangekoop word en dit eers later ontdek word dat sy reproduksievermoë onbevredigend is as gevolg van steriliteit of lae vrugbaarheid, of dat hy 'n besmetlike siekte in die ooikudde ingedra het. Genoemde feite het meegebring dat die veterinêr se hulp ingeroep is en so het ook die eise vir sertifisering van ramme geleidelik by die ramkoper toegeneem.

Die vraag ontstaan nou wat die veeartsenykundige benadering ten opsigte van hierdie aangeleentheid moet wees?

In die praktyk kom dit hierop neer, dat die veearts ingeroep word om vrugbaarheidstoetse te doen en sertifikate uit te reik vir die stoetteler wat ramme vir verkoop aanbied. Volgens gegewens wat spreker ingewin het, is die persentasie stoettelers wat van hierdie diens gebruik maak, minimaal.

Op georganiseerde ramvendusies word toetsing deur sommige telersverenigings aangemoedig, en doen sommige telers ook die moeite om teen aansienlike koste al hulle ramme te laat toets en sertifiseer. Volgens hierdie telers hou dit vir hulle geen meetbare ekonomiese waarde in nie, want, so beweer dié telers, die ramkopers is nie in die minste geïnteresseerd in dié sertifikaat nie. Getoetste diere geniet dus eintlik geen voorrang nie en dit bring mee dat die teler ook sy belang verloor om in die toekoms te laat toets.

Tweedens word die veearts ook ingeroep deur die ramkoper om sy aangekoopte ramme te toets, asook sy ander ramme wat vir paring gereed gemaak word.

In bogenoemde gevalle word sertifisering gewoonlik nie gedoen nie, behalwe as die aangekoopte ramme steriel of hul vrugbaarheid laag bevind word. Hier lê die knoop wat so dikwels deurgehaak moet word. So 'n sertifisering van dié nuut aangekoopte ramme het wettige implikasies, waarin nie net die teler en die koper betrek word nie, maar ook die veearts.

DIE SERTIFIKAAT:

Dit moet onthou word, dat as 'n veterinêr 'n sertifikaat van gesondheid en vrugbaarheid onderteken, hy dit moet doen met die grootste versigtigheid en nougesetheid vir die behoud van die goeie naam van die professie. So 'n sertifikaat is van dokumentêre aard en mag selfs as bewysstuk in die hof gebruik word. Die onfeilbaarheid van hierdie sertifikaat moet altyd bo alle twyfel bewys

kan word. So 'n dokument is nie vir die openbare vertoon bedoel nie en die betrokke veearts behoort dit te meld aan die eienaar tydens die uitreiking daarvan. Die etiese kode kom anders in gedrang.

Sommige aspekte in die sertifikaat wat deur ons vereniging aanvaar is, is vanselfsprekend, andere moet egter meer toegelig en benadruk word.

1. *Identifikasie*: Die noodsaaklikheid van 'n goeie identifikasiesisteem is van groot belang. Indien 'n goeie stelsel nie gebruik word nie, mag sertifisering aan misbruik en bedrog blootgestel word. 'n Stelsel van horingbranding en/of tattoeëring blyk by die skaap van uiterste belang te wees, en behoort gepropageer te word.

2. *Teelgeskiedenis van kudde van oorsprong*: Die inligting van die eienaar in hierdie verband sal van groot waarde wees vir die veearts, en hieruit kan hy sekere afleidings maak, veral wanneer subnormale vrugbaarheid bepaal moet word. Vroeë vasstelling van enige teelprobleme is van onskatbare waarde in enige teelkudde.

3. *Teelgeskiedenis van die Ram*: Dit is wenslik om te weet of 'n ram al geteel het al dan nie, want as 'n ram al geteel het, het hy bewys gelewer van sy teelvermoë. Daarteenoor bestaan die moontlikheid dat hy blootgestel kon gewees het aan geslagsiektes of ander besmettings wat sy vrugbaarheid kon benadeel het.

4. *Kliniese Ondersoek*: Die doel hiervan is:
   (1) om te verseker dat die ram in goeie gesondheid verkeer.
   (2) om aangebore gebreke te ontdek, veral dié gebreke wat sy voortplantingsvermoë kan belemmer, en
   (3) om te bepaal of daar enige waarneembare erflike gebreke is, wat onwenslik is vir teeldoeleindes, al is hy vrugbaar.

5. *Ondersoek van Semen*: Dit is die belangrikste ondersoek, wat dan veral twee dinge bepaal:
   (i) of die saad van die gewensde kwaliteit en kwantiteit is al dan nie, en
   (ii) of dit vry is van besmettings wat met paring oordraagbaar is, bv. brucellose en corynebakteriose.

6. *Inentings*: Enige naturlike aanval van besmetlike siektes het gewoonlik 'n nadelige invloed op die vrugbaarheid van 'n ram. Die eerste inenting teen so 'n siekte kan die kwaliteit van 'n ram se saad tydelik laat afneem.

7. *Transportering en Voedingswisseling*: Die vervoer van ramme oor lang afstande het dikwels 'n nadelige invloed op die kwaliteit van 'n ram se saad. 'n Vreemde omgewing en voedingsveranderings kan saadkwaliteit beinvloed en dit moet in aanmerking geneem word by die bepaling van vrugbaarheid.

   Alleen as al die faktore in aanmerking geneem is en die toetse gedoen is, kan 'n sertifikaat uitgereik word.

WETTIGE IMPLIKASIES:

Die argument word dikwels geopper dat 'n vrugbaarheidsertifikaat ten opsigte van die ram, uitgereik deur die veearts, die teler onthef van sy verantwoordelikheid en die onus daarmee op die veearts geplaas word.

Die wettige implikasies i.v.m. latente en aanverwante defekte of siektes by vee word duidelik uiteengesit in Wille en Millen se „Mercantile Law" onder die indeling „Purchase and Sale Warranties." Daar bestaan twee soorte van waarborge wat in geval van verkope by vee deur 'n stoetteler gegee kan word, nl:

(a) 'n uitdruklike waarborg („express warranty") en
(b) 'n stilswyende waarborg („implied warranty").

Ingeval van (a) sal 'n stoet of enige ander verkoper sy waarborg moet nakom. Ingeval van (b) sal dit algemeen aanvaar word dat 'n leek nie 'n stilswyende waarborg kan gee nie, maar in die geval van 'n stoet of geregistreerde teler sal dit beskou word dat hy 'n deskundige is en dat so 'n waarborg derhalwe aanwesig sou wees. Die onus rus dus op die teler en nie op die veearts nie. Die veearts bedien alleen die teler en die koper met *bona fide* advies en leiding.

Waar 'n dispuut mag ontstaan ten opsigte van die vrugbaarheid van 'n betrokke ram, kan die saak ook onderling of arbitrêr tussen koper en verkoper opgelos word en is dit nie vir die teler altyd nodig om summier die ram terug te neem ten einde sy goeie naam te beskerm ten koste van die betrokke veearts nie.

GEVOLGTREKKING:

Dit kan nie oorbeklemtoon word nie, dat die uitgangspunt altyd die vasstelling, bestryding en voorkoming van geslagsiektes moet wees, en dat dit primêr die funksie van die veearts is.

Die noodsaaklikheid van toetsing en sertifisering word deur produsente in die wol en vleisnywerheid al meer aangevra en daarom moet die telers oorreed word om hulle uiterste bes te doen om aan hierdie vereistes te voldoen.

Die Suid-Afrikaanse skaapstapel beloop volgens jongste statistiek 43 miljoen waarvan, 38.5 miljoen merinoskape is met 'n kuddesamestelling van nagenoeg:— ooie 39%; hamels 30%; jongskape 17% en lammers 14%.

Die jaarlikse byvoeging van merinoramme tot die bestaande getal is 37,000. Indien daar nie gewaak word teen aansteeklike geslagsiektes deur middel van toetsing en sertifisering en nie voorbehoedend opgetree word nie, kan dit net nadelige gevolge vir die wol- en vleisbedryf in Suid-Afrika meebring.

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FILAROIDES OSLERI (COBBOLD, 1879) INFESTATION IN THE DOG. †

J. E. DORRINGTON‡

The literature on *F. osleri* is reviewed showing it is of world-wide distribution and that infestation has been reported in most breeds of dogs. It is also indicated how some breeds in South Africa have become infested.

The symptomatology is described. It is pointed out that infested dogs may show no symptoms whatsoever. The age at which clinical symptoms are first observed may vary from two and a half months to well over two years with an average of four to six months.

Pathogenicity is shown to depend on the number of nodules present, their location in the trachea or bronchi, the age of the dog and the degree of infestation with other helminths. Emaciation and death follow prolonged interference with respiration.

All breeds of dogs are equally susceptible, regardless of age or sex. The apparent greater susceptibility of new-born pups is shown to be due to the nursing habits of the bitch.

The morphology of the parasite as well as the macro- and microscopic appearance of the lesions are described and illustrated.

Diagnostic methods and the differential diagnosis are discussed. The only method advocated is direct intratracheal examination using a distally illuminated bronchoscope.

Transmission experiments clearly indicate that the life cycle is direct without the necessity for an intermediate arthropod vector. Intra-uterine transmission does not occur. It is shown that, under natural conditions, infestation is acquired at a very early age. Cross infestation between individual puppies in a litter does not occur.

Dosing experiments demonstrate that larval migration occurs chiefly via the mesenteric lymphatic system and also via the portal. When dosed, the larvae pass rapidly to the small intestine and penetrate to the mesenteric lymph nodes where they are to be found in their highest concentration within twelve hours. Within twenty four hours, practically all the larvae have left the lymph nodes and are found in and around the terminal bronchioles. From here they migrate to the trachea at its bifurcation which is their predilection site. Six weeks later macroscopic lesions are visible. The morphology of the migrating larvae in the various sites is described and illustrated. Histopathological changes in the lymph nodes, liver and lungs are described, the absence of eosinophiles being especially noteworthy.

A surgical technique for the removal of the nodules is described. This involves the use of an illuminated bronchoscope and a 50 cm cupped biopsy forceps together with the aid of a good suction pump to maintain a clear operative field.

Various chemotherapeutic agents are evaluated. A completely successful treatment consists of the intravenous injection of 1 ml. thiacetarsamide (Caparsolate sodium, Abbotts) per 5 kg body weight for 21 consecutive days, followed six weeks later by surgical removal of any remaining papillomata. The operation may have to be repeated some weeks later. This method results in permanent cure.

Prophylactic measures are discussed.

In the light of the evidence advanced, it is suggested that there is a need for the reclassification of *F. osleri*.

†Summary of D.V.Sc. Thesis, University of Pretoria, 1966.

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THEILERIOSIS IN RHODESIA:

I. A STUDY OF DIAGNOSTIC SPECIMENS OVER TWO SEASONS

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SUMMARY

Theileriosis of cattle is a scheduled disease in Rhodesia. Study of fifty-five outbreaks detected during two seasons (1964 to 1966) shows that *Theileria lawrencei* is the main cause. *Theileria mutans* is also present. *Theileria parva* was eradicated in 1954 and the present work confirms that it is no longer present in Rhodesia.

Mortality is generally low (in 23 outbreaks only single deaths occurred) although occasionally it is high (in a well documented outbreak 35 out of 100 head of cattle died). The disease is seasonal in nature, breaking out mostly during December to April which coincides with the flush of adult *Rhipicephalus appendiculatus* ticks.

In diagnostic specimens, parasitosis varied from less than one to 400 schizonts per 1000 lymphocytes with a mean of less than 46. Ten per cent of the field specimens contained microschizonts. Parasitaemia was very low and varied from less than one to 80 endoglobular parasites per 1000 erythrocytes with a mean of less than 6. Buffalo were associated with 9 of the 55 outbreaks and circumstantial evidence suggests that cattle to cattle transmission via the tick is the rule in the field.

In addition to the studies of field outbreaks 39 *Theileria* transmissions were made by adult *R. appendiculatus* ticks into clean high-grade dairy cattle (mostly splenectomised) in the laboratory. The theileriosis so produced was comparable to the cases seen in the field. Only 6 of the 39 cattle died of theileriosis while, with the exception of two, the remaining cases all recovered and became carriers. Clinical symptoms varied from a mild fever with swollen lymph nodes to very high fever, swollen lymph nodes, severe respiratory distress, loss of condition, collapse and rapid death in 19 to 23 days following tick infestation in the four most severe cases. Parasitosis in the laboratory cattle varied from less than one to 690 schizonts per 1000 lymphocytes with a mean of less than 40. Two per cent of the laboratory specimens contained microschizonts. Parasitaemia varied depending on whether or not the cattle were splenectomised. In intact cattle parasitaemia varied from less than one to 11 endoglobular parasites per 1000 erythrocytes with a mean of less than 2. Maximum parasitaemia was reached between 19-24 days after tick infestation. By contrast, the parasitaemia in splenectomised cattle progressed for 58 to 121 days after tick infestation reaching peaks which varied from less than one to 480 endoglobular parasites per 1000 erythrocytes with a mean of less than 62. Apart from a transitory mild anaemia at the crisis of parasitaemia, the endoglobular parasites were not associated with illness, so that the clinical symptoms of theileriosis were attributed to schizont activity. An intermittent low-grade parasitaemia persisted in both splenectomised and intact cattle. In the laboratory cattle, counts of chromatin particles varied from 2 to 37 per macroschizont while the sizes varied from 1.4 to 7.8 microns. Cattle to cattle transmission via the tick, *R. appendiculatus*, was proven and practically all the strains were isolated from areas where no buffalo were present.

Rhipicephaline tick toxicosis and *Ehrlichia bovis* infection transmitted by adult *R. appendiculatus* were both found to complicate the theileriosis picture.

INTRODUCTION

Theileriosis is a scheduled disease in Rhodesia. *Theileria parva, Theileria lawrencei, Theileria bovis* and *Theileria mutans* have been identified from cattle in the past. Following Neitz's[1] lead *T. bovis* is regarded as synonymous with *T. lawrencei* and the latter term has precedence.

T. parva, the cause of East Coast fever, was first identified in Rhodesia in 1903 by Koch[2] who referred to the disease as African Coast fever. Koch's detailed description of the parasite leaves no doubt as to the precise identity. However, the parasite was not named until, in 1904, Theiler[3] proposed the term *Piroplasma parvum.* Subsequently, through reclassification by Bettencourt and colleagues in 1907, as cited by Henning[4], this

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became *Theileria parva,* the term we use today. According to Wenyon[5], Stephens and Christophers preceded Theiler[3] by proposing the name, *Piroplasma kochi,* for the parasite of "African Coast fever" in 1903, so that *Theileria kochi* may be the correct term. (The writer has not seen the original proposal by Stephens and Christophers. The practical study of malaria and other blood parasites. First edition (1903). University Press of Liverpool — and so is not able to verify Wenyon's opinion). It is likely that Gray and Robertson[6] who in 1902 investigated what they concluded to be "Texas fever or Redwater in Rhodesia" in fact preceded Koch in unwittingly observing *T. parva,* as their description suggests a mixed infection of *Babesia bigemina* and *T. parva.*

At this time East Coast fever was spreading as an epizootic throughout Rhodesia. By 1905 approximately one third of the cattle population had died from the scourge. Gradually, however, the disease was contained by vigorous veterinary measures sustained over the next fifty years until finally in 1954 the country was declared free of East Coast fever; no subsequent outbreaks have been detected.

In the 1930's Lawrence[7] investigated a disease which appeared frequently in certain areas of the Rhodesian lowveld, and was associated by farmers with the presence of buffalo. This gave rise to the name "buffalo disease". Further investigation proved the disease to be caused by a *Theileria* species which differed strikingly from *T. parva.* In contrast to the high rate of parasitosis and parasitaemia that is present in advanced cases of East Coast fever, in this new disease the *Theileria* schizonts and endoglobular forms were extremely difficult to find. It was not until over twenty years later, in 1955, when Neitz, Canham and Kluge[8] in South Africa reported a similar condition in the corridor between the Umfolozi and Hluhluwe Game Reserves that a detailed identification became possible. Neitz[9] concluded from his investigations that his "Corridor disease" was similar to Lawrence's "buffalo disease" and in honour of the earlier work by this colleague he named the protozoon *T. lawrencei.*

A theileriosis similar to "buffalo disease" was investigated by Lawrence and Orr on the farm Fortuna in the Melsetter district of Rhodesia in 1936. This was at first referred to as "Fortuna" or "specific disease", Lawrence[10]. The parasite picture was similar to that in "buffalo disease" but in this instance buffalo were not involved.

For administrative purposes these latter diseases were subsequently referred to as "Theileriosis" in order to differentiate them from "East Coast fever". In the context of the present paper I am using the term theileriosis to imply infection with any of the *Theileria* species.

T. mutans was named by Theiler[11] in 1906 and first recorded in Rhodesia in 1910 by Bevan as reported by Nobbs[12]. As elsewhere in the world, this parasite has been regarded as non-pathogenic and of little importance except in circumstances of concurrent disease, examples of which were studied by Lawrence[13].

Gradually, with the eradication of East Coast fever, theileriosis, due to *T. lawrencei,* has come more to the fore. Outbreaks of buffalo disease have been reported mostly from the lowveld ranches in Fort Victoria and Bulawayo districts where cattle frequently come into contact with veld grazed by buffalo. By no means all these contacts are followed by disease. In the higher veld such as in Salisbury, Umtali and Sinoia districts, where buffalo are less frequent, theileriosis outbreaks similar to those at Fortuna have been detected frequently now. In most of these outbreaks buffalo are definitely not present and in these circumstances it would seem that transmission via the tick must be taking place from cattle to cattle rather than from buffalo to cattle as is apparently the case in the lowveld.

Furthermore, the outbreaks in the higher veld have become very seasonal in character, commencing mostly with the summer season of rainfall, from which the term "January disease" has come into common use. Thus, in the last few years, outbreaks of theileriosis have been detected at the rate of 20 to 40 per annum, mostly under circumstances pertaining to "January disease". Mortality rates are occasionally as high as in East Coast fever but in general these newer forms of theileriosis have been readily controlled by well-established procedures, including short interval dipping, hand-dressing and quarantine (for details see Matson[14 15]). Relatively little mortality has resulted and recoveries occur.

Table I summarises the number of theileriosis outbreaks and the mortality experienced per ten year period since 1903 when the initial epizootic of East Coast fever eliminated approximately one third of the country's cattle population. The data have been compiled from the reports of the Directors of Agriculture and Veterinary Services for the years 1902 to 1965 (catalogued in the National Archives of Rhodesia, Salisbury)[16].

From a consideration of Tables 1 and 2 it is evident that theileriosis is playing a decreasing role in causing mortality in relation to the increasing cattle population. This is in spite of the increasing number of outbreaks of *T. lawrencei* in recent decades. Thus the importance of the disease appears to be diminishing.

TABLE 1.—THEILERIOSIS OUTBREAKS AND MORTALITY

Period	Outbreaks			Mortality		
	T. parva	*T. lawrencei*	Total	*T. parva*	*T. lawrencei*	Total
*1903–1905	?	?	?	19,270	0	19,270
1906–1915	101	0	101	5,010	0	5,010
1916–1925	115	0	115	6,116	0	6,116
**1926–1935	89	0	89	5,813	0	5,813
1936–1945	62	34	96	1,213	86	1,299
***1946–1955	2	170	172	47	2,451	2,498
1956–1965	0	314	314	0	2,770	2,770

*1903–1905 = period of initial epizootic of East Coast fever.
**1934–1935 = buffalo disease discovered — detailed records not available.
***1946–1947 = records incomplete.

From data in the same sources the cattle population is estimated as shown in Table 2.

TABLE 2.—CATTLE POPULATION IN RHODESIA

Year	*Approximate total*
1905	66,000
1915	850,000
1925	2,100,000
1935	2,500,000
1945	2,900,000
1955	3,100,000
1965	3,800,000

Recent research work has once more focussed attention on the disease. Brocklesby[17 18] working in East Africa, showed that under experimental conditions, continued tick passage of *T. lawrencei* in cattle, may induce it suddenly to acquire the more lethal characteristics of *T. parva*. In Rhodesia, as noted previously, cattle to cattle passage now seems to be the rule rather than the exception: if Brocklesby's findings on the lability of *T. lawrencei* are correct then the danger exists that East Coast fever may once more break forth. Brocklesby emphasised the suddeness with which the change could take place. A feature of East Coast fever in Rhodesia has been the way the disease has unexpectedly cropped up in places which have been free for years and where no contact could be traced to previous outbreaks. This peculiarity has been stressed by successive commissions of enquiry (McIlwaine *et al.*[19 20 21], Shand *et al.*[22] and Speight *et al.*[23]).

An immediate enquiry, therefore, was initiated into theileriosis in Rhodesia and a programme of study was formulated[14]. The first objective was to determine if *T. lawrencei* in Rhodesia was already behaving in the field as *T. parva* and the present paper presents the results of this study.

As a scheduled disease all suspected cases of theileriosis are investigated by Government Veterinary Officers and confirmation of diagnosis is made by examination of spleen, lymph node and blood smears. In the light of Brocklesby's work, detailed parasite counts were commenced on all the diagnostic specimens available from these sources. Simultaneously, isolations were made of *Theileria* by tick transmission from representative outbreaks and cases so produced were studied in detail in the laboratory.

MATERIALS AND METHODS

Preparation of smears for diagnosis

Blood, lymph node and spleen smears were dried in air, fixed with methyl alcohol or May-Grünwald and stained with Giemsa's stain. The specimens from the natural outbreaks were subject to the adverse circumstances prevailing in the field and were prepared by either district veterinary staff, farmers or ourselves. The specimens from the laboratory isolates were prepared under more favourable circumstances.

Isolation of Theileria

Theileria isolations for intensive study in the laboratory were made by transmission through *Rhipicephalus appendiculatus* ticks obtained from the following sources:

A Removal of recently attached infected adult ticks from sick animals.

B Feeding of laboratory reared clean nymphae on sick animals and allowing these ticks to moult through to the adult instar.

C Collecting adult infected ticks by blanket drag or by hand from infested pasture. These ticks were held in an atmosphere of approximately 75% relative humidity (over saturated sodium chloride salt solution) at room temperatures (18-24°C) until they were fed on either intact or splenectomised clean high-grade cattle of dairy breed.

D Exposing the test bovine to tick infestation in the field.

Estimation of parasitosis

The frequency with which Koch's blue bodies could be found was estimated by recording the number of observed intracellular and extracellular parasites, both macroschizonts and microschizonts, whilst 200 to 1000 lymphocytes were counted. Where parasites were frequent, the smaller number of lymphocytes was all that was required to be examined in order to obtain this estimate. Negative findings were not recorded until the specimens had been examined exhaustively and smears in which lymphocytic activity (mitotic figures) were observed were regarded with particular suspicion. The highest count made, whether for spleen, lymph node or blood, was taken as the estimate of parasitosis of each case.

Counts of the number of chromatin particles within schizonts

In a proportion of the specimens counts were made of the number of chromatin particles within each schizont. Macroschizonts and microschizonts were differentiated.

Estimation of the size of schizonts

In a proportion of the specimens the schizonts were measured by means of an ocular micrometer calibrated against a slide micrometer in a system giving a magnification of approximately 1:1000. The maximum diameter was measured followed by the diameter at right angles to this. The mean of these two measurements was taken as the estimate of size.

Estimation of parasitaemia

The frequency with which endoglobular parasites could be found, was estimated by counting the number of such organisms seen whilst examining approximately 1000 erythrocytes. In the ocular system used, five microscope fields frequently contained this number of erythrocytes. Negative findings were not recorded until at least fifty fields had been examined. The highest count recorded was taken as the estimate of parasitaemia of each case.

Results

Details of outbreaks studied

During the period October 1964 to September 1966 fifty-five outbreaks of theileriosis were detected in Rhodesia. Their distribution according to area is shown in fig. 1.

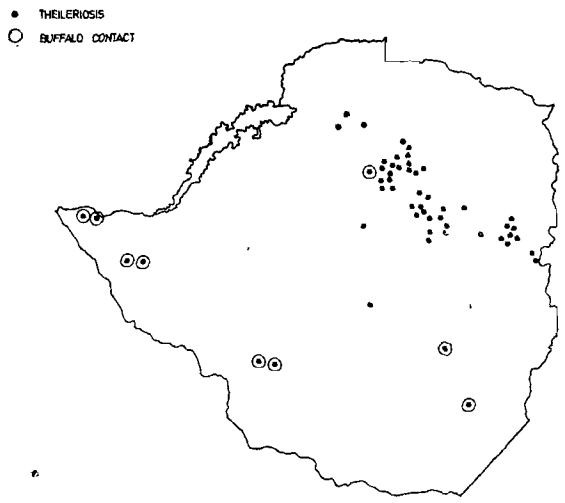

Fig. 1
Distribution of theileriosis outbreaks in Rhodesia October 1964–September 1966

Buffalo were definitely known to be present in only 9 of the 55 outbreaks of theileriosis.

Isolation of Theileria

During the period October 1964 to September 1966 thirty-nine isolates of *Theileria* were made from the districts of Sinoia, Salisbury, Umtali, Gwelo and Chipinga by tick transmission with adult *R. appendiculatus* ticks into clean, intact or splenectomised high-grade cattle of dairy breeds. Bovine 374 was infected with *Theileria* by method A, bovine 3991 by method B, bovine 414 by method D and the remaining 36 bovines by method C. In these districts positive isolations were obtained on every occasion, irrespective of whether the ticks were from theileriosis farms or even farms where no theileriosis had been detected. In seven similar attempts to isolate *Theileria* from theileriosis centres in Bulawayo and Fort Victoria districts, the tick feedings were equally successful but no transmission of pathogens was observed. The distribution of the *Theileria* isolated according to area is shown in fig. 2.

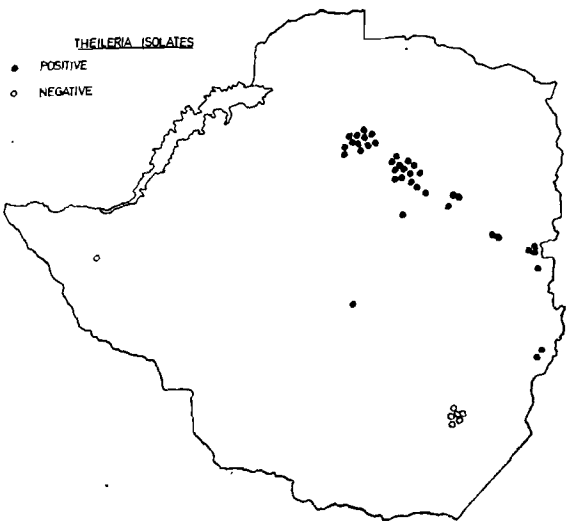

FIG. 2
Distribution of Theileria *isolates in Rhodesia October* 1964–*September* 1966

The period October 1964 to September 1966 covers two rainy seasons (December to April) and the first appearance of the theileriosis outbreaks in relation to time of year is shown in fig. 3.

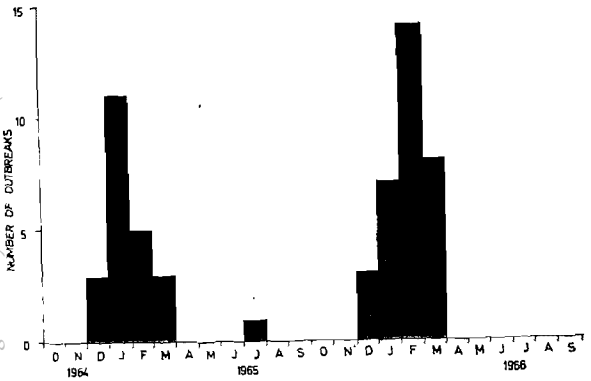

FIG. 3
Appearance of theileriosis outbreaks in Rhodesia October 1964–*September* 1966

In the laboratory we have isolated *Theileria* practically throughout the year, except in the coldest weather when the adult *R. appendiculatus* ticks attach with difficulty, provided the ticks collected were still alive and vigorous.

A total of 428 cattle deaths were reported from the 55 outbreaks of theileriosis. The total cattle population on these properties was 27,925. Diagnostic specimens were studied from 138 theileriosis cases in the field. (The discrepancy in relation to the larger number of cattle that died is accounted for by the fact that diagnostic specimens are not obtained from every animal that dies). Control measures were applied in all instances and in only 11 of the outbreaks was mortality severe (i.e. more than ten deaths). In 23 of the outbreaks only single deaths were recorded. In fig. 4 the mortality of cattle in the field outbreaks is summarised according to number of farms.

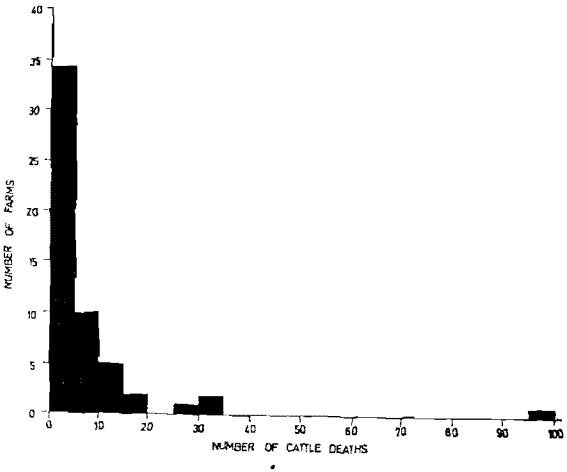

FIG. 4
Mortality of cattle due to theileriosis October 1964–*September* 1966

The outbreak, during which approximately 100 cattle died, occurred in an outlying area of the lowveld frequented by buffalo. Owing to the remote locality of this outbreak, only a single smear specimen was obtained and this was sufficient only to raise the suspicion that theileriosis was present. The next most severe outbreak occurred in the higher veld far away from buffalo and was well documented. On this farm 35 out of 100 newly acquired cattle died between the 21st and 31st day after introduction onto the farm. The *post mortem* appearances were typical and positive specimens were obtained from 11 of the animals. The cattle already on this property were not affected.

Among the theileriosis cases produced in the laboratory only eight of the 39 positive *Theileria* transmissions terminated fatally but in two of these instances (bovines 414 and 486 which died respectively on days 126 and 23 after tick infestation) the immediate cause of death was not related to theileriosis. Of the six which died due to theileriosis, four (bovines 381, 382, 453 and 469 which died respectively on days 20, 23, 19 and 21 after tick infestation) had typical lesions of acute Rho-

desian type theileriosis at *post mortem* examination, while the remaining two (bovines 419 and 421 which died respectively on days 26 and 37 after tick infestation) had lesions consistent with less acute theileriosis at *post mortem* examination. Among the 31 theileriosis cases which recovered, the clinical symptoms varied from very severe illness, including prolonged fever, swollen lymph nodes, respiratory distress and loss in condition, to just a few days of fever with swollen lymph nodes. The least affected were three animals (bovines 3338, 3587 and 471) which developed no fever but whose lymph nodes became swollen: Koch's blue bodies (bovines 3338 and 3587) and endoglobular parasites (bovines 3338, 3587 and 471) had been seen to develop. In a single instance (bovine 727, an intact animal) parasites were not found but theileriosis was diagnosed on the fever reaction. There was no clear cut division in the pathogenicity of the 39 isolates which caused reactions varying from inapparent illness to severe and fatal theileriosis. Fever and extensive oedema of the head as seen in rhipicephaline tick toxicosis were observed during the period of rapid tick engorgement in most of those animals on which more than 100 female ticks were simultaneously engorging; but in general the toxicosis fever had subsided by the time theileriosis set in, (nine severe cases were seen between days 4 to 8 after tick infestation). *Ehrlichia bovis* was the only other parasite to be transmitted by the adult *R. appendiculatus* ticks (thirteen cases were seen between days 20 to 41 after tick infestation) and fever accompanied the *Ehrlichia* parasitaemia.

Estimation of parasitosis

Parasitosis was studied in 110 positive cases from the field and estimates ranged from less than one to 400 schizonts per 1000 lymphocytes with a mean of less than 46.

Among the 39 theileriosis cases produced in the laboratory schizonts were not seen in seven instances. In the remaining cases the estimates ranged from less than one to 690 schizonts per 1000 lymphocytes with a mean of less than 40. About day 14 after tick infestation seemed the best time to look for schizonts. Microschizonts appeared later than macroschizonts. Microschizonts were seen in approximately 10% of the field smears compared to approximately 2% of the equivalent laboratory smears.

The estimates of parasitosis of both the field cases and the laboratory isolates are summarised in fig. 5.

Counts of the number of chromatin particles within schizonts

Chromatin counts were made from the macroschizonts in well prepared smears from seven of the laboratory theileriosis cases and the counts ranged from 2 to 37 particles although the majority of chromatin counts were in the lower range.

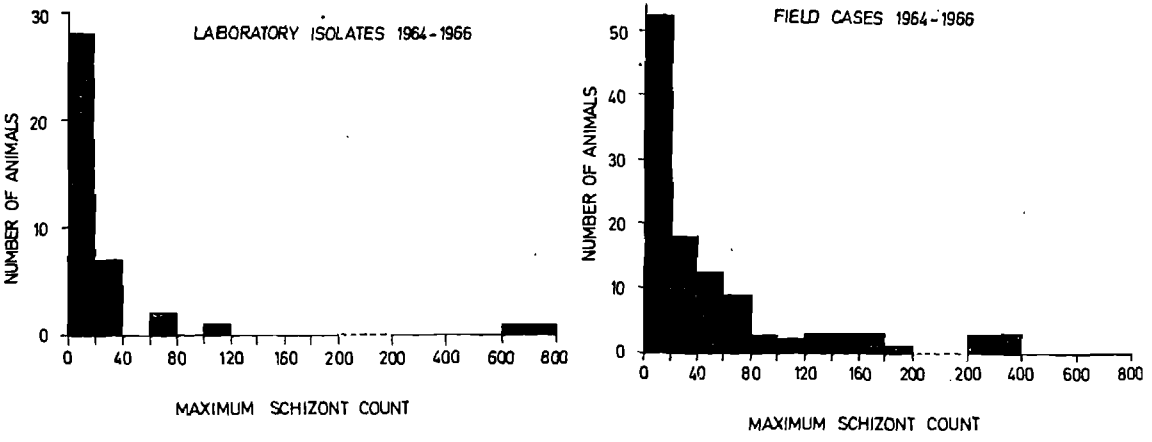

FIG. 5
Estimates of parasitosis in theileriosis October 1964–September 1966

The data on the chromatin counts are summarised in table 3.

TABLE 3.—CHROMATIN COUNTS

Laboratory isolates		
Animal	*Range*	*Mean*
420	2–34	8
437	2–37	13
453	2–21	6
454	2–26	7
463	3–10	6
469	2–20	5
470	2–11	6
	Schizonts examined = 200	

Estimation of the size of schizonts

Schizont sizes were determined of macroschizonts in well prepared smears from seven of the laboratory theileriosis cases and measurements varied from 1.4 microns to 7.8 microns. Larger macroschizonts (up to 25 microns) have been seen in specimens from other theileriosis cases studied in the laboratory.

The data on macroschizont sizes are summarised in table 4.

TABLE 4.—MACRO-SCHIZONT SIZES

Laboratory isolates		
Animal	*Range*	*Mean*
	μ	μ
420	1.4—5.2	3.1
437	2.2—7.1	4.1
453	1.4—6.0	3.1
454	2.1—7.3	3.1
463	2.1—7.8	4.4
469	1.9—5.2	3.5
470	1.7—5.6	3.6
	Schizonts examined = 450	

Estimation of parasitaemia

Parasitaemia was studied in 66 positive cases from the field, and estimates varied from less than one to 80 parasites per 1000 erythrocytes. Dust and other contamination makes it difficult to make this examination in some field specimens while in others endoglobular parasites could not be found although schizonts were present.

Among the 39 theileriosis cases produced in the laboratory, endoglobular parasites were seen in 35. In these the parasitaemia varied markedly, depending on whether the animal was intact or splenectomised. In the intact animals maximum counts were observed mostly between days 19 and 24 whereafter the parasitaemia subsided with only the occasional appearance of an erythrocytic parasite. In the surviving splenectomised animals the build up of parasites continued progressively and maximum counts were observed mostly between days 58 and 121. The progression of parasitaemia did not appear to cause illness, except for a transitory bout of mild anaemia over a day or two immediately preceding the decline of parasite numbers to a persistent lower grade of parasitaemia. Thus the clinical symptoms of theileriosis in Rhodesia are primarily attributed to the activity of schizonts.

The intra-erythrocytic forms are pleomorphic. In the early stages of the disease smaller forms are more common, including comma, rod and anaplasma types, while later the picture is dominated by the characteristic acorn shaped parasite.

The data on parasitaemia is summarised in table 5.

TABLE 5.—ENDOGLOBULAR PARASITAEMIA

Source	*Number of examinations*	*Range per 1,000 RBC*	*Mean per 1,000 RBC*
Positive field cases	66	<1–80	6
Isolates in intact animals	9	<1–11	2
Isolates in splenectomised lab. animals	26	<1–480	62

DISCUSSION

The aim of this study was to discover whether *T. lawrencei* in Rhodesia was already behaving in the field as *T. parva*. The results show that this change has not occurred here, although cattle to cattle passaging is likely to have been present for decades. (Cattle to cattle passage is proven by the success of isolation method B into bovine 3991.) *T. parva* (East Coast fever) has been eradicated and is not present in Rhodesia today.

The *Theileria* which killed so many cattle in Rhodesia in the past was characterised by a very high rate of parasitaemia. Koch[2] states that in the advanced stage of the disease "in each or every other red blood corpuscle there are one or more small parasites". Among the numerous fatal cases studied by Koch only one remained as a low parasitaemia (about one parasite in six blood corpuscles). As noted earlier in the paper, Theiler[3] was responsible for naming this protozoon and his descriptions tally with those of Koch[2]. The Schoonspruit strain of *T. parva* which has been maintained experimentally by tick passage in cattle for decades at the Onderstepoort laboratories in South Africa behaves in precisely the same manner. Neitz[1], who works with this strain, describes the

parasitaemia in similar terms to Koch and Theiler while he notes that the schizonts in the advanced stage of the disease usually exceed 600 per 1000 lymphocytes. In reviewing the literature, Wenyon[5] concludes that the parasitaemia increases rapidly "till 80 to 90 per cent of the corpuscles are affected". By contrast, in Kenya, Barnett *et al.*[24] and Brocklesby[17 18] describe the Muguga strain of *T. parva* as being very variable with an average of 264 schizonts per 1000 lymphocytes (range 4-760) although the endoglobular parasitaemia is apparently frequently very high.

For comparison the Kenya authors give average schizont sizes as 5.2 microns (range 1.6 to 15.9) in "post mortem" material and 4.8 microns (range 0.8 to 13.7) in biopsy specimens. Their average chromatin counts are respectively 5.44 to 18.40 and 5.30 to 12.82 chromatin particles per schizont for autopsy and biopsy specimens respectively. They conclude that the great variation in degree of parasitosis makes this estimate valueless for differential diagnosis. They appear to place more reliance on parasitaemia.

Study of the earlier records in Rhodesia[16 25], confirms that *T. parva* was the main problem. Similarly, the records confirm that in the 1930's a new *Theileria* parasite, distinguished by its relatively few schizonts and far fewer intra-erythrocytic parasites was associated with the theileriosis outbreaks. This new form of theileriosis gradually predominated. Careful search of the archive records[16 25], revealed several earlier records of isolated cases of theileriosis characterised by the presence of schizonts but with few, if any, endoglobular parasites present, namely in 1911, 1912, 1914 and 1928.

Until such time as further knowledge is available I propose to identify the *Theileria* studied in this investigation as *T. lawrencei* (Neitz, 1955)[9] with the following description:

A *Theileria* species infecting cattle and buffalo, characterised by a low rate of parasitosis and parasitaemia, and transmitted by adult *R. appendiculatus* ticks which acquire the infection by feeding in the previous instar on either infected cattle or buffalo. Pathogenicity of the *Theileria* varies from causing inapparent illness to severe and fatal theileriosis whose main feature is the rapid course (frequently as brief as 19 to 23 days after tick infestation) with oedema of the lungs as the prominent lesion at *post mortem* examination.

T. lawrencei as defined above is readily distinguished from *T. parva* (Theiler, 1904)[3] provided a full examination is made. In the early stages of East Coast fever or where only lymph node and spleen smears are available, differential diagnosis is not possible.

T. lawrencei as defined above is indistinguishable in carrier animals or during inapparent illness from *T. mutans* (Theiler, 1906)[11].

In Rhodesia in the higher veld on the minority of farms where ticks are not controlled effectively, the infection rate of the *Theileria* in adult *R. appendiculatus* ticks is virtually universal in contrast to the position in the lower veld. The virulence of *T. lawrencei* varies and only a small proportion of the ticks are infected with the severest forms.

It is interesting to compare the epizootiology of *T. lawrencei* in Rhodesia, as outlined above, to the situation prevailing in South Africa and Kenya.

In South Africa, according to Neitz *et al.*[8], Neitz[9] and Mansvelt[20], *T. lawrencei* is found in certain localities of Zululand and the Eastern Transvaal where cattle come into contact with buffalo. Mansvelt[20] states: "Mortality rate in cattle may be as high as 90% but under natural conditions cattle do not serve as reservoirs and no cattle-to-cattle transmission occurs. Neitz has only succeeded to do this experimentally from splenectomised infected cattle." *T. parva*, once widespread, has been eradicated from South Africa and no cases have been detected for over a decade.

In Kenya, where *T. parva* is still endemic, a situation prevails which is different from both that in Rhodesia and in South Africa. According to Brocklesby[17 18], laboratory experiments show that *T. lawrencei* is readily transmissible from cattle to cattle, furthermore, this may result in such alterations of its characteristics that it is indistinguishable from *T. parva*. Thus the Kenya authorities cast doubt on the validity of *T. lawrencei* as a separate species.

No explanation is available as to why *T. lawrencei* should behave so differently in Kenya, Rhodesia and South Africa.

In Rhodesia, theileriosis may be complicated by concurrent rhipicephaline tick toxicosis due to heavy tick infestation. Laboratory evidence has been presented to show that ehrlichiosis may also aggravate the condition. The disease is now seasonal in nature in contrast to the position of East Coast fever in the early days which broke out frequently at other times[16]. Following my first season's work as reported by my Director, May[27], I concluded that "*Theileria mutans*, aggravated by tick toxicosis, is the apparent cause of theileriosis in the Sinoia district". While this may be the true position in respect of the less severe outbreaks of theileriosis, I am now inclined to think that the evidence equally supports the conclusion stated above that "only a small proportion of the ticks carry the severest forms". Thus, from the practical point of view, heavy tick infestation is more likely

to lead to fatal theileriosis. However, from my unpublished experiments which are still in progress, I am now aware that the feeding to engorgement of even less than five ticks infected with the severest form of *T. lawrencei* is sufficient to kill a beast in 20 days, so that heavy tick infestation need not be present.

In relation to our increasing cattle population the ravages of this disease are diminishing. However, our individual animals are becoming increasingly valuable, so that the loss of a single beast is becoming important to the farmer. The majority of our leading herds, even in the heart of the theileriosis prone areas, are under complete tick control with total interruption of disease transmission. This trend could be hastened on the field side by increasing the effectiveness of tick control by greater attention to correct dipping procedures such as accurate tank measurements, full strength dips, premixing, hand-dressing of the tick predilection sites, dry season dipping[28], and the training of farmers in tick control techniques. There would seem less reason now to eliminate buffalo solely as prophylaxis against theileriosis.

On the research side attention should be concentrated on elucidating the factors which influence the epizootiology of theileriosis, discovering means for detecting theileriosis carriers and improving techniques for tick control. Also *Theileria* of non-bovine origin should be isolated for comparison with the cattle isolates already in hand; as a corollary to this, a search should be made for reservoirs of theileriosis other than cattle and buffalo.

The success of the campaign to eradicate East Coast fever was based on our ability to eliminate the reservoir of infection — a task to which the dominating characteristics of *T. parva* were particularly favourable. The eradication of this parasite has made possible the thriving Rhodesian cattle industry of today; thus learning from the past every effort should be made similarly to eradicate *T. lawrencei*. Farmers would be well advised to follow the dictum: NEVER BUY BREEDING STOCK FROM A THEILERIOSIS PROPERTY.

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Bovines 3991, 3338, 3319 and 3587 were infested at the Veterinary Laboratories, Onderstepoort, South Africa in collaboration with Professor W. O. Neitz and with the kind permission of the South African authorities.

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BOOK REVIEW

THE ARTHROPOD PARASITES OF VERTEBRATES IN AFRICA SOUTH OF THE SAHARA
(Ethiopian Region)

VOLUME III (INSECTA EXCLUDING PHTHIRAPTERA)

by

F. ZUMPT

in collaboration with

E. HAESELBARTH AND J. SEGERMAN

Published and obtainable: S.A. Institute for Medical Research, Box 1038, Johannesburg. pp. 282, Price R10.00

This volume, the second to appear in a series of four volumes contemplated to cover the invertebrate parasitic fauna of Africa south of the Sahara, should prove to be a welcome addition to the libraries of veterinarians and biologists in general and entomologists in particular.

The style adopted conforms with that of the previous volume but in view of the very much wider field covered some sections have had to be curtailed to the bare essentials in order to make possible a volume of reasonable size.

The work opens with the listing of the nine known species of the *Hemimeridae* which are included in the order *Dermaptera*. The synonomy, distribution and taxonomic literature of these interesting parasites of the giant rats of Africa are dealt with shortly.

The bugs or *Cimicidae* of the order *Hemiptera* follow, with a key to the seven known genera. Details of cimicid morphology, illustrated by means of line drawings, are included together with biological notes and a discussion of the current theories regarding the association with disease of those species parasitizing man. The *Polyctenidae*, associated only with bats, are dealt with in similar fashion, a key for the recognition of the three genera occurring in the Ethiopian region being included with the species listed under each genus.

The order *Diptera* or two-winged flies is dealt with in detail only in so far as those forms showing close association with specific hosts are concerned. For the rest, summarizing literature is listed and a key to the families of both the *Nematocera* and *Brachycera* is included. The species of those families which have attracted attention by virtue of their association or potential association with disease transmission together with taxonomic literature references are given e.g. the *Psychodidae*, *Stomoxyinae*, *Glossinidae*, *Calliphorinae* and *Gasterophilinae*. For the myiasis producing *Oestridae* and *Gasterophilidae*, which are dealt with in some detail, a key to the genera is included for the Ethiopian region. The family *Hippoboscidae* is discussed very extensively; a key to the thirteen genera south of the Sahara is included and the known species, including the small louse flies of bats, are elaborated upon.

Fully one third of the volume is devoted to the *Siphonaptera* or fleas. This treatise deals with the species found on mammalian hosts including those occurring on bats. In view of the excellent paper of De Meillon, Davis & Hardy on the mammalian forms and that of Marcus on the bat fleas, the present work may appear to be in part somewhat repetitive. However, the geographical region included extends beyond that dealt with by the above mentioned authors and would appear to justify bringing the flea fauna of a vast region of Africa together in a single publication.

The host-parasite list has been compiled in great detail and is most comprehensive. Seven pages of literature references, referred to in the text by author only are followed by an index of parasites which greatly facilitates reference to all the forms listed. An index of the common names of hosts, with page references as to where they occur, is of considerable value to the non-specialist.

The volume is printed on semi-glazed paper of fine quality and is profusely illustrated throughout by means of excellent line drawings and photographs.

R. du T.

PROFESSIONAL PROVIDENT SOCIETY OF SOUTH AFRICA

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IMPORTANT ANNOUNCEMENT

INCREASE IN MAXIMUM SHAREHOLDING

The Board has pleasure in announcing an increase in the maximum shareholding from 60 to 70, with effect from the 1st January, 1967. This will increase the maximum sickness benefit to R98 per week, and the maximum permanent disability benefit to R49 per week.

The maximum sum assured under the Group Life Assurance Scheme will be correspondingly increased to R14,000, subject, however, to the proviso that it will be reduced to R12,000 at the end of the year in which the member reaches the age of 65, or on his earlier retirement.

The effective date of the additional shares will, on request, be backdated to 1st January, 1967, provided applications are made before the 30th April, 1967.

W. H. C. KOHLER
General manager.

March, 1967.

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