CORRIGENDUM

"Symposium on Canine Biliary Fever" Vol. 47, No. 4, p. 285-287. 3. Fluid Therapy in Canine Babesiosis by C. Button: Table 1 on p. 286 should be replaced by the following revised table:

Table 1: ACID BASE PARAMETERS AND SERUM ELECTROLYTES IN NORMAL DOGS, DOGS SURVIVING SEVERE BF AND FATAL CASES OF BF

	Normal Values ± SD	Dogs surviving severe BF ± SD	Fatal cases of BF ± SD
Aterial pH	7,39 ± 0,02 (20)*	7,33 ± 0,09 (19)	7,02 ± 0,16 (4)
Aterial PCO_2 mm Hg	34,1 ± 3,4 (20)*	22,1 ± 10,1 (19)	15,0 ± 9,0 (4)
Arterial HCO_3 m eq/ℓ	20,0 ± 2,5 (20)*	11,2 ± 4,3 (19)	6,6 ± 1,1 (4)
Aterial base excess	—3,9 ± 2,1 (19)*	-14,1 ± 5,2 (19)	-21,6 ± 2,8 (4)
Aterial Lactate m eq/ℓ	1,53 ± 0,84 (20)*	4,33 ± 4,90 (20)	16,1 ± 2,87 (5)
Na m eq/ℓ	141,1 – 152, 3†	139 ± 14,8 (18)	147 ± 8,10 (3)
K m eq/ℓ	4,37 – 5,65 †	3,8 ± 0,7 (18)	4,0 ± 1,5 (3)
C1 m eq/ℓ	105,2 – 114,8†	119 ± 7,20 (15)	—

Figures in brackets are the number of dogs.
*Author's figures, derived from normal dogs of large breeds; bled approximately 4 hours after feeding and 20 minutes after being held quietly on a table.
†In clinical biochemistry of domestic animals.

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SCHISTOSOMA MATTHEEI IN THE OX: THE CHRONIC HEPATIC SYNDROME

J.A. LAWRENCE*

ABSTRACT: Lawrence J.A. *Schistosoma mattheei* **in the ox: the chronic hepatic syndrome.** *Journal of the South African Veterinary Association* (1977) **48** No. 2, 77 – 83 (En) Veterinary Research Laboratory, P.O. Box 8101, Causeway, Rhodesia.

A Friesland steer infested on four occasions at intervals of 4–6 weeks with 20 000 cercariae of *Schistosoma mattheei* developed progressive hepatic failure and died after 74 weeks. The condition was characterised by enlargement and induration of the liver with portal fibrosis, inflammation of the portal veins and "piecemeal necrosis", and was associated with a severe circulating eosinophilia and hypergammaglobulinaemia. Similar cases were encountered in two natural outbreaks. The syndrome is considered to be of immunological origin, initiated by the reaction in the portal veins to antigen from schistosomes killed by the immune response of the host. It is usually seen in animals exposed to repeated heavy infestation but may occur occasionally after light infestation.

INTRODUCTION

Schistosoma mattheei Veglia and le Roux (1929) is a common parasite of the ox in Southern Africa, incidence rates as high as 92% having been recorded in Rhodesia[3], and occasional outbreaks of clinical schistosomiasis have been reported [9 14 17]. In most instances the clinical disease takes the form of an intestinal syndrome, associated with the passage of large numbers of eggs through the intestinal wall in the early weeks after heavy primary infestation[8]. The first indication that the parasite could cause any other form of clinical disease was provided by van Wyk and Bartsch[16]. They reinfested an experimental ox with a large number of cercariae 18 months after primary infestation with a small number. The animal showed no clinical signs for 4 months and then lost condition and showed straining, knuckling over at the hind fetlocks and nervous derangement. At post-mortem examination the authors found marked periportal hepatic fibrosis and they named the condition "hyperergic schistosomiasis". The syndrome was recognised subsequently in a natural outbreak[17].

During the course of an investigation into bovine schistosomiasis in Rhodesia this syndrome was reproduced experimentally in one animal and was encountered in the field on two occasions. It has been renamed the chronic hepatic syndrome, in order to avoid the presumption on the aetiology implicit in the original name.

EXPERIMENTAL DISEASE

MATERIALS AND METHODS

Two Friesland male calves, Nos. 924 and 925, reared under conditions of minimal exposure to trematode parasites, were infested through the skin for the first time at the ages of 8 and 9 months with 20 000 cercariae of *S. mattheei,* a mean of 111 cercariae/kg body mass. They were reinfested with 20 000 cercariae on three subsequent occasions at intervals of 4 to 6 weeks. Cercariae were obtained from aquarium-reared *Bulinus (Physopsis) globosus* snails infested with a strain of parasite originating from cattle at the Salisbury abattoir and maintained thereafter in cattle and snails. The animals were stabled and held on a high plane of nutrition, with a mean mass gain of 0,7 kg/day, throughout the experiment except for a period of 14 weeks when they were turned out to grass. There were two uninfested controls.

The calves were examined daily for clinical evidence of disease and were mass measured every 2 weeks. Blood samples were collected in sequestrene at intervals of 1 to 2 weeks for routine haematological examination[4] and serum proteins were determined at similar intervals by a modification of the biuret method of Wooton[20], using a 27,2% solution of sodium sulphite for precipitation of the globulin and filtration at 37°C for separation. Faecal egg counts were determined by the sieving technique of Lawrence[5].

Body mass, haemoglobin, serum albumin and serum globulin values of the infested animals were adjusted for the variation in the controls in order to standardise the method of recording with that used in a study of clinical pathological changes after primary infestation (Lawrence, in preparation). For each parameter the mean change in the controls from one observation to the next was assumed to represent the expected change in the infested animals over the same period. From this figure an "expected value" for each observation was calculated and the "actual value" was expressed as a percentage of the "expected value". Percentage values were used in compiling the figures for this paper.

One animal died as a result of infestation. The other was slaughtered and parasites recovered from mesentery, stomachs, liver, lungs and bladder by a modification of the perfusion technique used in sheep by McCully and Kruger[11]. Egg counts in liver, lung and intestine at various levels were determined by the sieving technique of Lawrence[5] after digestion of the tissue in 5% potassium hydroxide. Tissue blocks were fixed in formol-saline and processed for histological examination by conventional methods.

RESULTS

Between 7 and 16 weeks after the initial infestation both calves showed an acute episode of diarrhoea followed by spontaneous recovery, similar to that described in animals infested only[8] once. Both animals were turned out to grass with five other calves at 27 weeks post-infestation, and after 6 weeks it was noticed that No. 924 was losing condition and was being bullied by other animals in the group. It was isolated in an adjacent paddock for the next 8 weeks, until the whole group was stabled once again.

The condition of the animal deteriorated steadily

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and, although appetite was maintained, it became thin and pot-bellied. A liver biopsy at 65 weeks revealed proliferative changes in the portal veins, thickening of the portal tracts and reduction of the parenchyma relative to the portal tissue. There was "piecemeal necrosis" involving small groups of parenchymal cells immediately adjacent to the portal tracts. At 74 weeks after initial infestation the animal suddenly became dejected, refused to feed, lay down and died quietly during the course of one day.

After recovery from the initial acute reaction No. 925 showed no clinical abnormalities and was slaughtered for post-mortem examination 80 weeks after initial infestation.

Clinical pathological changes in No. 924 are shown in Fig. 1. Faecal egg counts showed an initial sharp rise followed by a gradual fall, similar to the pattern observed after a single infestation, and acute clinical signs, namely anorexia and diarrhoea, were confined to the period of peak egg output. Body mass showed a fall relative to that of the controls over the early stages of clinical illness, followed by a resumption of normal growth until 25 weeks. At this point there was a clearly defined second phase of retardation of growth which persisted until death. Haemoglobin and serum albumin showed a sharp fall associated with clinical illness followed by recovery, as after a single infestation, but recovery was interrupted at between 20 and 25 weeks and the values remained very low until death. The anaemia in the terminal stages was normochromic and macrocytic, with a mean corpuscular volume of 46,4 μm^3, as against 33,4 μm^3 at the time of infestation. Serum globulin and eosinophils showed an initial increase similar to that seen after a single infestation followed by a dramatic secondary response which increased progressively until death.

The major fall in body mass and haemoglobin coincided with the period of 14 weeks out at grass. All the other infested animals also showed slight falls in these values while out at grass, but they were too small to be statistically significant and the values recovered immediately to previous levels on return to the stables.

By contrast the picture in No. 925 is shown in Fig. 2. Serum globulins and eosinophils showed a more prolonged elevation than that seen in animals after a single infestation, but it was less marked than in No. 924 and was not progressive, remission occuring after about 50 weeks. There was a secondary fall in body mass, haemoglobin and serum albumin which was less severe than in No. 924 and was followed by a gradual return to near normal levels.

It is apparent from the figures that in both animals the eosinophil and globulin responses were very marked. In No. 924, at the last examination before death, the differential eosinophil count was 69% and total serum protein was 9,1 g/100 mℓ, with globulins at 7,3 g/100 mℓ. Electrophoretic examination revealed that the increase in globulin was confined almost entirely to the gamma fraction.

In the case of No. 924 it was not possible to count the parasites or to recover them for examination, but they did not appear to be very numerous. From No. 925

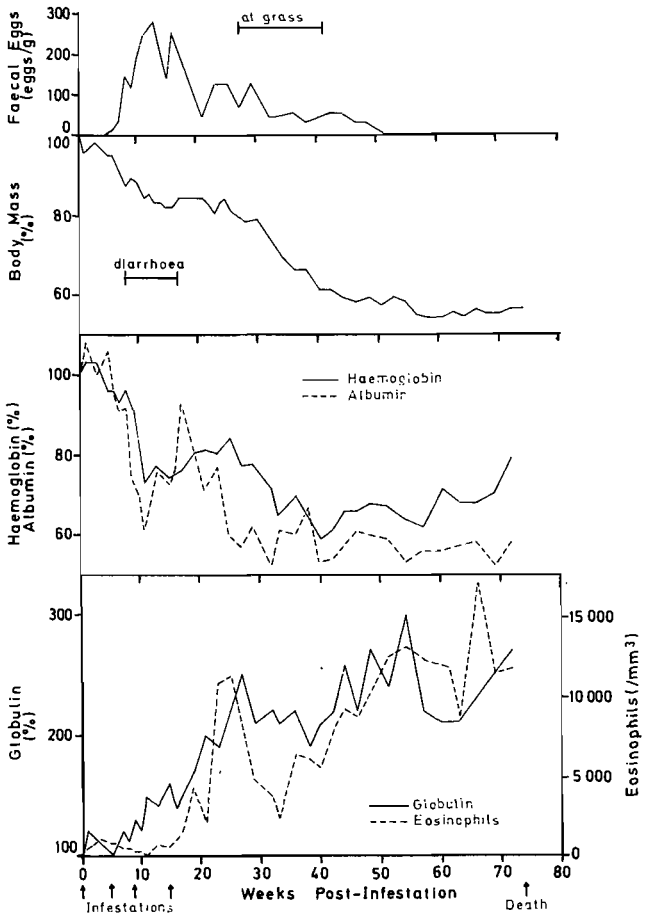

Fig 1 Clinical pathological changes in calf no. 924.

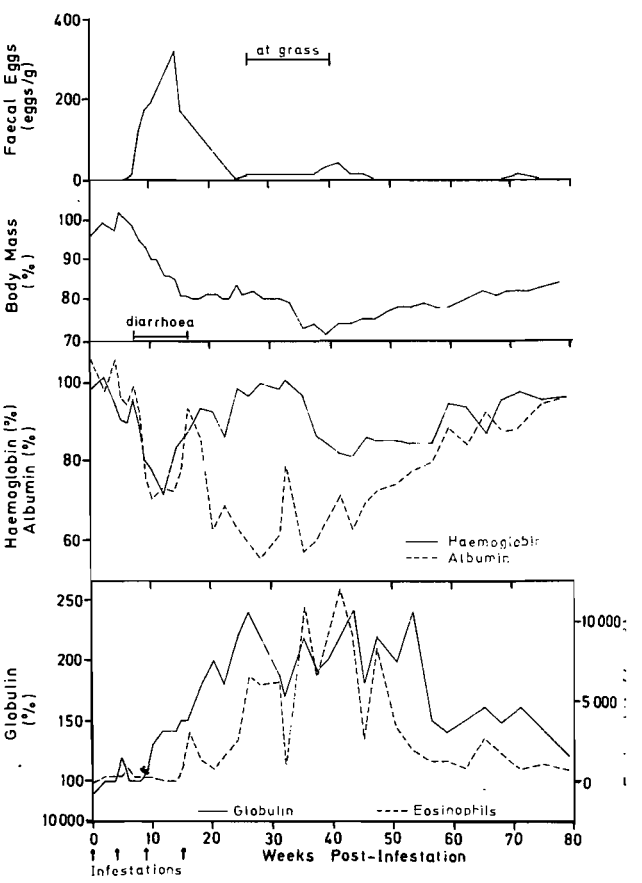

Fig. 2 Clinical pathological changes in calf no. 925.

3 175 worms were recovered at slaughter, representing 4,0% of the total infesting dose. In terms of distribution in the body, length and numbers of eggs *in utero* the parasites were similar to those encountered in other animals at a similar interval after a primary infestation (Lawrence, in preparation).

Tissue egg counts in No. 924 are presented in Table 1. Counts were low in all organs, but the eggs in the liver constituted 66,8% of the total eggs in the body. The total number of eggs in the tissues in No. 925 was estimated to be 10,6 x 10^5 and there was no difference in distribution from that found in other animals at a similar interval after a single infestation (Lawrence, in preparation).

Table 1: TISSUE EGG COUNTS IN CALF NO. 924

Site	Mean eggs/g	Total eggs (× 10^5)
Small intestine†	161	15,0
Ileo-caeco-colon*	33	1,0
Distal colon	40	0,4
Rectum	20	0,2
Liver	192	33,8
Lung	4	0,2

†at 4 levels
*terminal ileum, caecum and proximal colon

Post-morten examination of No. 924 revealed the salient feature to be gross enlargement and induration of the liver. The organ had a mass of 11,6 kg, as against a mean mass of 4,6 kg in five other infested calves of approximately the same age. The capsular surface was grey while the cut surface was yellow-brown in colour and presented a diffuse stellate pattern of grossly thickened portal tracts. Histological examination revealed massive thickening of the portal tracts resulting from medial hypertrophy of the portal veins and fibrosis.

From the portal tracts fibrous tissue and small bile ducts (Fig. 3) had proliferated radially and invaded the parenchymatous tissue of the lobule. There were foci of "piecemeal necrosis" adjacent to the portal tracts (Fig. 4). The portal veins showed marked endothelial hyperplasia and proliferation of the tunica intima, which was infiltrated with eosinophils and mononuclear leukocytes. A few eggs and remnants of parasites were present in granulomas.

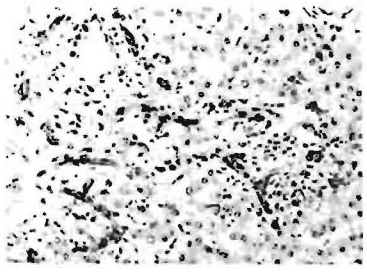

Fig. 3 Bile ducts invading parenchyma. x 250. Haematoxylin and eosinstain (HE).

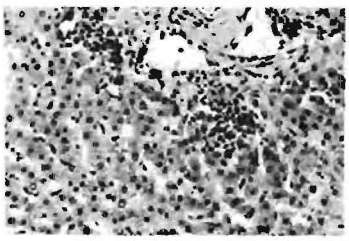

Fig. 4 Foci of piecemeal necrosis adjacent to portal tract. x 250. HE.

Liver failure had been the cause of death. This was indicated by the presence of numerous, small, scattered, focal haemorrhages in the parenchyma, often adjacent to the portal tracts or the central veins, and foci of early necrosis with commencing polymorph infiltration. Moderate quantities of partly digested blood in the abomasum and throughout the intestine and poor clotting of the blood in the large vessels and heart were further evidence of liver failure. The lungs were moderately oedematous and showed a number of infarcts, both recent and of long standing, caused by the lodging of parasites in the pulmonary arteries. In other respects pathological changes resembled those seen at a similar interval after a single infestation (Lawrence, in preparation). In addition to the liver and intestine, eggs were seen in the omasum but were not found in the rumen, reticulum, abomasum, pancreas, mesenteric lymph nodes or bladder.

Post-mortem examination of No. 925 revealed changes similar to those seen after a single infestation. The liver was normal in size and, although there was macroscopic evidence of thickening of the portal tracts, the reaction in the portal veins was subsiding, with few active lesions. There was no invasion of bile ducts and fibrous tissue into the parenchyma. There were no pulmonary infarcts. In addition to the liver and intestine, eggs were present in the rumen, omasum, abomasum and bladder.

NATURAL DISEASE

The chronic hepatic syndrome was recognised in two herds investigated during the study of naturally occurring schistosomiasis.

Herd 1

The first herd was involved in an outbreak of which a brief description has been published previously (Farm D)[9] and was used in a therapeutic trial with stibophen* (Farm B)[10]. The herd consisted of 100 Angus x Mashona cows run on a high intensity grazing system involving the use of small paddocks which were alternately heavily grazed for 2 weeks and rested for 2 months. Most of the paddocks included a marshy stream ("vlei") which was infested with snails. In January, 1968, the owner became concerned at the deterioration in the condition of his cows. One cow died on 5th February, after exhibiting debility and profuse haemorrhage from the rectum for a few days.

*Stibophen Bp – 6,4% Antimony trioxide.

reaction, as appeared to occur in No. 925. It is not clear why, in the absence of continuing reinfestation, the condition should have become progressive in No. 924, to the extent that it terminated in hepatic failure.

A possible explanation is provided by Popper and his colleagues[12 13]. These authors studied the histological features of a variety of liver diseases of man, some of which were progressive, and concluded that peripheral "piecemeal necrosis" was the best indicator of progressive liver disease of any type. They considered that the continuing damage to liver tissue after a primary insult, which characterises progressive conditions, was probably immunological in origin. The most likely cause was the cytotoxic effect of a cell-mediated delayed hypersensitivity reaction involving the T lymphocytes in the liver, following activation by a persisting antigen. Alteration of the proteins of the hepatic cells which resulted might lead to further activation of lymphocytes. They did not regard the conditions as auto-immune diseases, in that they were not initiated by specific auto-antibodies, but as self-perpetuating diseases. The presence of "piecemeal necrosis" in the liver of No. 924 and the progressive nature of the lesion suggest that the chronic hepatic syndrome in bovine schistosomiasis should be classified with the progressive liver diseases of possible immunological origin in man, the reaction in the liver to antigens from dead parasites acting as the initiating cause of liver damage.

It is possible that Symmers' fibrosis in man should also be included among these diseases, as "piecemeal necrosis" is a feature[12 18]. The condition has been reproduced experimentally in the chimpanzee with *S. mansoni*[15] and *S. japonicum*[19] and it is clear that the initial stimulus in this species is not a reaction to dead parasites. It appears to originate partly from the reaction to eggs and partly from an unexplained hyperreactive immunological response in the portal veins[19]. A similar hyperreactive response has been suggested as the cause of the condition in man[1] and it may be a sequel of heavy infestation[2].

The distribution of eggs in the organs of No. 925 was similar to that encountered in animals after a single heavy infestation and included evidence of a partial shift of parasites to the stomachs and bladder (Lawrence, in preparation). In No. 924 there was no evidence of such a shift and a very high proportion of the total eggs present was in the liver. Whether these differences are of any significance in the aetiology of the hepatic syndrome is unknown. In naturally occurring cases in Herd 1, bladder lesions were absent in spite of very heavy infestation, and it is possible that there may be an association between a failure of parasites to move from the mesenteric veins to alternative habitats and an exaggerated immunological response in the liver.

There are indications that even in the absence of the development of a progressive hepatic syndrome heavy reinfestation does have a transient adverse effect on the host. In No. 925 a fall in body mass, haemoglobin and serum albumin occurred while the animal was out at grass. It may have been attributable to the effects of reinfestation as the fall in serum albumin preceded the turning out to grass and the reduction in growth rate and the fall in haemoglobin were more marked than those seen in other animals turned out to grass after a single infestation. A second animal infested three times at intervals of 20 and 34 weeks also showed a marked eosinophil and globulin response following the third infestation, which coincided with a fall in body mass, haemoglobin and albumin[7]. As in No. 925 the reaction was self-limiting.

The outbreak in Herd 1 was characterised by a very heavy infestation of schistosomes resulting from the unusual practicé of concentrating cattle for short, recurrent periods in snail-infested paddocks during the summer, the period of peak transmission. The primary infestation was probably not very heavy as no clinical reaction to it was recognised, but a massive build up of parasites must have occurred, leading to heavy, continuous reinfestation. Two of the cows examined post-mortem were certainly cases of the chronic hepatic syndrome, and the very high globulin and eosinophil levels in the others suggest that they too were showing a response to reinfestation or repeated infestation which was, however, not progressive. Treatment with stibophen apparently reduced the degree of antigenic stimulation and the immunological response, as measured by serum globulin levels, with a corresponding improvement in other blood values, and although it had no effect on body mass it may have been of value in reducing the possibility of the reaction in the liver becoming progressive..

In Herd 2 clinical pathological evidence suggested that the overall level of infestation with schistosomes was low, and this was confirmed in the calf from which parasites were recovered. Nevertheless, it appeared that at the time of slaughter this calf was on the point of succumbing to the chronic hepatic syndrome. It is possible that the animal had a particular sensitivity to the development of a progressive liver condition which became manifest after a relatively minor stimulus.

CONCLUSIONS

It is confirmed that *S. mattheei* can cause a progressive hepatic syndrome of immunological origin in the ox akin to Symmers' fibrosis in man. The syndrome is usually seen in animals exposed to heavy reinfestation but it may occur occasionally after light infestation. It is apparently associated with some individual predisposition, the majority of animals exposed to reinfestation showing a response which is initially similar but is self-limiting.

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LEVAMISOLE: ANTHELMINTIC ACTIVITY IN CALVES FOLLOWING DERMAL APPLICATION

D. ap T. ROWLANDS* and J. BERGER†

ABSTRACT: Rowlands D. ap T.; Berger J. **Levamisole: Anthelmintic activity in calves following dermal application.** *Journal of the South African Veterinary Association* (1977) **48** No. 2, 85 – 93 (En) Wellcome Research Laboratories, Berkhamstead, Hertfordshire, England.

A series of seven experiments designed to evaluate the anthelmintic efficacy of levamisole (1-tetramisole) by dermal application is described. This work involved use of 181 artificially infested calves. The drug was formulated at a concentration of 10% m/v levamisole base in a solvent system promoting dermal absorption and was applied to both sides of the spine in the lumber region at a dose rate of 10 mg levamisole base per kg livemass. A consistent performance was achieved in these investigations, the results from all of which were based upon necropsy findings. Efficacy of treatment was assessed against the third and fourth larval stages and fifth stage larvae/adult worms of six nematode species; the respective efficacies obtained were *Haemonchus placei* 72,0%; 99,3%; and 100%: *Ostertagia ostertagi* 85,5%; 38,1% and 74,5%: *Cooperia* spp. 98,9%; 99,9% and 100%: *Bunostomum phlebotomum* 83,0%; 100% and 98,5%: *Oesophagostomum radiatum* 47,4%; 94,9% and 99,6% and *Dictyocaulus viviparus* 79,5%; 94,1%; 90,9% (fifth stage larvae) and 93,8% (adult worms). The anthelmintic efficacy of levamisole in these experiments is of the same order as that achieved by orthodox methods of administration at dose rates between 7,5 and 10,0 mg levamisole HC1 per kg livemass. These findings add a new dimension to the use of levamisole and to anthelmintic therapy in general.

INTRODUCTION

Thienpont *et al*[18] first described the anthelmintic activity of dl-tetramisole. Considerable evidence has since been presented which illustrates the broad spectrum of efficacy of the drug against the major parasitic nematodes of livestock. Rubin & Hibler[16] reported that most of this activity was attributable to the laevoisomer, levamisole, a finding which enabled the dose to be reduced by 50%. Numerous subsequent publications confirmed the high anthelmintic efficacy of levamisole. Over the last few years new methods of administration have been developed and stockmen now have the choice between oral drenching, subcutaneous and intramuscular injection and in-feed medication. Brooker & Goose[3] and Dorn & Federman[5] have recently drawn attention to yet another, novel method of treating with levamisole, viz. that of dermal application. Preliminary investigations were carried out by Curr[4] and Kingsbury[11] on the activity of a "pour-on" levamisole formulation and their results confirmed the high efficacy reported by Brooker & Goose[3] and Dorn & Federman[5]. Further detailed trials were therefore undertaken, the results of which form the basis of this report.

MATERIALS AND METHODS

A total of 181 calves was used in these experiments which were carried out at Kwanyanga Research Station, East London. These animals had been weaned at an early age and reared on slats, they were 3 to 6 months old at commencement and weighed, on average, 85 kg at treatment. Because of the large numbers involved it was impossible to keep to either a single breed or sex; the vast majority were, however, bull calves of the Friesian type. Throughout the experimental period these animals were kept on slats and fed a lucerne/concentrate ration. All calves received at least two doses of parbendazole at 60 mg/kg live mass during the pre-experimental period as an extra precaution against chance helminth infestations. Donor calves were kept for each of the following species of parasitic nematode: *Haemonchus placei, Ostertagia ostertagi, Cooperia* spp. *(C. pectinate* and *C. punctata), Bunostomum phlebotomum, Oesophagostomum radiatum* and *Dictyocaulus viviparus.* Larval cultures were maintained for each species according to standard procedures, the third stage larvae recovered being checked for purity of culture[9]. Larval doses were made up immediately prior to use and infestation was accomplished either by administering the larvae, in water, by stomach tube or, in the case of *B. phlebotomum,* by dermal application of infective larvae in the following manner. Each calf received a single infestation with an estimated 2 000 larvae concentrated in 2 mℓ of water and applied by a capillary pipette to clipped areas (10 × 15 cm) both sides of the dorsal midline in the lumbar region. These areas had been washed with clean warm water immediately before larval application and were kept constantly damp for at least 10 minutes with additional drops of water. Each calf was restrained by neck-yoke in an individual pen, the walls of which were kept wet in an attempt to retard the drying rate of the infected skin. Trickle infestation techniques were used in all cases with the exception of *B. phlebotomum* and *D. viviparus.* Single infesting doses were used for these species due to the possibility of an immune reaction developing in response to trickle infestation.

Levamisole, (ℓ)-2,3,5,6-tetrahydro-6-phenylimidazo 2,1-b thiazole, was formulated at a concentration of 10% m/v base in a suitable solvent system. Treatment was administered at the rate of 10 mg levamisole base per kg livemass. The drug was applied dermally by means of a pipette to both sides of the spine in the lumbar region over a length of about 12 cm. After treatment all calves were housed in individual pens and were yoked in a restraining apparatus which totally prevented any self-licking of the treated area.

Experimental procedures adopted throughout were as described by Reinecke[14] for larval anthelmintic tests in ruminants suitable for analysis by a modified non-parametric method of evaluation. The only significant departure from these procedures was that total worm

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counts were carried out at necropsy in all cases, other than the indicator control calves, in preference to aliquot sampling techniques.

Identification of the various species and larval stages was carried out according to the following authorities:

H. placei	—	Bremner[2]
O. ostertagi	—	Douvres[6] and Rose[15]
Cooperia spp	—	Keith[10]
B. phlebotomum	—	Sprent[17]
O. radiatum	—	Andrews & Maldonado[1]
D. viviparus	—	Douvres & Lucker[7]

Worms in the 3rd moult were classified as 3rd stage larvae and those in the 4th moult as 4th stage larvae.

EXPERIMENTAL DESIGNS AND RESULTS

Experiment 1

Activity was assessed against both 4th stage larvae of *O. ostertagi* and *D. viviparus* (Group 1) and also against the 5th larval and adult stages of *O. ostertagi* and the 5th stage larvae of *D. viviparus* (Group 2).

This experiment was conducted along the lines of Reinecke's[14] "combined trial" using the same group of nine control animals for each of two groups of 11 treated calves.

At necropsy the abomasum, first 5 metres of small intestine, trachea and lungs of each of the calves and including the mesenteric and hepatic lymph glands of the two indicator control calves, Nos. 700 and 650, were processed for worm recovery. The design of this experiment is illustrated in Table 1a.

Table 1a: *EXPERIMENT 1.* DESIGN OF THE EXPERIMENTS ON ACTIVITY AGAINST BOTH THE FOURTH STAGE LARVAE OF *O. OSTERTAGI* AND *D. VIVIPARUS* (GROUP 1), AND THE FIFTH LARVAL AND ADULT STAGES OF *O. OSTERTAGI* AND THE FIFTH STAGE LARVAE OF *D. VIVIPARUS* (GROUP 2).

Day	Number of infective larvae dosed to each calf *O. ostertagi*	*D. viviparus*
−23	397	
−22	397	
−21	397	
−20	393	
−19	393	
−18	393	
−17	407	733
−16	407	
−15	407	
−14	407	
Total	3 998	733

−11	Randomization into three groups		
	Control	Treated – Group 1	Treated – Group 2
−10	Slaughter Indicator Control Calf 700	Treat 11 calves dermally: levamisole at 10 mg/kg	
0	Slaughter Indicator Control Calf 650		Treat 11 calves dermally: levamisole at 10 mg/kg
+4 +5	Slaughter 4 Control Calves Slaughter 5 Control Calves		
+6		Slaughter 5 Treated Claves Slaughter 6 Treated Calves	
+8 +11			Slaughter 5 Treated Calves Slaughter 6 Treated Calves

RESULTS

Indicator control calves

Calf No. 700 slaughtered on the day of treatment of Group 1 (Day -10) yielded 1 811 *O. ostertagi,* more than 95% of which were in the 4th larval stage. In addition 41 4th stage *D. viviparus* larvae were found in the lungs and two 3rd stage lungworm larvae were recovered from the mesenteric lymph glands. Calf No. 650 slaughtered on the day of treatment of Group 2 (Day 0) yielded a total of 1 913 *O. ostertagi,* more than 93% of which were in the 5th larval or adult stage. From the lungs of this animal 251 5th stage larvae of *D. viviparus* were recovered.

Control and treated calves

The results are summarised in Table 1b.

Control calves

Mean numbers of 2 484 *O. ostertagi* and 294 *D. viviparus* were recovered from the 9 untreated calves.

Treated calves

Group 1 – Activity against 4th stage larvae: Mean numbers of 1 537 *O. ostertagi* and 17,5 *D. viviparus* were recovered from the 11 treated calves, representing mean treatment efficacies of 38,1% and 94,1% respectively.

Group 2 – Activity against 5th stage larvae and adult worms: Mean numbers of 634 *O. ostertagi* and 26,7 *D. viviparus* were recovered from the 11 treated calves, representing mean treatment efficacies of 74,5% against 5th stage larvae and adult *O. ostertagi* and 90,9% against the 5th stage larvae of *D. viviparus.*

Experiment 2

Activity was assessed against the 3rd stage larvae of *O. ostertagi* and the 4th stage larvae of *Cooperia* spp. and *O. radiatum.* At necropsy the abomasa, small and large intestines of all the animals were processed for worm recovery. The design of this experiment is illustrated in Table 2a.

Table 1b: *EXPERIMENT 1.* NUMBER OF WORMS RECOVERED AT NECROPSY, PERCENTAGE REDUCTION IN WORM BURDENS AND EFFICACY CLASSIFICATION

	O. ostertagi			*D. viviparus*		
	Control	*Group 1* (Dosed at L_4 stage) Treated	*Group 2* (Dosed at L_5 Ad stage) Treated	Control	*Group 1* (Dosed at L_4 stage) Treated	*Group 2* (Dosed at L_5 stage) Treated
Control Group = 9 calves	1 981	1 224	320	183	0	0
	2 208	1 310	370	219	1	0
	2 262	1 311	380	241	3	0
	2 403	1 326	428	244	6	0
	2 513	1 437	543	294	6	3
Treatment Group = 11 calves	2 604	1 488	558	297	10	6
	2 612	1 583	710	320	17	14
	2 626	1 654	808	365	19	23
	3 147	1 764	877	483	23	79
		1 766	924		32	79
		2 045	1 055		75	90
Group Mean	*2 484*	*1 537*	634	294	17,5	26,7
Group Mean Reduction		38,1%	74,5%		94,1%	90,9%
Control Median	2 513			294		
Median x 0,25	628			74	1/11 exceeds 74	
x 0,40	1 005		1/11 exceeds 1 005	118		0/11 exceeds 118
x 0,50	1.257	10/11 exceeds 1 257				
Efficacy Classification		Class X	Class B		Class A	Class B

Table 2a: *EXPERIMENT 2.* DESIGN OF THE EXPERIMENT ON ACTIVITY AGAINST FOURTH STAGE LARVAE OF O. *RADIATUM* AND *COOPERIA* SPP. AND THE THIRD STAGE LARVAE OF O. *OSTERTAGI.*

Day	Number of infective larvae dosed to each calf		
	O. radiatum	*Copperia spp.*	*O. ostertagi*
-20	260		
-19	226		
-18	226		
-17	226		
-16	242		
-15	242		
-14	242		
-13	264		
-12	264		
-11	264		
-10			
- 9			
- 8		1 067	
- 7		1 067	
- 6		1 067	
- 5		1 067	
- 4		1 067	
- 3			1 246
- 2			1 246
- 1			1 246
Total	2 456	5 335	3 738
0	Treat 11 calves dermally with levamisole at 10 mg/kg: Slaughter Indicator Control Calf 861		
+21	Slaughter 4 Control Calves		
+22	Slaughter 5 Control Calves		
+23	Slaughter 4 Treated Calves		
+24	Slaughter 4 Treated Calves		
+25	Slaughter 3 Treated Calves		

RESULTS

Indicator control calf

Totals of 385 3rd stage *O. ostertagi*, 2 855 *Cooperia* spp., of which 99,7% were identified as being 4th stage larvae, and 1 077 *O. radiatum*, of which 99,3% were in the 4th stage of larval development, were recovered.

Control and treated calves

The results are summarized in Table 2b.

Control Calves

The mean burdens of these animals were, *O. ostertagi* – 1 721, *Cooperia* spp. – 2 141 and *O. radiatum* – 1 022 worms.

Treated Calves

Mean numbers of 249 *O. ostertagi;* 0,7 *Cooperia* spp. and 52,2 *O. radiatum* were recovered at necropsy, representing mean treatment efficacies of 85,5%; 99,9% and 94,9% respectively.

Experiment 3

Activity was assessed against the 5th larval and adult stages of *H. placei*, *Cooperia* spp., *O. radiatum* and the adult stage of *D. viviparus.*

At necropsy the abomasa, small and large intestines, trachea and lungs of all the animals were processed for worm recovery. As the control group was slaughtered at the time of treatment, no indicator control calves were used in this experiment. The design of this experiment is illustrated in Table 3a.

Table 2b: *EXPERIMENT 2.* NUMBER OF WORMS RECOVERED AT NECROPSY, PERCENTAGE REDUCTION IN WORM BURDENS AND EFFICACY CLASSIFICATION

	O. ostertagi (L_3 stage)		*Cooperia* spp. (L_4 stage)		*O. radiatum* (L_4 stage)	
	Control	Treated	Control	Treated	Control	Treated
Control Group = 9 calves Treatment Group = 11 calves	1 363 1 436 1 437 1 556 1 760 1 896 1 951 2 041 2 050	5 62 146 148 209 219 230 254 301 535 632	4 1 267 1 519 2 322 2 329 2 404 2 834 2 861 3 732	0 0 0 0 0 0 0 0 2 2 4	205 804 863 927 1 003 1 210 1 286 1 362 1 538	3 5 7 7 27 48 49 86 93 121 128
Group Mean	1 721	249	2 141	0,7	1 022	52,2
Group Mean Reduction		85,5%		99,9%		94,9%
Control Median	1 760		2 329		1 003	
Median x 0,25 x 0,40 x 0,50	440 704	0/11 exceed 704	582	0/11 exceeds 582	251	0/11 exceed 251
Efficacy Classification		Class B		Class A		Class A

Table 3a: *EXPERIMENT 3.* DESIGN OF THE EXPERIMENT ON ACTIVITY AGAINST THE FIFTH LARVAL AND ADULT STAGES OF *O. RADIATUM, H. PLACEI* AND *COOPERIA* SPP. AND ADULT *D. VIVIPARUS*

Day	Number of infective larvae dosed to each calf			
	O. radiatum	*H. placei*	*D. viviparus*	*Cooperia* spp.
–40	126			
–39	126			
–38	126			
–37	126			
–36	140			
–35	140			
–34	140			
–33	140			
–32	132			
–31	132			
–30	132	164		
–29	132	164		
–28	132	164		
–27	162	164		
–26	162	196		
–25	162	196	730	
–24	80	196		
–23	80	196		
–22	80	188		
–21	80	188		
–20		188		
–19		188		
–18		166		514
–17		166		514
–16		166		514
–15		166		514
–14				610
–13				610
–12				610
–11				610
–10				610
– 9				610
Total	2 530	2 856	730	5 716

0	Treat 11 calves dermally with levamisole at 10 mg/kg
+1 +2	Slaughter 4 Control Calves Slaughter 5 Control Calves
+6 +7 +8	Slaughter 4 Treated Calves Slaughter 4 Treated Calves Slaughter 3 Treated Calves

RESULTS

The results are summarized in Table 3b.

Control calves

Mean numbers of 426 *H. placei,* 1 441 *Cooperia* spp., 811 *O. radiatum* and 115 *D. viviparus* were recovered from the nine untreated calves.

Treated calves

At necropsy no *H. placei* and no *Cooperia* spp. were recovered from any of the treated calves; treatment efficacy was therefore 100% against these species. Mean numbers of 3,2 *O. radiatum* and 7,1 *D. viviparus* were recovered indicating mean treatment efficacies of 99,6% and 93,8% respectively.

Experiment 4

Activity was assessed against the 3rd larval stages of *Cooperia* spp., *O. radiatum* and *D. viviparus.*

At necropsy the small and large intestines, trachea and lungs of all the calves, and the mesenteric lymph glands (viz. hepatic, cranial and right colic) of the indicator control calf No. 666, were processed for worm recovery. The design of this experiment is illustrated in Table 4a.

RESULTS

Indicator control calf

Calf No. 666 yielded 669 *Cooperia* spp. of which approximately 6% were identified as being in the 3rd stage of larval development and the remaining 94% as early 4th stage larvae. In addition 255 *O. radiatum* were recovered of which 91,8% were 3rd stage larvae.

Table 3b: *EXPERIMENT 3.* NUMBER OF WORMS RECOVERED AT NECROPSY, PERCENTAGE REDUCTION IN WORM BURDENS AND EFFICACY CLASSIFICATION

	H. placei (L_5/Adult stages)		*Cooperia spp.* (L_5/Adult stages)		*O. radiatum* (L_5/Adult stages)		*D. viviparus* (Adult stages – 25 days)	
	Control	Treated	Control	Treated	Control	Treated	Control	Treated
Control Group = 9 calves Treatment Group = 11 calves	228 302 317 332 392 393 519 616 735	0 0 0 0 0 0 0 0 0 0 0	455 541 798 1 130 1 261 2 000 2 066 2 326 2 388	0 0 0 0 0 0 0 0 0 0 0	346 411 484 756 819 875 1 179 1 195 1 237	0 0 0 1 1 2 2 3 4 10 12	26 33 42 82 128 163 171 179 207	0 0 0 0 0 0 1 5 7 27 38
Group Mean	426	0	1 441	0	811	3,2	115	7,1
Group Mean Reduction		100%		100%		99,6%		93,8%
Control Median	392		1 261		819		128	
Median x 0,25 x 0,40 x 0,50	98	0/11 exceed 98	315	0/11 exceed 315	205	0/11 exceed 205	32	1/11 exceeds 32
Efficacy Classification		Class A		Class A		Class A		Class A

Only three 3rd stage *D. viviparus* larvae were found at necropsy, all of which were recovered from the mesenteric lymph glands.

Table 4a: *EXPERIMENT 4.* DESIGN OF THE EXPERIMENT ON ACTIVITY AGAINST THE THIRD STAGE OF LARVAE OF *O. RADIATUM, D. VIVIPARUS* AND *COOPERIA* SPP.

Day	Number of infective larvae dosed in each calf		
	O. radiatum	*Cooperia spp.*	*D. viviparus*
-10	224		
- 9	224		
- 8	350		
- 7	350		
- 6	350		
- 5	350		
- 4	350		
- 3	350	2 003	
- 2	207	2 003	785
- 1	207	2 003	
Total	2 962	6 009	785
0	Treat 11 calves dermally with levamisole at 10 mg/kg Slaughter Indicator Control Calf 666		
+26	Slaughter 4 Control Calves		
+27	Slaughter 5 Control Calves		
+28	Slaughter 5 Treated Calves		
+29	Slaughter 5 Treated Calves		

Control and treated calves

The results are summarized in Table 4b.

Control Calves: Mean numbers of 2 242 *Cooperia* spp., 747 *O. radiatum* and 178 *D. viviparus* were recovered from the 9 untreated calves.
Treated Calves: Mean numbers of 25,5 *Cooperia* spp., 393 *O. radiatum* and 36,5 *D. viviparus* were recovered, indicating mean treatment efficacies of 98,9%, 47,4% and 79,5% respectively.

Experiments 5 and 6

Activity was assessed against 3rd stage larvae (Experiment 5) and 4th stage larvae (Experiment 6) of *H. placei.* At necropsy the abomasum and first 5 metres of small intestine of each of the calves were processed for worm recovery.

The design of these experiments is illustrated in Table 5a.

RESULTS

The results are summarized in Table 5b.

Experiment 5

Indicator control calf: Calf No. 966 yielded 118 3rd stage larvae of *H. placei.*
Control and treated calves: Control calves – A mean number of 589 *H. placei* was recovered from the 9 control calves; *Treated calves* – A mean number 165 *H. placei* was recovered, indicating a mean treatment efficacy of 72,0%.

Table 4b: *EXPERIMENT 4.* NUMBER OF WORMS RECOVERED AT NECROPSY, PERCENTAGE REDUCTION IN WORM BURDENS AND EFFICACY CLASSIFICATION

	Cooperia spp. (L_3 stages)		*O. radiatum* (L_3 stages)		*D. viviparus* (L_3 stage –3 days)	
	Control	Treated	Control	Treated	Control	Treated
Control Group = 9 calves Treatment Group = 11 calves	1 496 1 883 2 108 2 150 2 184 2 533 2 600 2 609 2 615	1 2 3 6 9 10 15 38 41 56 100	304 427 542 654 738 793 799 1 025 1 437	218 228 254 276 380 394 456 470 527 555 564	102 124 143 152 154 207 220 241 263	0 0 1 10 15 29 36 37 56 83 134
Group Mean	2 242	25,5	747	393	178	36,5
Group Mean Reduction		98,9%		47,4%		79,5%
Control Mean	2 184		738		154	
Median x 0,25 x 0,40 x 0,50	546	0/11 exceed 546	185 295 369	7/11 exceed 369	39 62	2/11 exceed 62
Efficacy Classification		Class A		Class X		Class B

Table 5a: *EXPERIMENTS 5 & 6.* DESIGN OF THE EXPERIMENTS ON ACTIVITY AGAINST THE THIRD AND FOURTH STAGE LARVAE OF *H. PLACEI*

Day	Number of *H. placei* infective larvae dosed to each calf Experiment 5	Experiment 6
–14		726
–13		493
–12		545
–11		505
–10		505
– 9		505
– 8		493
– 7		470
– 6		470
– 5		484
– 4		384
– 3		384
– 2	2 112	
– 1	2 112	
Total	4 224	5 964
0	Treat 11 calves dermally with levamisole at 10 mg/kg: Slaughter Indicator Control Calf 966.	Treat 11 calves dermally with levamisole at 10 mg/kg: Slaughter Indicator Control Calf 724.
+16		Slaughter 4 Control Calves
+17		Slaughter 5 Control Calves
+19	Slaughter 4 Control Calves	
+20	Slaughter 5 Control Calves	Slaughter 5 Treated Calves
+21	Slaughter 5 Treated Calves	Slaughter 6 Treated Calves
+22	Slaughter 6 T	

Experiment 6

Indicator control calf: Calf No. 724 yielded 2 503 *H. placei,* of which more than 87% were in the 4th stage of larval development.

Control and treated calves: Control calves – A mean number of 3 707 *H. placei* was recovered from the 9 control calves; *Treated calves* – A mean number of 25,9 *H. placei* was recovered, indicating a mean treatment efficacy of 99,3%.

Experiment 7

Activity was assessed against 3rd stage larvae (Group 1), 4th stage larvae (Group 2) and adult stages (Group 3) of *B. phlebotomum.* This experiment was conducted along the lines of Reinecke's[14] "combined trial" using the same group of nine control animals for each of three groups of 11 treated calves.

At necropsy, the abomasum and small intestine of each of the calves and including the trachea and lungs of the indicator control calf No. 127 were processed for worm recovery. The design of this experiment is illustrated in Table 6a.

RESULTS

Indicator control calves

Calf No. 127 slaughtered seven days after infestation (on the day of treatment of Group 1) yielded only one 3rd stage larva from the processed lungs. Calf No. 76 slaughtered 17 days after infestation (on the day of treatment of Group 2) yielded 31 4th stage larvae from the small intestine residue and filtrate.

Table 5b: *EXPERIMENT 5 AND 6.* NUMBER OF WORMS RECOVERED AT NECROPSY, PERCENTAGE REDUCTION IN WORM BURDENS AND EFFICACY CLASSIFICATION

	Experiment 5 *H. placei* (L_3 stage)		Experiment 6 *H. placei* (L_3 stage)	
	Control	Treated	Control	Treated
Control Group = 9 calves Treatment Group = 11 calves	353 415 514 517 555 618 677 822 826	98 114 125 133 153 157 177 181 184 200 295	2 939 3 207 3 582 3 619 3 881 3 946 3 962 4 075 4 156	0 0 1 2 11 12 18 18 28 43 152
Group Mean	589	165	3 707	25,9
Group Mean Reduction		72,0%		99,3%
Control Median	555		3 881	
Median x 0,25 x 0,40 x 0,50	139 222	1/11 exceeds 222	970	0/11 exceed 970
Efficacy Classification		Class B		Class A

Table 6a: *EXPERIMENT 7.* DESIGN OF THE EXPERIMENT ON ACTIVITY AGAINST THE THIRD STAGE LARVAE (GROUP 1), THE FOURTH STAGE LARVAE (GROUP 2) AND THE ADULTS (GROUP 3) OF *B. PHLEBOTOMUM*

Day	Number of infective larvae of *B. phlebotomum* dosed to each calf			
-55	2 000			
-49	Randomization into four groups			
	Control	Treated – Group 1	Treated – Group 2	Treated – Group 3
-48	Slaughter Indicator Control Calf 127	Treat 11 calves dermally: levamisole at 10 mg/kg		
-38	Slaughter Indicator Control Calf 76		Treat 11 calves dermally: levamisole at 10 mg/kg	
-24 -23 -22 -21 -20		Slaughter 5 Treated Calves Slaughter 4 Treated Calves Slaughter 2 Treated Calves	Slaughter 3 Treated Calves Slaughter 4 Treated Calves Slaughter 4 Treated Calves	
0				Treat 11 calves dermally: levamisole at 10 mg/kg
+ 6 + 7 + 8	Slaughter 3 Control Calves Slaughter 3 Control Calves Slaughter 3 Control Calves			
+11 +12 +13				Slaughter 4 Treated Calves Slaughter 4 Treated Calves Slaughter 3 Treated Calves

Control and treated calves

The results are summarized in Table 6b.

Control Calves

A mean number of 125 adult *B. phlebotomum* was recovered at necropsy from the nine untreated calves slaughtered 61 to 63 days after infestion.

Table 6b: *EXPERIMENT 7.* NUMBER OF *BUNOSTOMUM PHLEBOTOMUM* RECOVERED AT NECROPSY, PERCENTAGE REDUCTION IN WORM BURDENS AND EFFICACY CLASSIFICATION

	Controls	*Bunostomum phlebotomum* Group 1 – Dosed (L_3 at 7 days) Treated	Group 2 – Dosed (L_4 at 17 days) Treated	Group 3 – Dosed (Adult at 55 days) Treated
Control Group = 9 calves	46	0	0	0
	52	0	0	0
	78	0	0	0
	100	1	0	0
	107	1	0	0
Treated Groups = 11 calves	112	2	0	1
	116	3	0	1
	161	4	0	1
	352	5	0	4
		55	0	5
		163	0	9
Group Mean	125	21,3	0	1,9
Group Mean Reductions		83,0%	100%	98,5%
Control Median	107			
Median x 0,25	27		0/11 exceed 27	0/11 exceed 27
x 0,40	43	2/11 exceed 43		
Efficacy Classification		Class B	Class A	Class A

Treated Calves

Group 1 – Activity against 3rd stage larvae: Eleven calves treated seven days after infestation and slaughtered 31 to 33 days after infection yielded at necropsy a mean number of 21,3 fifth stage *B. phlebotomum,* representing a mean treatment efficacy of 83,0%.

Group 2 – Activity against 4th stage larvae: None of the eleven calves treated 17 days after infestation and slaughtered 33 to 35 days after infestation yielded at necropsy any *B. phlebotomum,* representing a mean treatment efficacy of 100%.

Group 3 – Activity against adult stages:

Eleven calves treated 55 days after infestation and slaughtered 66 to 68 days after infestation yielded at necropsy a mean number of 1,9 adult *B. phlebotomum,* representing a mean treatment efficacy of 98,5%.

DISCUSSION

The numbers of worms recovered from control animals in these experiments were satisfactory in most cases with mean percentage takes of: *H. placei* – 30% (Range 14 – 62%); *O. ostertagi* – 55% (Range 46 – 64%); *Cooperia* spp. – 34% (Range 25 – 40%); *O. radiatum* – 33% (Range 25 – 42%) and *D. viviparus* – 26% (Range 16 – 40%). The mean percentage take for *B. phlebotomum* was 6,25% (Range 2,3 – 17,6%). Although low the range of total worm recoveries made from the nine control calves was considered satisfactory in view of the considerable variation in individual response to infestation with this species and also bearing in mind that peak infestations are likely to be recorded earlier than 61 to 63 days after infestation. With the exception of *D. viviparus* in Experiment 1 and *B. phlebotomum* in Experiment 7, the recovery of 4th stage larvae from indicator control calves in Experiments 1, 2, 6 and 7 was also satisfactory. Percentage recovery of the six infestations was as follows: *H. placei* – 42%; *O. ostertagi* – 45%; *Cooperia* spp. – 54%; *B. phlebotomum* – 2%; *O. radiatum* – 44% and *D. viviparus* – 6%. The low recovery of 4th stage lungworm and *B. phlebotomum* larvae is probably explained by the fact that most of them were still undergoing tissue migration at the time of slaughter. Recovery of 3rd stage larvae from the indicator control calves in Experiments 2, 4, 5 and 7 was disappointing with percentage takes of: *H. placei* – 3%; *O. ostertagi* – 10%; *Cooperia* spp. – 11%; *B. phlebotomum* – 0,05%; *O. radiatum* – 9% and *D. viviparus* – 0,4%. Bearing in mind that recoveries from the control calves infested at the same time as these indicator controls were generally satisfactory, these poor results suggest that the techniques adopted for larval recovery were not sufficiently sensitive. The very poor recovery of lungworm larvae in Experiment 4 and *B. phlebotomum* larvae in Experiment 7 is again probably explained by the fact that the larvae would all have been in the process of tissue migration at the time of slaughter and would therefore have been difficult to locate. Only approximately 6% of the *Cooperia* spp. larvae recovered from the indicator control calf in Experiment 4 were in the expected 3rd stage of larval development, the remainder being early 4th stage larvae. As infective larvae had been administered to this calf at one, two and three days before slaughter the only conclusion that can be drawn is that the worms recovered at nectopsy were those given on the first day, the remainder having been retained in the fore-stomachs. The majorities of all other larvae recovered from each of the indicator control calves were in the expected stages of development.

The results obtained in these experiments confirm the previous reports by Brooker & Goose[3], Dorn & Federman[5], Curr[4] and Kingsbury[11]. Unlike earlier re-

ports of anthelmintic activity following topical application of other drugs (Knapp *et al*[12] and Poole & Dooley[13]), results consistent with the known efficacy of levamisole have been achieved in these investigations, all the results of which have been based on necropsy findings. The efficacy of levamisole following dermal application at the rate of 10 mg levamisole base per kg livemass has been shown to be equivalent to that recorded in the extensive literature following the use of more orthodox methods of administration at dose rates of between 7,5 and 10,0 mg levamisole HC1 per kg livemass.

These experiments were designed in such a way as to enable the results obtained to be evaluated by the modified non-parametric method originally devised by Groeneveld & Reinecke[8] and cited by Reinecke[14]. This method of evaluation permits anthelmintic efficacy to be classified according to the following system: *Class A* implies that treatment is more than 80% effective in more than 80% of the treated herd; *Class B* implies that treatment is more than 60% effective in more than 60% of the treated herd; *Class C* implies that treatment is more than 50% effective in more than 50% of the treated herd and *Class X* implies that treatment is ineffective.

Such analysis of the results obtained in these experiments shows that the anthelmintic efficacy of levamisole at 10 mg base per kg livemass in the solvent system used when applied dermally to cattle against the developmental stages of the six species examined, is as follows:

GRADES OF CLASSIFICATION FOR EFFICACY AGAINST

	3rd larval stages	4th larval stages	5th larval & adult stages
H. placei	B	A	A
O. ostertagi	B	X	B
Cooperia spp.	A	A	A
B. phlebotomum	B	A	A
O. radiatum	X	A	A
D. viviparus	B	A	B A

The ability to control round-worm infestations by "pour-on" medication adds a new dimension to the use of anthelmintics in general and to therapy with levamisole in particular. This development should lead to greater convenience and ease of administration under all systems of herd management.

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dri·staf

staphylococcal mastitis vaccine

TRIALS WITH RAFOXANIDE
8. EFFICACY OF AN INJECTABLE SOLUTION* AGAINST TREMATODES AND NEMATODES IN CATTLE

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ABSTRACT: Schröder, J.; Honer, M.R.; Louw, J.P. **Trials with rafoxanide 8. Efficacy of an injectable solution against trematodes and nematodes in cattle.** *Journal of the South African Veterinary Association* (1977) **48** No. 2, 95 – 97 (En) MSD Research Centre, Private Bag 3, 1685 Halfway House, Rep. of South Africa.

Four experiments are described in which the efficacy of an experimental 5% injectable solution of rafoxanide was evaluated against various adult and immature helminths in cattle. Subcutaneous injection at a dosage of 3 mg/kg live mass resulted in the following reductions in mean worm burdens: adult *Fasciola hepatica,* 82,6%; adult *Fasciola gigantica,* 99,8% immature *Paramphistomum microbothrium,* 10,1% adult *Haemonchus placei,* 99,6%, third stage *H. placei,* 73,7%; adult *Bunostomum phlebotomum,* 99,8%; adult *Oesophagostomum radiatum,* 99,9%; and fourth stage *O. radiatum,* 76,9%. At 5 mg/kg live mass, rafoxanide solution was 97,5% and 99,2% effective against 8-week old *F. gigantica* and third stage *H. placei* respectively and at 7,5 mg/kg, 92,4% against 6-week old *F. gigantica.*

INTRODUCTION

Previous papers in this series have reported the efficacy of rafoxanide {3,5 – diiodo – 3′ – chloro – 4′ – (p-chloro-phenoxy) – salicylanilide} as a preformed suspension* against natural and artificial infestations of adult and immature *Fasciola gigantica, Fasciola hepatica,* adult and fourth stage *Haemonchus placei,* and adult *Bunostomum phlebotomum* in cattle at dosages ranging from 3,75 to 11,25 mg/kg live mass[6 7 9] Efficacy has also been described against *F. hepatica, F. gigantica, Paramphistomum microbothrium, Haemonchus contortus, Gaigeria pachyscelis, Chabertia ovina* and *Oestrus ovis* in sheep[1 2 8] and against *Gedoelstia hässleri* in blesbuck[5].

This paper describes four experiments in which the efficacy of subcutaneous injections of an experimental 5% solution of rafoxanide against adult and/or immature artificial infestations of *F. gigantica, F. hepatica, P. microbothrium, H. placei, B. phlebotomum* and *Oesophagostomum radiatum* was evaluated according to the non-parametric method[3].

MATERIALS AND METHODS

The times of infestation, treatment and slaughter are summarized in the Experimental Designs in Table 1.

Experimental animals and infestations

Seventy one crate-reared Friesian-cross bullocks, weaned at the age of 2 months and varying in age from 3 to 6 months, were used in the four experiments. Infestations with *H. placei* and *O. radiatum* were performed orally, as were the trematode infestations. *B. phlebotomum* infestations were effected by the percutaneous route.

Treatments

In each trial a minimum of 11 and five animals were randomly allocated to the treated and control groups respectively. Treatment was by subcutaneous injection 5 to 10 cm anterior to the left scapulo-humeral joint.

Experiment 1:

Treated at 3 mg/kg against adult *F. hepatica* (84 days) and *B. phlebotomum* (60 days) and fifth stage and adult *H. placei* (15-31 days) and *O. radiatum* (21-40 days).

Experiment 2:

Treated at 3 mg/kg when the *F. gigantica* infestation was adult (98 days), *P. microbothrium* immature (6, 12 and 18 days), *H. placei* in the third larval stage (1-2 days) and *O. radiatum* in the fourth larval stage (11-20 days).

Experiment 3:

Treated at 5 mg/kg when the *F. gigantica* were 56 days old and the *H. placei* 1-2 days old (third stage larvae).

Experiment 4:

Treated at 7,5 mg/kg when the *F. gigantica* were 42 days old, the *B. phlebotomum* adult, and the *O. radiatum* fifth stage and adult worms.

Helminth recovery

Necropsies and worm recoveries were carried out by methods described previously[6]. All helminths were counted macroscopically and removed from the ingesta, of which 1/10th aliquots were then examined microscopically and any additional worms recovered recorded, except in Experiment 4, in which only macroscopic counts were performed. In Experiment 2, the ruminal contents were collected and the rumen walls carefully washed. All macroscopically visible conical fluke were collected for counting. One tenth aliquots of the ruminal ingesta were examined macroscopically for paramphistomes and the rest discarded.

RESULTS

The ranked worm burdens with percentage reductions and non-parametric label claims for the four experiments are presented in Table 2.

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Table 1: EXPERIMENTAL DESIGNS

Number of metacercariae or infective larvae administered													
Experiment		1			2				3		4		
Species	*F. hepatica*	*H. placei*	*B. phlebotomum*	*O. radiatum*	*F. gigantica*	*H. placei*	*P. microbothrium*	*O. radiatum*	*F. gigantica*	*H. placei*	*F. gigantica*	*B. phlebotomum*	*O. radiatum*
Day –98 –84 –60 –56 –42	300		1930		300				200		200	2800	
–40 to –6		150/day days –31 to –28 & –26 to –15		60/day days –40 to –21			500/day days –18, –12 & –6	150/day days –20 to –11					60/day days –40 to –21
–2 –1						1000 1000				1510 1510			
TOTAL	300	2 400	1930	1200	300	2000	1500	1500	200	3020	200	2800	1200
Day 0	Treat with rafoxanide at												
	3 mg/kg				3 mg/kg				5 mg/kg		7,5 mg/kg		
14 to 16 28 to 32 35 & 36 45 to 48	Slaughter				Slaughter				Slaughter		Slaughter		

Table 2: RANKED WORM BURDENS AND EFFICACIES

The efficacy of an injectable solution of rafoxanide against various helminths in cattle													
Experiment and dosage	1 3 mg/kg				2 3 mg/kg				3 5 mg/kg		4 7,5 mg/kg		
Parasite and age at treatment	*F. hepatica* Adult	*H. placei* Adult	*B. phlebotomum* Adult	*O. radiatum* Adult	*F. gigantica* Adult	*H. placei* 1-2 days	*P. microbothrium* Immature	*O. radiatum* 11-20 days	*F. gigantica* 8 weeks	*H. placei* 1-2 days	*F. gigantica* 6 weeks	*B. phlebotomum* Adult	*O. radiatum* Adult
Control	129	979	155	521	125	1091	916	460	821	1261	122	209	859
	82	970	105	470	92	980	882	432	143	1257	113	177	827
	81	833	98	452	90	943	798	378	111	1232	103	160	759
	64	736	96	417	73	775	747	342	111	1211	100	144	742
	62	676	80	416	72	743	659	239	106	879	92	75	694
	55	500	76	362	42	682	145	95	92	742	64	72	671
	11	163	30	295	—	—	—	—	—	—	50	66	466
Mean	69	694	91	419	82	869	691	324	103	1097	92	129	717
Treated	113	33	2	1	2	740	1072	141	13	34	18	0	2
	5	2	0	0	0	531	865	115	4	32	12	0	2
	3	1	0	0	0	295	722	107	4	11	10	0	1
	3	1	0	0	0	242	670	101	3	6	8	0	0
	2	0	0	0	0	232	618	98	2	3	6	0	0
	2	0	0	0	0	225	612	75	2	2	6	0	0
	1	0	0	0	0	84	514	45	1	1	5	0	0
	1	0	0	0	0	71	501	45	0	1	3	0	0
	1	0	0	0	0	52	468	42	0	1	2	0	0
	0	0	0	0	0	38	397	30	0	0	2	0	0
	0	0	0	0	0	0	393	23	0	0	0	0	0
Mean	12	3	<1	<1	<0,2	228	621	75	3	8	7	0	<1
%	82,6	99,6	99,8	99,9	99,8	73,7	10,1	76,9	97,5	99,2	92,4	100	99,9
NPM Claim	A	A	A	A	A	B	X	B	A	A	A	A	A

At 3 mg/kg live mass, the rafoxanide solution was highly effective (≥ 80%) against adult infestations of *F. hepatica, F. gigantica, H. placei, B. phlebotomum* and *O. radiatum,* moderately effective (70-79%) against third stage *H. placei* and fourth stage *O. radiatum,* but ineffective against immature *P. microbothrium.* A dosage of 5 mg/kg gave high efficacy against 8-week old *F. gigantica* and third stage *H. placei,* whereas 7,5 mg/kg was highly effective against 6-week old *F. gigantica.*

DISCUSSION

Rafoxanide is effective against internal parasites which consume blood and tissue[1 2 5 6 7 8 9]. For this reason it was decided to include *O. radiatum* in the present infestations. The results of Experiments 1 and 2 confirm that this parasite also falls within this category.

The inefficacy against immature *P. microbothrium* in Experiment 2 probably indicates that this parasite neither sucks blood, nor feeds on its host's intestinal mucosa. The efficacy of rafoxanide at 15 mg/kg against immature *P. microbothrium* in sheep[2] could indicate that at this dosage the drug is excreted into the gut lumen in quantities lethal to the parasite. Similarly, its efficacy against *O. ovis*[1 8] seems to suggest that the drug is excreted in the nasal mucus. If this is indeed the case, continuous partial reabsorption could in some way account for the residual effect against reinfestation by *O. ovis* and egg-production by *H. contortus*[1 8].

The compound's efficacy against haematophagous and 'histophagous' parasites is probably due to the high blood-levels which are obtained within 24 hours, and which show a gradual decline from about the fourth day onwards[4]. Attempts in this laboratory to demonstrate efficacy against trichostrongylid nematodes, other than those already mentioned, have mostly been unsuccessful.

With the exception of *P. microbothrium,* the results of the present experiments show that rafoxanide is effective against the most important internal parasites of cattle in the greater part of South Africa. The geographical distribution of *Ostertagia* spp. is confined to a relatively small region of the country whilst *Cooperia* spp., although ubiquitous and usually present in large numbers, are not very pathogenic.

Rafoxanide in solution is more effective when injected subcutaneously, than is the suspension when administered by the oral or intraruminal routes. Thus 3 mg/kg subcutaneously was more effective than 7,5 mg/kg intraruminally[6] against adult *B. phlebotomum.* Similarly, 5 mg/kg subcutaneously was more effective against 8-week old *F. gigantica* than 11,25 mg/kg administered orally[7]. The above, and the almost complete efficacy against third stage *H. placei* at 5 mg/kg makes it probable that dosages in excess of this level will seldom be necessary.

As can be seen from Table 2, the numbers of *B. phlebotomum* recovered from the untreated animals were small in comparison with the numbers of larvae given. The virtual complete absence of these worms from the treated animals, however, precluded the necessity for large worm burdens in the controls. These results are in accordance with those previously observed with this nematode[6].

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HAEMATOLOGICAL CHANGES CAUSED BY *TRICHOSTRONGYLUS COLUBRIFORMIS* IN LAMBS FED A DYSTROPHOGENIC DIET

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ABSTRACT: Horton G.M.J.; Owen N.C.; Horak I.F.; Schröder J. **Haematological changes caused by** *trichostrongylus colubriformis* **in lambs fed a dystrophogenic diet.** *Journal of the South African Veterinary Association* 1977) **48** No. 2, 99 – 103 (En) Dept. Anim. Science, Univ. of Saskatchewan, Saskatoon, Canada S7N OWO

The effect of *Trichostrongylus colubriformis* on lambs maintained on a ration containing a low level of selenium and on animals receiving vitamin E and Se supplementation was investigated. The pathological changes seen in control animals slaughtered at the start of the experiment and in the animals which died during the course of the investigation revealed a high level of nutritional muscular dystrophy (NMD) in the lambs. There were no marked haematological changes in the control or infested sheep. Infestation was characterized by slight hypoalbuminaemia and gamma-globulinaemia. Serum levels of the enzymes AAT and CPK, which are important indicators of muscle necrosis and NMD, were greatly increased in sheep infested with *T. colubriformis* and not receiving supplementary Vit. E + Se. Data from this study therefore indicates that trichostrongylosis may aggravate the degree of muscle necrosis in lambs prone to the development of NMD.

INTRODUCTION

Haematological changes have been reported for lambs infested with *Trichostronglylus colubriformis*[8] and selenium has been shown to have a specific enzyme function in sheep erythrocytes[7]. Defects in both vitamin E and selenium metabolism are believed to be aetiological factors in nutritional muscular dystrophy (NMD) of lambs[13]. NMD may be diagnosed by routine serum enzyme analyses[3]. The possibility that verminosis may complicate the diagnosis warrants investigation.

In this report the pathological, haematological and serum enzyme changes in lambs fed a dystrophogenic diet and infested with *T. colubriformis* were investigated.

MATERIALS AND METHODS

Sixty-one 3 month (Experiment 1) and thirty 5 month (Experiment 2) Merino lambs, reared worm-free at the MSD Research Centre, Hennops River, were maintained on a ration of lucerne hay obtained from the Vaal Hartz area which is known to be deficient in selenium. Selenium and vitamin E levels in the lucerne diets were 0,028 and 34,1 ppm in Exp. 1 and 0,039 and 26,4 ppm in Exp. 2 respectively. Necropsies were performed on some of the animals in Exp. 1 and haematological and blood enzyme studies were done on the animals in Exp. 2. The general layout of the experiments is summarized in Tables 1 and 2. The duration of both experiments was 98 days.

Infestation

Lambs were infested with 100 000 infective larvae of *T. colubriformis* at the rate of 20 000 larvae per week for 5 weeks in Exp. 1 and with a single dose of 50,000 larvae in Exp. 2. Infestation was established by oral drenching with larvae obtained from the faeces of donor sheep infested with a pure culture of *T. colubriformis*.

Table 1: EXPERIMENTAL DESIGN, EXPERIMENT 1

Groups	No. sheep per group	Treatments	Challenge 100,00 *T. colubriformis*	Investigations
Pre-exp.	5	—	No	Pathology (5 lambs)
1	7	—	No	—
2	7	—	Yes	Pathology (3 lambs)
3	7	Se*	No	—
4	7	Se	Yes	—
5	7	Vit. E†	No	Pathology (2 lambs)
6	7	Vit. E	Yes	Pathology (4 lambs)
7	7	Se* + Vit. E†	No	—
8	7	Se + Vit. E	Yes	Pathology (1 lamb)

*3 mg *per os* at 14 day intervals
†450 IU *per os* at 14 day intervals

Table 2: EXPERIMENTAL DESIGN, EXPERIMENT 2

Groups	No. sheep per group	Treatments	Challenge 50,000 *T. colubriformis*	Investigations
1	7	—	No	Haematology, serum proteins and enzymes
2	8	—	Yes	Haematology, serum proteins and enzymes
3	7	Vit. E + Se*	No	Haematology, serum proteins and enzymes
4	8	Vit. E + Se	Yes	Haematology, serum proteins and enzymes

*450 IU Vit. E + 3 mg Se *per os* at 14 day intervals

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Table 3: INCIDENCE OF DEGENERATIVE LESIONS IN SKELETAL AND CARDIAC MUSCULATURE FROM LAMBS IN EXPERIMENT 1

Treatment	Lamb no.	Died days after infestation	Gross lesions*		Micro lesions*	
			Skeletal	Myocardial	Skeletal	Myocardial
Pre-experimental	I	0		+		
slaughtered group	II	0			+	+
	III	0			++	
	IV	0				
	V	0		+++		+++
Non-infested + vit. E	G154	42		+		++
	G158	14	++	++	+	
Infested control	Y2	46		++		
	Y3	42			+	
	Y5	35	+		++	
Infested + vit. E	G9	42				+
	G10	42			++	
	G15	35			+	
	G16	14			+	++
Infested + Vit. E + Se	B27	42			+	

*Severity of lesions: + light; ++ moderate; +++ severe

Vitamin E and selenium

Vitamin E and selenium supplementations were as follows: vitamin E – 450 IU vitamin E*; selenium – 3 mg Se as Na_2SeO_3. 5 H_2O; vitamin E plus selenium (Vit. E + Se) – 450 IU vitamin E plus 3 mg Se. The supplements were administered as an oral drench every 14 days throughout both experiments.

Pathology

Necropsies were carried out on five randomly selected pre-experimental lambs and a further 10 (two non-infested and eight infested) that died during the first 49 days of Exp. 1 (when all lambs were treated with thiabendazole). Specimens of right ventricle wall, *Musculus semimembranosus* and *M. supraspinatus* were fixed in 10% formalin for histopathological examination. Histological sections were stained with haematoxylin and eosin. Degenerative lesions in the wall of the right ventricle, or either of the skeletal muscle samples were considered as indicative of NMD[1 10]. Bilateral symmetry of skeletal muscle lesions was not determined microscopically.

Haematology

In Exp. 2 venous blood samples were withdrawn from the jugular vein into evacuated 10 mℓ glass tubes (Venoject, Comopharm) every 2 weeks at 07 00 hours, immediately prior to the morning feed. Both heparinized samples for haematological studies and samples without anticoagulant for serum enzyme determinations were collected on the same day.

Haemoglobin (Hb) concentrations were measured using a haemoglobinometer (Coulter Electronics), while erythrocyte counts, mean corpuscular volumes (MCV), haematocrits and total leucocyte counts were determined by Coulter Counter*. The methods used were those given in the manufacturer's instruction manuals.

Total serum proteins (TSP) were determined by the Biuret method[18]. The protein fractions were calculated from cellulose acetate electrophoretograms[16] after integration using a Beckman Densitometer.

Serum enzyme determinations

Aspartate aminotransferase (AAT) activity was measured at 25° C[11] and creatine phosphokinase (CPK) by the method of Wiesmann Colombo, Adam & Richterich[17].

Statistical analysis

Analysis of variance and mean comparisons by Scheffe's test[14] were done using an IBM 350 computer.

RESULTS

Pathology

Bilaterally symmetrical pale streaks, diagnosed as degenerative lesions, were seen macroscopically in the semimembranous muscles of two (one infested and one non-infested) lambs. Greyish-white subendocardial plaques of varying size were grossly detected in the right ventricular walls of five (two pre-experimental, two non-infested and one infested) lambs (Table 3). However, histological examination of the sections from these sites failed to confirm the diagnosis in two of the five cases. Microscopic examination revealed focal hyaline degeneration and necrosis in eight (two pre-experimental and six infested) lambs where no lesions could be detected grossly in either the cardiac or skeletal muscles. The microscopic lesions varied from focal disseminate swollen, homogeneously eosinophilic muscle fibres to frank necrosis followed by fragmentation of the necrotic tissue, and attempted regeneration.

*Rovimix E Type 20W, F. Hoffmann – La Roche and Co., Ltd.
*Model FN, Coulter Electronics.

Table 4: MEAN RESULTS OF HAEMATOLOGICAL INVESTIGATIONS ON LAMBS IN EXPERIMENT 2

	Non-infested		Infested		
	Control	Vit. E + Se	Control	Vit. E + Se	SE
Haemoglobin concentration (g/dℓ)	9,64[a]	10,20[b]	9,85[ab]	9,41[ab]	0,17
Erythrocyte count (x10⁶/mℓ)	6,80	7,23	7,14	6,80	0,17
Mean corpuscular volume (fℓ)	31,90	32,20	32,50	32,00	0,22
Haematocrit (%)	28,30[ab]	29,40[a]	30,20[a]	26,90[b]	0,52
Leucocyte count (x10³/mℓ)	8,09[a]	8,34[a]	10,14[b]	9,14[ab]	0,41

Means with in the same line bearing different superscripts differ significantly ($P < 0.05$). An [ab] superscript denotes that the value does not differ significantly from values bearing superscripts [a,b]

Table 5: MEAN PLASMA PROTEIN LEVELS OF LAMBS IN EXPERIMENT 2

	Non-infested		Infested		
	Control	Vit. E + Se	Control	Vit. E + Se	SE
Total serum protein (g/dℓ)	7,46[a]	7,46[a]	6,68[b]	7,39[a]	0,11
Albumin (g/dℓ)	3,94[a]	4,00[a]	3,25[b]	3,49[ab]	0,10
α – Globulin (g/dℓ)	1,24	1,24	1,19	1,23	0,03
β – Globulin (g/dℓ)	0,37	0,39	0,44	0,42	0,01
γ – Globulin (g/dℓ)	1,91[ab]	1,83[a]	1,93[ab]	2,26[b]	0,08
Albumin: Globulin ratio	1:0,91[a]	1:0,87[a]	1:1,32[b]	1:1,15[ab]	0,07

Means within the same line bearing different superscripts differ significantly ($P < 0.001$). An [ab] superscript denotes that the value does not differ significantly from values bearing superscripts [a,b]

Haematology

Hb, erythrocyte and haematocrit values were highest for non-infested lambs receiving Vit. E + Se, and lowest for infected lambs receiving Vit. E + Se or infec- MCV was not affected by either Vit. E + Se or infection. Leucocyte counts were higher in non-supplemented, infested lambs than in either of the non-infested groups.

Serum proteins

TSP and albumin concentrations were depressed by about 14% ($P < 0,001$) in control infested lambs as compared to non-infested animals (Table 5), and while alpha-globulin levels were similar in all treatment groups, beta- and gamma-globulin fractions were higher in infested animals. Consequently, albumin to globulin ratios were lowest for infested lambs, and ranged from 1:0,87 (non-infested – Vit. E + Se) to 1:1,32 (infested-control). In the non-infested groups there was little difference between the protein levels in control lambs and those receiving Vit. E + Se supplement but in infested animals those receiving supplement had significantly higher gamma globulin and TSP levels ($P < 0,001$).

Serum enzymes

AAT and CPK values were significantly ($P < 0,001$) higher in unsupplemented lambs infested with *T. colubriformis* than in either the control or the supplemented non-infested groups. Enzyme activities followed similar trends during the course of the experiment, and reached peak values, 42 (CPK) and 56 days (AAT) post-infestation (Figs. 1 & 2).

DISCUSSION

Vit. E + Se supplementation was characterised by a mild increase in erythrocyte counts, haematocrits and haemoglobin concentrations in non-infested lambs, and since the MCV was unchanged, it may be concluded that the increase in both haemoglobin and haematocrit values merely reflected the increase in erythrocyte numbers. The reverse situation was observed in infested lambs. These changes were not large and their biological significance is questionable. Serum AAT and CPK concentrations for non-infested lambs were consistently lower in the Vit. E + Se group, than the controls and although these changes were not statistically significant ($P < 0,05$), they are indicative of a beneficial effect due to the supplement, and suggest a marginal selenium deficiency in the diet. This conclusions is supported by the finding of overt myopathy (NMD) in Experiment 1, and the established role of vitamin E and selenium in the aetiology of NMD[2,12].

The failure of infestation with *T. colubriformis* to give rise to significant haematological changes apart from a reduced haematocrit and a mild leucocytosis is consistent with the findings of Horak, Clark & Gray[8]. The slight hypoalbuminaemia, gamma-globulinaemia and the consequently decreased albumin to globulin ratios are similar to those reported for kids[6] and for lambs[8] with trichostrongylosis. The hypoalbuminaemia was probably the result of albumin seepage into the

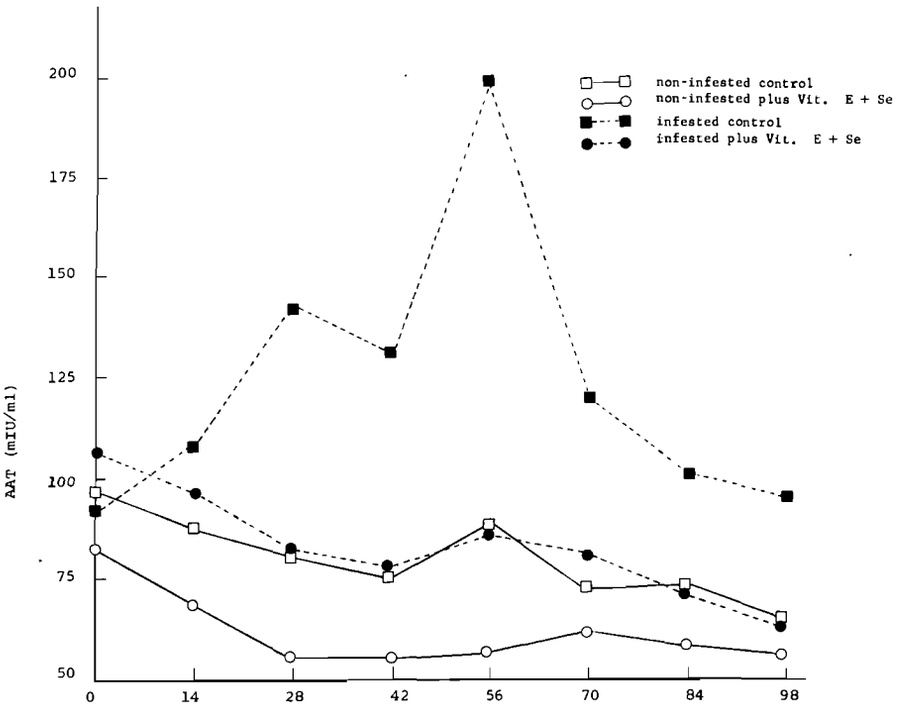

Fig. 1 Serum AAT levels following infestation with *T. colubriformis* and supplementation with selenium and vitamin E.

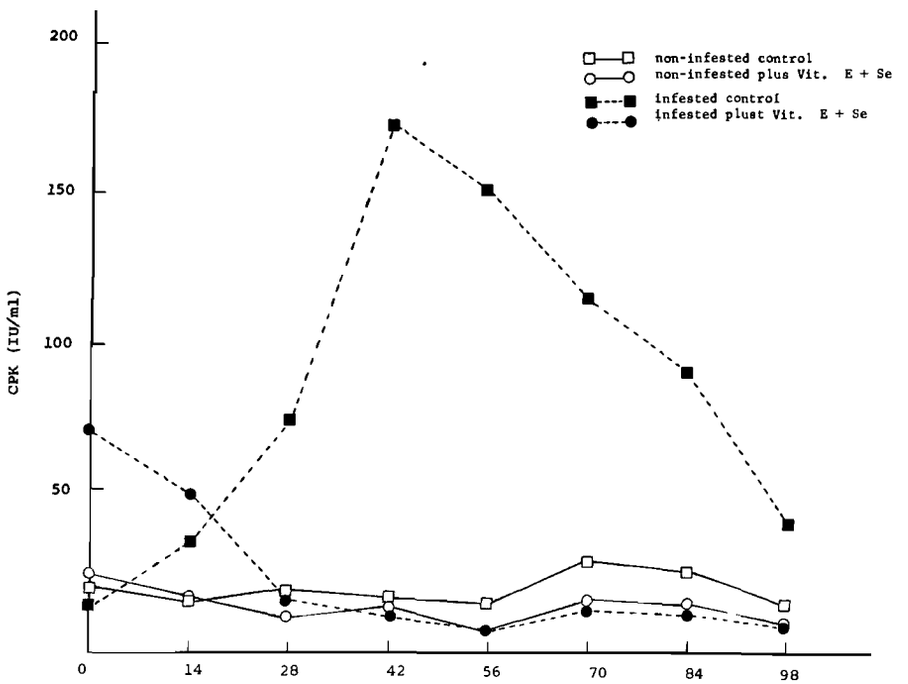

Fig. 2 Serum CPK levels following infestation with *T. colubriformis* and supplementation with selenium and vitamin E.

intestinal lumen via lesions produced by the parasites[15]. Increased gamma-globulinaemia together with evidence of decreased worm burdens as judged by faecal egg counts in groups receiving Vit. E + Se[9] suggests that the supplement may have enhanced the immune response of the lambs to infestation.

The marked increases in serum AAT and CPK concentrations in the infested sheep are of particular interest. Previous studies have shown that nutritional myopathies in lambs are associated with increased concentrations of both serum AAT and CPK[5], and appear to be directly related to the extent of muscular

damage[4]. It therefore appears that *Trichostrongylus* infestation may exacerbate the effects of a selenium deficiency in lambs. This conclusion is supported by the finding that supplementation of similarly infested sheep with selenium and vitamin E decreased the concentrations of these serum enzymes to control values. Unfortunately, this hypothesis could not be fully substantiated by pathological data, since lambs were not sacrificed at the conclusion of the experiments. However, of the eight infested lambs that died, and in which lesions characteristic of NMD were detected, only one had received supplementary Se. Vit. E did not appear to prevent the development of lesions in either non-infested or infested lambs.

ACKNOWLEDGEMENTS

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BOOK REVIEW

BOEKRESENSIE

THE BUFFALOES OF CHINA

W. ROSS COCKRILL

Food and Agriculture Organization of the United Nations. Rome 1976
pp VII 96 Figs 37 (17 Colour) Tabs 7, Publ. Price U.S. $8.00

This is a supplement to the book "The Husbandry and Health of the Domestic Buffalo" by W. Ross Cockrill published by the FAO of the United Nations. It deals with the buffaloes of China which were not described in this book. There is a complete account of the buffaloes of Taiwan and China.

Particulars about their distribution, characteristics, appearance, size, type, production, breeding, feeding and management are given. Their uses and potential are discussed. There is a final chapter on the development of agriculture, animal disease control and medical services in China which includes veterinary and animal husbandry training. The book is clearly printed on good paper. The illustrations are interesting and informative. There is a bibliography and index.

G D S

THE GASTROSCOPE AS AN AID IN VETERINARY DIAGNOSTICS

S.W. PETRICK*

ABSTRACT: Petrick, S.W. **The gastroscope as an aid in Veterinary diagnostics.** *Journal of the South African Veterinary Association* (1977) **48** No. 2, 105 – 107 (En) Dept. Surgery, Faculty of Veterinary Science, University of Pretoria, Box 12580, 0110 Onderstepoort, Rep. of South Africa.

The gastrointestinal fibrescope is introduced to the veterinary profession and its uses are emphasised. Various anatomical structures were examined in 251 domestic mammals and two birds. The findings for referred cases and routine examinations are listed. Twenty colour pictures taken with the fibrescope are reproduced.

INTRODUCTION

The first gastroscopic examination was performed by Adolf Kussmaul in 1868[1]. The instrument consisted of a metal tube and the patient none more suitable than a sword swallower. Since then the instrument has been frequently modified until the first gastrointestinal fibrescope was manufactured in 1958[1]. This flexible endoscope with its optical fibres can, with certain exceptions, be used in any patient without fear of internal damage.

This paper describes the use of a gastrointestinal fibrescope in several species of domestic mammals and birds and points out the range of possible applications of this valuable diagnostic instrument when the correct size is chosen.

APPARATUS

Olympus Gastrointestinal Fibrescope, model G.I.F. type P. (pediatric) Specifications: Insertion tube with an outer diameter of the distal end of 7,2 mm and a working length of 1,1 meter.

Control unit: Flexible section with a length of 60 mm. Flexion angle 150° from axial position. Control for air-water administration and air-water suction.

Accessories: Biopsy forceps; cytology brush.

Optional accessories: Olympus Photographic apparatus, model OM–1, standard set.

MATERIAL

The fibrescope was used for the endoscopic examination of 251 domestic mammals and two birds over a period of 10 months. Thirteen animals were examined more than once. Table 1 details the species that were examined as well as the total number of referred cases and animals subjected to routine examination. The 154 dogs and 11 cats were routinely examined directly after surgery.

Table 1: DETAILS OF THE SPECIES EXAMINED AND THE TOTAL NUMBER OF REFERRED CASES AND ROUTINE EXAMINATIONS

Species	Referred cases	Routine examinations	Total
Horses	12	23	35
Cattle	3	2	5
Sheep	1	—	1
Dogs	41	154	195
Cats	3	11	14
Chimpanzee	1	—	1
Birds	—	2	2

METHODS

Horses were examined without difficulty by passing the endoscope through the nasal passage. In some cases a slight cough occurred when the instrument entered the trachea. A twitch was the only necessary means of control.

Cattle sneezed tremendously while the scope passed the anterior half of the nasal passage and it is thus considered advisable to cover the nostrils with a cloth. A nosering or pliers are necessary for proper restraint.

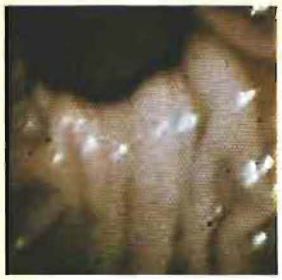
The Karakul ram was placed on a table and examined by holding its head for introduction of the instrument via the nasal passage.

The dogs, cats, the chimpanzee and the two birds were examined under general anaesthesia. In these cases the endoscope was passed orally.

The various anatomical structures that were examined are summarised in Table 2.

Table 2: THE VARIOUS ANATOMICAL STRUCTURES EXAMINED IN EACH SPECIES

Species	Nasal passage	Larynx(L) Trachea(T) Bronchus(B) Oesophagus(O)	Pharynx(P) triculus	Provent- Stomach	bulb	Duodena Cervix	Vagina Uterus	u
Horses	35	35L 10T	35P 6O	—	—	—	—	—
Cattle	5	5L 1T	5P	—	—	—	—	
Sheep	1	1L	1P	—	—	—	—	—
Dogs	—	129LTB	192PO	—	192	4	37	1
Cats	—	6L	14PO	—	14	1	—	—
Chimpanzee	—	—	1PO	—	—	—	—	—
Birds	—	—	2PO	1	2	—	—	—



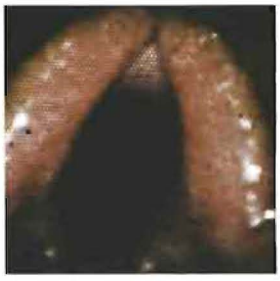
1. Dog
Stomach

2. Dog
Cardia

3. Dog
Pylorus

4. Dog
Trachea

5. Horse
Cardia

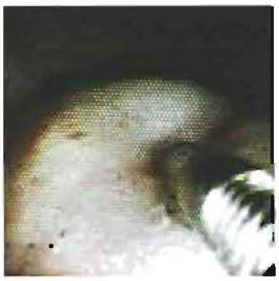


6. Horse
Larynx

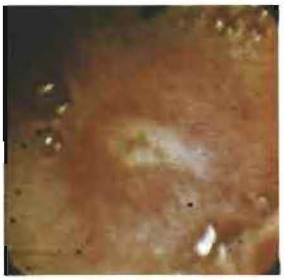
7. Cat
Oesophagus

8. Dog
Oesophagus

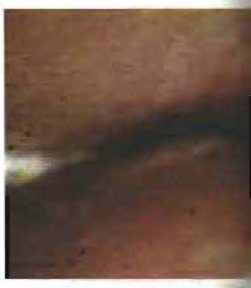
9. Dog
Oesophagus



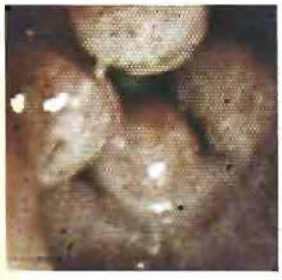
10 Dog
Biopsy forceps



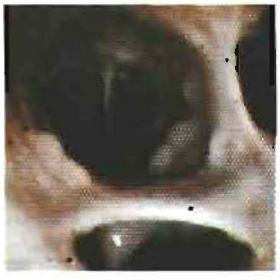
11. Dog
Stomach



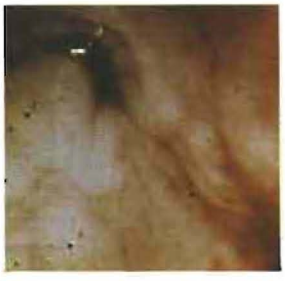
12. Dog
Trachea



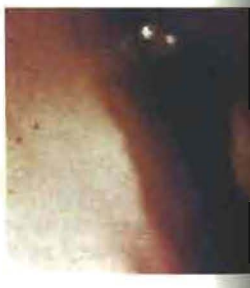
13. Dog
Trachea



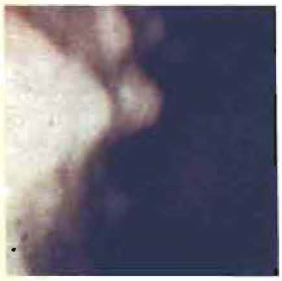
14. Dog
Bronchus



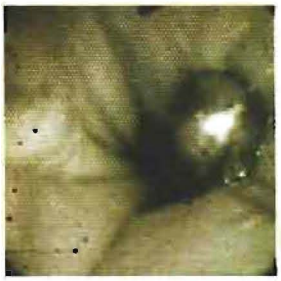
15. Dog
Vagina



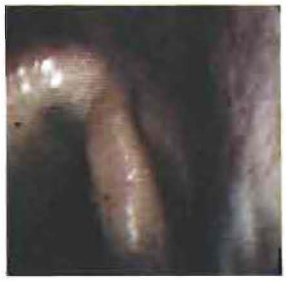
16. Cow
Trachea



17. Horse
Pharynx



18. Horse
Oesophagus



19. Horse
Larynx

20. Ram
Larynx

Plate 1. TWENTY PICTURES TAKEN WITH OLYMPUS FIBRESCOPE

Plate 1 shows 20 pictures taken with Olympus Fibrescope coupled to a model OM–1 Olympus camera.

1. Dog – Normal gastric mucosa and antrum peristalsis
2. Dog – Cardia and scope with retroversion
3. Dog – Dilated pylorus
4. Dog – Normal trachea with exceptionally large ridge
5. Horse – Dilated cardia
6. Horse – Normal larynx
7. Cat – Oesophageal obstruction
8. Dog – Oesophageal obstruction with foreign body
9. Dog – Oesophagus with *Spirocerca lupi* granuloma
10. Dog – *Spiirocerca lupi* granuloma with biopsy forceps
11. Dog – Gastric ulcer
12. Dog – Collapsed trachea
13. Dog – Trachea with *Filaroides osleri* lesions
14. Dog – Bronchiectasis
15. Dog – Cervix and vagina with blood
16. Cow – Collapsed trachea
17. Horse – Follicular pharyngitis
18. Horse – Oesophageal fistula with probe
19. Horse – Laryngeal defect, compare with No 6
20. Ram – Laryngeal defect, compare with No 6

RESULTS

Even routine examinations can lead to an early diagnosis of unsuspected conditions. The following conditions were diagnosed in the course of routine examination of dogs:

1. *Spirocerca lupi* infestation.
2. Enlarged oesophagus.
3. Gastritis.
4. Gastric erosion.
5. Gastric ulcer.
6. Pin-point bleeding of gastric mucosa.
7. Giant folds of gastric mucosa.
8. Ascarid worms in duodenum.
9. Tracheitis.
10. Free blood in vagina after oophorectomy.

Most riding and racehorses showed some development of follicles in the pharyngeal mucosa. The development in some cases was such as to make it difficult to decide whether or not they were cases of follicular pharyngitis.

Of the 61 referred cases a few turned out to be normal on examination. Listed below are the findings in the various species.

Horses

1. Structural defect of larynx
2. Free blood in trachea
3. Follicular pharyngitis
4. Oesophageal fistula
5. Osteitis in nasal passage

Dogs

1. Ectopic tonsil
2. Achalasia
3. Oesophageal obstruction
4. Free blood in stomach
4. Gastritis
6. Laryngeal paralysis
7. Tracheitis and bronchitis
8. *Filaroides osleri* infestation
9. Bronchiectasis
10. Tracheo-oesophageal fistula
11. Collapsed trachea.

Cattle

1. Structural defect of larynx
2. Collapsed trachea

Sheep

1. Paralysed left half of larynx

Cats

1. Oesophageal obstruction

Chimpanzee

1. Achalasia

DISCUSSION

The working length of 1,1 meter of this gastroscope is too short to permit the entrance and examination of the stomach of horses and cattle. In sheep and goats this would be possible. The instrument is long enough to enter the duodenum in all breeds of dogs. To date it has been found impossible to enter the trachea of even a large cat due to the induced laryngeal spasm.

Use of the gastroscope has not resulted in any damage to any structure except minor scratches and bleeding in:

1. the nasal passage of a horse due to some pathological obstruction.
2. the tender mucosa of a dog's oesophagus examined post operatively and
3. the mucosae of the stomachs of a few dogs after frequent introductions of the endoscope at a single examination.

CONCLUSION

By choosing the correct gastrointestinal fibrescope (i.e. the GIC type P), it can be successfully used in all species to examine a wide range of anatomical structures. It handles easily, takes little time, is safe and needs little experience to make it an useful diagnostic aid.

ACKNOWLEDGEMENTS

Grateful thanks are extended to Messrs Protea Electro-Medical Services, Johannesburg for sponsoring the printing of the colour plate.

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FELINE INFECTIOUS PERITONITIS IN SOUTH AFRICA

P. BLAND VAN DEN BERG* and W.S. BOTHA†

ABSTRACT: Bland van den Berg, P. Botha W.S. **Feline Infectious Peritonitis in South Africa.** *Journal of the South African Veterinary Association* (1977) **48** No. 2, 109 – 116 (En) Dept. Med., Fac. Vet. Science, Univ. Pretoria, Box 12580, 0110 Onderstepoort, Rep. of South Africa.

The clinical, clinical pathological and pathological findings in ten natural and two experimental cases of feline infectious peritonitis are described. The literature is reviewed and compared with the findings of these cases. It is concluded that feline infectious peritonitis occurs as a distinct clinical disease entity in domestic cats in the Republic of South Africa.

INTRODUCTION

Feline infectious peritonitis (FIP) is a condition of cats first reported in North America in 1963[13] and 1966[34]. Since then numberous reports have appeared in the American literature[1 2 3 6 7 8 9 10 12 17 19 20 22 23 24 30] and the disease has also been reported in the United Kingdom[14 33], Ireland[11], Holland[18], Canada[25], Australia[15 31] and Japan[16]. To date no definitive reports have appeared in the South African literature concerning the disease in domestic cats, although a suspected outbreak has been reported in large carnivora at the Zoological Gardens in Johannesburg[5]. In this paper the clinical and pathological findings in ten natural and two experimental cases examined at the Faculty of Veterinary Science, University of Pretoria, are described, followed by a discussion.

CASE REPORTS

Clinical Findings

The ten natural cases involved both breed and nondescript cats of both sexes with ages (where known) ranging from 6 months to 4 years (Table 1).

Table 1: DETAILS OF TEN NATURAL CASES

Case	Breed	Sex	Age
1	Burmese	Male	4 years
2	Siamese	Female	6 months
3	Nondescript	Male	Unknown
4	Siamese	Male	1 year
5	Nondescript	Male	3 years
6	Nondescript	Male	1 year
7	Nondescript	Female	Unknown
8	Siamese	Male	2 years
9	Nondescript	Female	Unknown
10	Siamese	Female	6 months

History on admission revealed that cats were being presented because of one or more of the following clinical signs:

anorexia	8 cases
abdominal distension	8 cases
vomiting and/or diarrhoea	7 cases
persistent febrile reactions	4 cases
respiratory symptoms	3 cases
listlessness and depression	2 cases
weight loss	1 case
presence of typical ascitic fluid as an incidental finding during routine ovarectomy	1 case

Antibiotic therapy had been admininistered before admission to seven cases but response to treatment had in all cases been transient or absent.

Clinical examination following admission was usually fairly conclusive with one or more of the following signs being present:

ascites – (great variation in volume see Table 7);	9 cases
weight loss and dull coat	9 cases
Elevated rectal temperature (39,2–40,2°C)	7 cases
palor of visible mucous membranes	7 cases
increased respiratory rate	4 cases
dehydration (slight)	3 cases
icteric mucous membranes	3 cases
subcutaneous oedema	3 cases
enlarged scrotum	1 case
ocular and nasal discharge	1 case
hydrothorax (confirmed radiographically)	1 case

Ocular and neurological signs were not observed in any of the cases.

Laboratory examination of material obtained from 9 of the cats (Cat 8 died before samples could be taken) included haematology (Table 2); blood chemistry (Table 3); peritoneal fluid analysis and bacterial culture (Table 4) and electrophoresis on serum and peritoneal fluid (Table 5). Urine (Table 6) was analysed in three cases. The interpretation of these results is given in the discussion.

Treatment instituted for most cases consisted primarily of the administration of tylosin at 160 mg/kg and prednisolone at 4 mg/kg, orally, in two divided daily doses. Supportive treatment comprised administration of vitamins, fluid therapy and tube feeding. This treatment proved ineffectual and all the cats died or were euthanased within 1 to 33 days after initial admission (average 10,3 days).

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Table 2: HAEMATOLOGY

Case No.	1	2	3	4	5	6	7	8	9	10	Normal
Haemoglobin g%	9,5	10,9	11,4	10,2	10,0	10,0	8,1	—	8,9	9,7	8-14
Red cell count x 10^6/mm^3	6,06	4,05	6,09	5,96	5,32	4,84	4,28	—	5,97	5,47	5,5-10,0
Haematocrit (%)	33	27,7	43,3	37,5	33,7	32,3	32,7	—	39,8	39,4	24-45
White cell count /mm^3	11 900	23 900	19 900	18 000	19 200	1 700	6 900	—	4 400	10 300	8-19,000^3
Neutrophiles (Band)(Cells)%	87(2)	63	81(7)	73(6)	89(4)	—	89(2)	—	99	88	35-75
Lymphocytes (%)	11	32	18	26	10	—	11	—	1	9	20-55
Monocytes (%)	1	4	1	1	1	—	0	—	0	0	1- 4
Eosinophiles (%)	1	1	0	0	0	—	0	—	0	3	2-12
Basophiles (%)	0	0	0	0	0	—	0	—	0	0	rare

Table 3: BLOOD CHEMISTRY

Case No.	1	2	3	4	5	6	7	8	9	10	Normal
Blood urea nitrogen mg%	—	—	12,8	—	15,0	—	31,3	—	18	—	20-30
SGOT i.u./litre	53	27	18	—	—	31	40	—	—	—	15-30
SGPT i.u./litre	28	7	11	—	5	45	14	—	65	29	6-25
LDH i.u./litre	—	592	150	—	291	254	162	—	400	288	48-94
SAP i.u./litre	55	46	14	—	37	37	7,7	—	—	74	50-122
Total bilirubin mg%	1,0	—	—	—	2,2	—	—	—	—	—	0,1-1,0
Total serum protein g%	6,9	6,7	7,8	—	8,1	6,8	8,5	—	8,2	13,2	5,4-7,2

Table 4: PERITONEAL

Case No.	1	2	3	4	5	6	7	8	9	10
appearance	yellow	straw-coloured	clear	straw-coloured	yellow	clear	red, tacky	—	cloudy	—
Specific gravity	1,033	1,040	1,032	1,028	1,044	1,031	1,048	—	1,040	—
Red cell count/mm^3	—	150 000	190 000	94 000	109 000	40 000	270 000	—	20 000	—
White cell count/mm^3	—	12 200	8 300	13 800	3 800	2 600	9 100	—	2 900	—
Neutrophiles %	—	70	75	—	—	—	88	—	—	—
Mononuclear %	—	30	25	—	—	—	13	—	—	—
Total protein g%	—	7,4	6,2	4,2	7,2	4,6	8,0	—	6,2	—
Microbiological examination	—	Negative	Negative	Negative	Negative	Negative	Staphylococcus aureus	—	Negative	—

Table 5: ELECTROPHORESIS
A. SERUM

Case No.	1	2	3	4	5	6	7	8	9	10	Normal
Total protein g%	6,9	6,7	7,8	—	8,1	6,8	8,5	—	8,2	13,2	5,4-7,2
Albumin g%	—	1,88	1,84	—	2,18	1,50	2,46	—	1,99	2,42	3,2
Alpha-1-globulin g%	—	0,36	0,22	—	0,12	0,22	0,29	—	0,66	0,08	0,3
Alpha-2-globulin g%	—	0,36	0,44	—	0,36	0,67	0,39	—	0,60	0,08	0,4
Alpha-3-globulin g%	—	1,17	1,30	—	0,96	1,35	1,17	—	1,16	1,21	0,7
Beta-1-globulin g%	—	0,47	0,22	—	0,73	0,45	0,59	—	0,58	0,33	0,4
Beta-2-globulin g%	—	0,36	0,65	—	0,36	0,52	0,48	—	0,66	0,16	0,4
Beta-3-globulin g%	—	0,82	—	—	0,36	—	0,48	—	—	0,65	0,3
Gamma-globulin g%	—	1,29	3,14	—	3,02	2,10	2,64	—	2,67	8,26	0,8
A : G ratio	—	0,39	0,31	—	0,37	0,28	0,41	—	0,32	0,22	1,0

B. PERITONEAL FLUID

Total protein g%	—	7,4	6,2	4,2	7,2	4,6	8,0	—	6,2	—	
Albumin g%	—	2,09	1,86	1,61	1,93	1,20	2,48	—	—	—	
Alpha-1-globulin g%	—	0,26	) 0,20	)	)	0,06	0,18	—	—	—	
Alppa-2-globulin g%	—	0,35		)	)	0,39	0,27	—	—	—	
Alpha-3-globulin g%	—	1,04	0,82	)	)	0,90	0,92	—	—	—	
Beta-1-globulin g%	—	0,61	) 0,62	2,59	5,27	0,24	0,64	—	—	—	
Beta-2-globulin g%	—	0,35		)	)	0,42	0,46	—	—	—	
Beta-3-globulin g%	—	0,95	—	)	)	—	0,37	—	—	—	
Gamma globulin g%	—	1,74	2,68	)	)	1,38	2,68	—	—	—	
A : G ratio	—	0,39	0,43	0,62	0,37	0,35	0,45	—	—	—	

Table 6: URINE ANALYSIS

Case No.	2	4	8	Normal
Colour	Yellow-brown	dark yellow	orange-yellow	Yellow
Odour	typical	typical	typical	typical
Deposits	nil	nil	nil	nil
Specific Gravity	1,027	1,040	1,020	1,018-1,040
pH*	6,5	6,0	6,0	5,0-7,0
Protein*	+	+	++	—
Glucose*	+	++	—	—
Ketones*	—	—	—	—
Bilirubin*	—	—	—	trace
Red blood cells	—	—	—	nil
White blood cells	—	—	+	occasional
Epithelial cells	—	—	+	occasional
Casts	—	—	+	occasional
Crystals	—	—	+	occasional

*"Bili-Labstix" (Ames)

Pathology

The macroscopic pathology was characterised by visceral and parietal fibrinous peritonitis, focal disseminate necrotic plaques, and a varying degree of abdominal exudation. (Table 7)

The visceral peritonitis (marked in eight cases, moderate in two cases) was evidenced by a fibrinous exudate covering the serosal surfaces of the liver (10 cases), omentum (10 cases), spleen (8 cases), gastro-intestinal tract (7 cases), kidneys (2 cases) and urinary bladder (1 case). This exudate was thickest and most noticeable on the liver and spleen. Associated with the exudate were focal disseminate white necrotic plaques varying in size from pinpoint to 2 millimetres in diameter – these were marked in three cases, but were present in all the cases. The parietal peritonitis (marked in four cases, moderate in six cases) involved mainly the ventral abdominal peritoneum and sometimes the diaphragmatic peritoneum. Abdominal exudation was present in nine cases in the form of a clear yellowish fluid, varying in volume from less than 10 mℓ to 1 000 mℓ. The fluid was invariably viscid with fibrin clots and strands present. Fibrinous adhesions between the omentum and the abdominal organs (liver, spleen and gastro-intestinal tract) were found in 5 cases. In seven cats the peritonitis infiltrated the pancreas causing oedema, petechial haemorrhages and fibrinous inflammation of the parenchyma and interstitial tissue. Three cases had a mild splenomegaly, hepatomegaly and mesenteric lymph node hyperplasia.

Icterus was present in three of the cats, two of which showed a focal disseminate hepatitis with dull grey foci (2 mm in diameter) extending subcapsularly into the liver parenchyma. These same foci were also found, on cut section, to be present deep in the liver tissue.

Involvement of the kidney parenchyma as an extension of the peritonitis was evident in two cats, one of which showed numerous foci of necrosis distributed throughout the cortex. A fibrinous pleuritis associated with pneumonia of the underlying lung tissue was encountered in one animal. In some of the cases an exudation into the thoracic cavity of a small amount of a clear, viscous, tacky fluid had taken place.

The subcutaneous tissue of the ventral abdomen was oedematous, gelatinous and yellowish in three cases.

In three of the six males a serofibrinous peri-orchitis was observed. For histopathological examination samples (number indicated in brackets) of the following organs were fixed in 10% buffered formalin, embedded in paraffin, sectioned and stained with haematoxylin and eosin: liver (10), spleen (10), omentum (4), stomach (1), intestine (5), brain (7), pancreas (5), kidney (8), heart (4), lung (3), testis (3), parietal peritoneum (3), eye (2) and mesenteric lymph nodes (2).

The most striking histopathological change was the irregular fibrinous exudate (1 mm) present on the peritoneal surfaces. The exudate was composed primarily of fibrin, with focal accumulations of cells, necrotic nuclear and proteinaceous material. The cell types pre-

Table 7: MACROSCOPIC PATHOLOGY

A *Natural cases*

Case number	Abdominal exudation	Thoracic exudation	Visceral peritonitis	Parietal peritonitis	Focal necrosis	Icterus	Focal hepatitis	Focal nephritis	Fibrinous pleuritis
	(mℓ)	(mℓ)							
1	10	0	+++	++	+++	+++	++	—	—
2	450	10	+++	++	++	—	+	—	—
3	1 000	0	+++	+++	++	—	—	—	—
4	500	50	+++	++	+	—	—	—	+
5	200	0	+++	+++	+	+++	++	—	—
6	400	15	+++	+++	++	—	—	—	—
7	0	0	++	++	+	—	—	—	—
8	750	0	+++	++	+	++	—	+++	—
9	250	10	+++	+++	+++	—	—	++	—
10	10	150	+	+	+++	—	—	—	+++
% marked			80	40	30	20	—	10	10
% moderate			10	50	30	10	20	10	—
% mild			10	10	40	—	10	—	10

B. *Experimental cases*

11	10	0	+	—	+++	—	+++	—	—
12	0	0	+	—	+	—	+++	—	—

\+ mild
++ moderate
+++ marked

sent in the exudate varied from case to case and from organ to organ, but were predominantly of the histiocytic type with some lymphocytes and occasional neutrophiles. The main cellular infiltration, comprising macrophages, lymphocytes, plasma cells and Russell body giant cells, was found in and immediately below the capsule or serous membrane. In most abdominal organs the exudate and inflammatory processes were confined to the superficial portions but subcapsular extension into the liver (6 cases), pancreas (5 cases) and lung (1 case) was observed. In addition focal disseminate and randomly distributed necrosis of the parenchyma was observed in the liver (5 cases), kidney (2 cases) and spleen (1 case). The lesion was a focal coagulative necrosis of the parenchyma with a histiocytic and lymphocytic cellular reaction forming a typical granulomatous lesion in some cases. In other cases the necrosis was predominant and cell infiltration less evident.

In most of the ten cats a severe inflammation of the omentum manifested by multiple areas of necrosis, oedema, and a histiocytic, plasma cell and lymphocytic infiltration was found. A marked hypertrophy and hyperplasia of the mesothelium was especially prominent on the spleen and liver capsule. The visceral peritoneum of the intestinal wall was similarly affected with focal areas of granulomatous infiltration extending into the underlying muscle layers. Meningitis was not observed in this series of cases.

A vasculitis with severe peri-vascular round cell and plasma cell infiltration was found only in cat 10 where it was marked in the liver and lung.

Transmission Experiment

This experiment was conducted early on in the investigation in an attempt to confirm the diagnosis. An 0,3 mℓ volume of a 10% bacteriologically sterile suspension of pooled liver, spleen and kidney in normal saline, prepared from Case 3, was injected intraperitoneally under strictly aseptic conditions into two healthy, 3 month old kittens which had previously been vaccinated against feline panleucopenia. Both kittens showed a marked temperature rise (40,5°C) within 48 to 72 hours after injection. Anorexia and listlessness were followed by progressive loss of condition, dehydration, mucopurulent ocular and nasal discharges, sneezing and dyspnoea (one kitten), melaena, soft stools, mucoid anal discharge and slight rectal prolapse. No sign of abdominal distension was noted although one kitten experienced pain on abdominal palpation. Ataxia developed in one kitten which died 14 days after inoculation. The other kitten died 2 days later. Postmortem examinations showed emaciation, slight peritoneal exudation, a mild fibrinoid peri-hepatitis and multiple focal disseminated opaque white foci (approximately 1 mm diameter) in the liver parenchyma. Similar small white foci were also seen in the spleen, and a nephrosis and enlargement of mesenteric lymph nodes was evident. A small pyloric erosion approximately 1 cm in diameter was observed in one kitten. Fresh organ specimens for viral and bacterial isolation proved to be negative. Microscopic examination revealed a fibrinous peritonitis and perihepatitis and subcapsular focal areas of necrosis and lymphocyte infiltration in the liver. These lesions were more marked in one kitten than the other. In both kittens, however, focal disseminated arreas of necrosis were observed in the hepatic portal tracts with cell infiltration (lymphocytes and macrophages) especially marked around the blood vessels. A peri-splenitis with plasma cell and macrophage infiltration in and under the splenic capsule was again more prominent in one kitten, but both kittens showed the presence of numerous small focal areas of necrosis in the deeper tissues of the spleen

associated with a macrophage and lymphocyte cell reaction. Mesothelial proliferation and lymphoid hyperplasia with prominent lymph follicles was also observed. The mesenteric lymph glands showed a reticulo-endothelial and lymphoid hyperplasia together with small areas of necrosis and occasional neutrophiles.

These results closely resembled those obtained in other transmission experiments[15 16 18 30 31 33 34].

DISCUSSION

Feline infectious peritonitis is now a well described clinical entity of cats. It has been described in cats of all ages, ranging from 3 months to 17 years[22], although young cats appear to be more susceptible[10 14] (the average age, where known, of the cases described was 20 months). A recent report describes acute death due to FIP in a 23 day old kitten[20]. Early indications of a pre-dilection for the Siamese breed[34] have not been substantiated in later surveys[3 10 30]. Some reports[10 22 34] indicate that the incidence of the disease is higher in males than in females; others,[3 14 30] however, have refuted this (in the ten cases reported 6 were males and 4 females).

Climatic factors do not apparently play a role in the incidence of the disease[22]. Multiple cases have been observed in the same household or cattery[10 22 30], sometimes in association with feline leukaemia[6 10]. Morbidity is usually low, but mortality rates are high and virtually all cases terminate fatally[10 22].

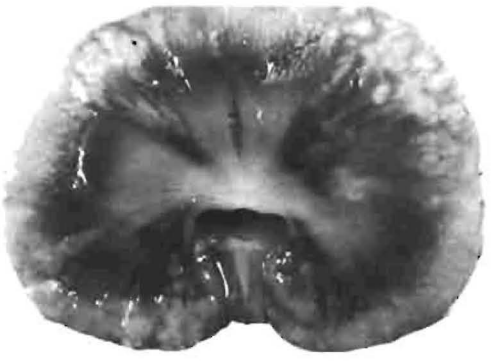
The case of FIP has still to be proved conclusively, although there is general agreement on a probable viral aetiology[3 29 30 34 36]. This view is based on negative bacterial, mycoplasmal, and chlamydial cultures, electron microscope demonstration of virus particles in mesothelial cells of experimentally infected cats[10 29 36], and successful transmission using 220 and 100 millimicron filtrates of tissue shown to contain virus particles in electron micrographs[29]. Recent work has further demonstrated the presence of a corona-like virus in ascitic fluid and liver homogenates of natural cases of FIP[21]. However, the virus has not been grown in tissue culture or embryonated hen's eggs, nor are the common laboratory animals susceptible to it and as a result the virus has neither been isolated nor characterised. No significant isolates were made from ante- and post-morten specimens taken from our cases.

Routes of natural transmission are unknown[3 10] although the production of symptoms in a cat fed urine from a natural case may mean the oral route is of importance[10]. Low morbidity rates, in the presence of a generally low level of immunity amongst cats of all ages probably indicates an inefficient form of natural spread[10]. Experimentally the disease is relatively easy to transmit using peritoneal fluid, organ suspensions, and blood, via the intra-peritoneal, subcutaneous, and oral routes[3 15 28 30 34]. From observations on multiple cases within the same household it appears that the natural incubation period may be as long as 4 months[10]. Experimentally it may be a short as 3 days (as was manifested by the two experimentally produced cases described above).

The pathogenesis of the disease is poorly understood. Although considered a primary virus infection it is thought that immunological factors may play a role in the development of the syndrome. This observation is based on the lesions of vasculitis[29] in many organs (vasculitis was encountered in only one case in the present series), hypergammaglobulinaemia and plasma cell reactions[28]. The many "variant" or "atypical" forms and the great variation in tissue involvement may be associated with variations in the mode of transmission, site or route of exposure, dosage or strain differences in the virus and age of the host at the time of exposure[24]. Experimental transmission studies using intraperitoneal and subcutaneous routes confirm the influence of the route of infection on the development of the lesions. Subcutaneous routes result in a typically haematogenous type of distribution with widespread involvement of many tissues and less involvement of the peritoneum. The intraperitoneal route results in the more classical peritonitis and exudation[28].

Symptomatology has evolved from the original "classical" descriptions of the disease, to incorporate many new aspects, the so-called "variant or atypical" forms. In its classic form[3 14 22 30] the syndrome has an insidious onset with mainly non-specific signs including an antibiotic resistant fever, anorexia, lethargy, loss of condition, and loss of weight. Anaemia, jaundice and darkened urine, sneezing, coughing and dyspnoea, diarrhoea and vomiting may be seen. The development of peritoneal exudation is variable both in stage of appearance and degree, sometimes appearing only in the later stages of the syndrome (in Case 4 clinically detectable exudate was only evident 24 hours prior to euthanasia), and varying in volume from as little as 10 mℓ to 1 ℓ or more. When present ascites constitutes the most characteristic symptom of the disease. Palpation of the enlarged abdomen is usually not painful. Clinical signs are invariably progressive with little or no response to treatment and death usually occurs within 5 to 6 weeks of the onset of symptoms. "Atypical" signs may include scrotal swelling through the development of periorchitis[12 30], bleeding gums, ventral oedema, abdominal "masses" (enlarged lymph nodes, viscera distorted by adhesions)[22] and ocular and nervouc signs. The opthalmological and neurological forms are being described with increasing frequency[1 2 7 8 9 24]. They may be associated with the characteristically persistant febrile reaction, anorexia, and loss of weight, but rarely with the development of peritoneal exudation. Ocular lesions, which may lead to blindness, are obvious and may include chemosis, corneal oedema, keratic precipitates, aqueous flare, hypopyon, iridocyclitis, posterior synechiae, exudative choroiditis, retinitis and retinal detachment. Neurological signs are indistinguishable from other feline encephalopathies and may include ataxia and inco-ordination, circling, muscle tremors and generalised seizures. (We did not observe any opthalmological or neurological signs in our 10 cases).

There are no specific serological or other diagnostic tests. Clinical pathology, therefore constitutes an important part of the diagnosis. A number of characteristic changes occur with reasonable consistency[3 10 14 22 23 27 30 32 34]. Haematological investigations typically reveal a normocytic, normochromic anaemia with haemoglobin values as low as 5 g% (average for present series was 9,8 g%) and haematocrit values of 10 to 15% (average for present series 35,5%), a leucocytosis with counts up to 52 000/mm^3 (average for present series was 12 910/mm^3, with highest value of 23 900/mm^3), a neutrophilia with a mild left shift, a lymphopenia and an eosinopenia. A leucopenia occurs occasionally;



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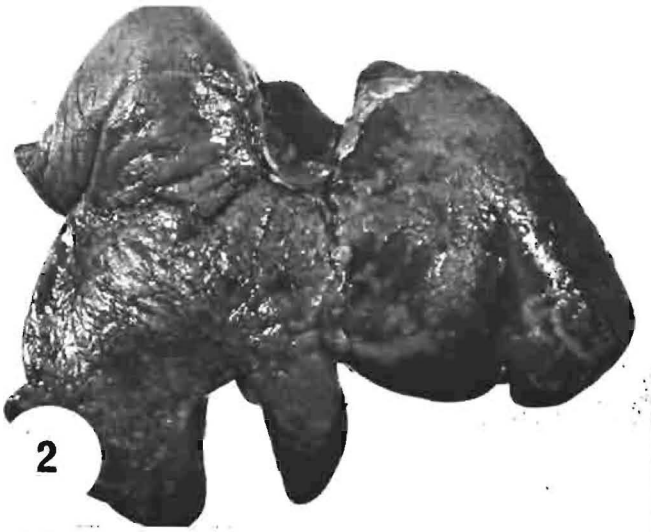

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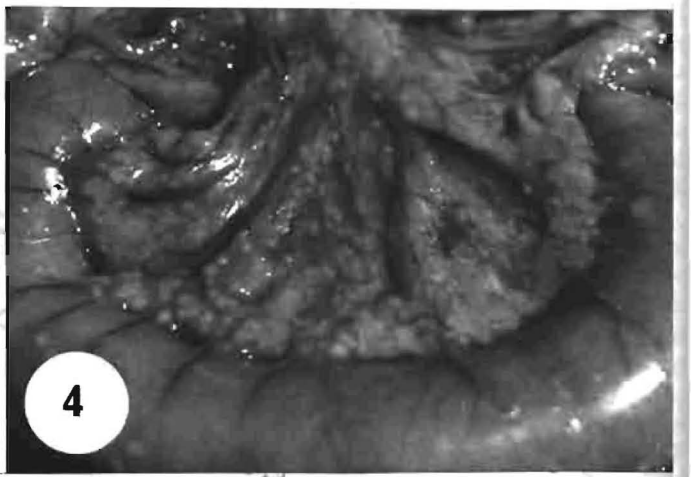

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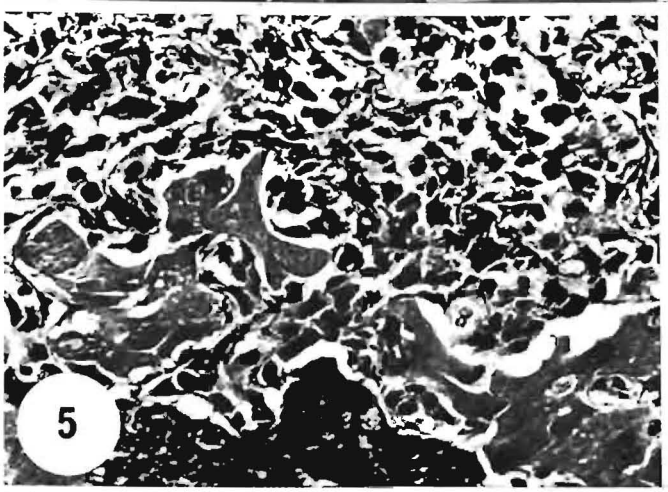

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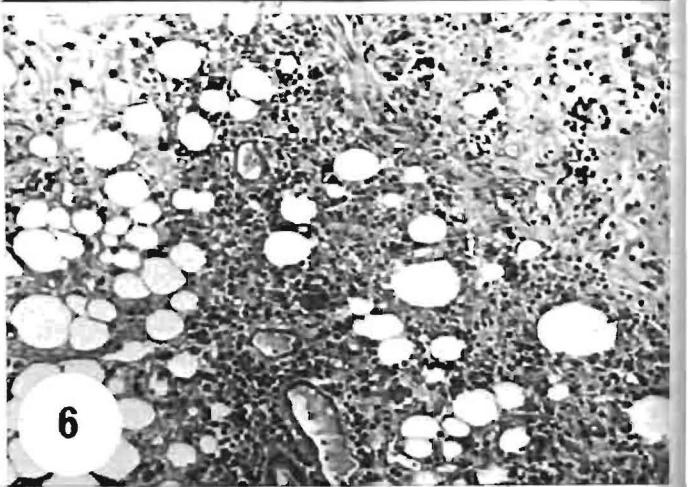

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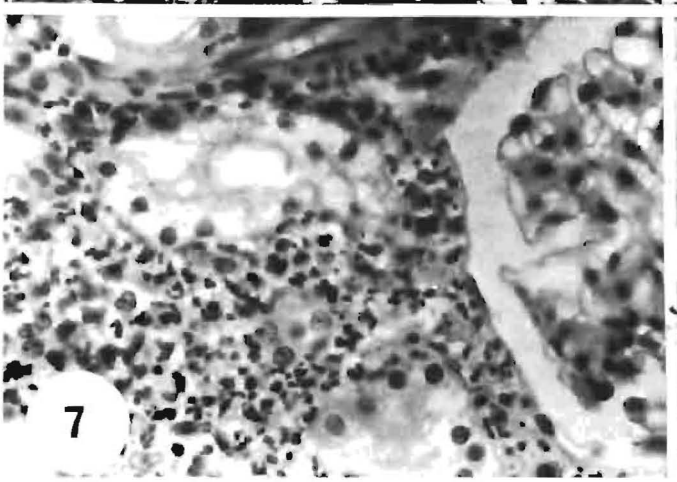

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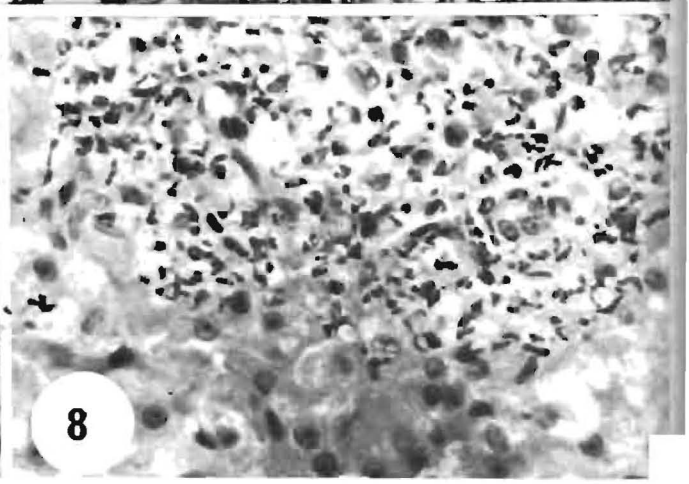

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counts may drop to 1 600/mm³ (one case in present series had a count of 1 700/mm³). Intracytoplasmic inclusions have been described in the neutrophiles. These were not seen in this series. Evaluation of blood chemistry may reveal an elevated blood urea nitrogen, prolonged bromosulphophthalein clearance, and increased total, direct and indirect reacting bilirubin. Increased serum glutamic pyruvic transaminase (SGPT), serum glutamic oxalic transaminase (SGOT), serum lactic dehydrogenase (LDH) and serum alkaline phosphatase (SAP) values indicate tissue injury including liver damage (as seen in some cases in the present series). Total serum proteins are elevated through a diffuse hypergammaglobulinaemia, with values ranging from 6 to 12 g% (average in present series was 8,3 g%), of which the gamma globulins constitute 1,5 to 7,4 g% (average in present series was 3,3 g%). Analysis of the peritoneal fluid is important as the fluid has characteristics which readily distinguish it from other forms of ascites. The fluid, although exudative in nature (specific gravity 1,017 to 1,047, high protein values), is clear or straw-coloured with occasional fibrin flakes, and has relatively few cells. The white cell count ranges from 70 to 10 000/mm³ (average in present series 7 500/mm³ with range of 2 600 to 13 800/mm³) and consists mainly of neutrophiles, and the red cell count may reach 350 000/mm³ (average in present series 124 000/mm³ with range of 20 000 to 270 000/mm³). Protein levels of the fluid are high (5 to 8 g%) (average electrophoresis of serum and peritoneal fluid mirror one another so closely that it would appear that the accumulation of fluid in the abdomen is through an outpouring of serum from the blood stream (this fact is well illustrated in the electrophoresis values recorded above). Analysis of urine is usually non-contributory apart from increased bilirubin and urobilinogen levels in some cases.

The macroscopic and microscopic pathology furthermore reflects the great variations of tissue involvement and has again evolved from the classical fibrinonecrotic form affecting the parietal and visceral serosal surfaces of the peritoneum to include a focal disseminated granulomatous form with more pronounced vascular lesions and minimal involvement of the serosae. This has led to the terms "wet" and "dry" FIP.

The descriptions of the classical fibrinonecrotic or "wet" form closely resemble the pathology of the 10 natural cases described in this report. In the disseminated granulomatous or "dry" form[2 19 24] a variety of organs, either singly or together, are affected, principally kidneys, visceral lymph nodes, lungs, liver, eye and leptomeninges, and less commonly pancreas, spleen, skeletal muscles, thyroid and bladder. Serosal surfaces are affected to a minimum degree. Macroscopically grey white foci 1 to 2 mm in diameter occur in these tissues, which may coalesce to involve fairly large areas of especially kidney cortex. Microscopically these lesions resemble those of the wet form, except that vasculitis and thrombovasculitis are more prominent, with lesions oriented around the blood vessels. Ocular lesions are concentrated in the uvea, retina, and meninges of the optic nerves. Extension into the nerves may result in a focal optic neuritis. Fibrino-cellular exudates collect in the ocular chambers. Lesions of the central nervous system are concentrated in the meninges, but may also occur in the choroid plexus and ependyma. Small grey plaques may be seen on the meninges which histologically may be seen to extend into the brain and cord parenchyma, mainly along blood vessels.

The diagnosis of FIP may be difficult especially during the early stages before peritoneal exudate develops. The "variant" forms in which exudation does not develop at all, present an even greater diagnostic challenge. In the absence of any serological or other tests, the diagnosis depends on the collation of clinical symptoms, clinical pathology (especially paracentesis) and macro- and micro-pathology, together with the elimination of differential diagnoses. With the multiplicity of signs this list is long and the following may be included. Syndromes causing a persistent fever, anaemia, anorexia, and loss of weight – feline leukaemia virus[6 10 22 28], infectious anaemia[14 22], toxoplasmosis, localised or systemic bacterial infection and pansteatitis[22]. Syndromes causing ascites and abdominal distension – bacterial peritonitis, tuberculosis, pyometra, congestive heart failure and cirrhosis, neoplasms, chylous ascites and ruptured bladder[14 22]. Syndromes causing pleural effusions – empyema, tuberculosis, feline leukaemia, heart failure, chylothorax and neoplasms[22]. Syndromes causing neurological symptoms – mycotic *(Cryptococcus)*, protozoal (toxoplasmosis), bacterial and viral encephalopathies, leukaemic infiltration and other neoplasms of the central nervous system[24]. Syndromes causing ocular lesions – toxoplasmosis, systemic mycotic disease, and intraocular leukaemic infiltration[26].

Treatment of FIP appears to be of little use in most cases and the prognosis must be regarded as very poor. All our cases died despite intensive treatment. However, a report in which a small number of cases was successfully treated has appeared[4]. Treatment included tylosin (160 mg/kg) and prednisolone (4 mg/kg) administered per os in 2 daily divided doses, plus supportive fluids and vitamins. In another report[22] a cat given tylosin (50 mg t.i.d.) orally, and prednisolone 10 mg and tylosin 200 mg intra-peritoneally after each of

Fig. 1 Focal necrosis and interstitial nephritis in the kidney cortex and medulla (Case 8)

Fig. 2 Severe fibrinous visceral peritonitis on the surface of the liver. (Case 8)

Fig. 3 Fibrinous adhesions between abdominal organs. (Case 4)

Fig. 4 Focal necrotic plaques in the mesenterium of the small intestine. (Case 3)

Fig. 5 Extension of the inflammatory reaction into the liver parenchyma. x 500. (Case 1)

Fig. 6 Inflammation replacing the normal fat tissue of the omentum. x 126. (Case 1)

Fig. 7 Interstitial nephritis with necrosis of the periglomerular tissue. x 400. (Case 8)

Fig. 8 Focal liver necrosis with experimental transmission. x 400. (Case 11)

several paracenteses, also apparently recovered. Nevertheless these cases are but a very small percentage of the total number of cases described, and by far the majority of authors consider the disease incurable.

Prophylaxis at this stage would appear very difficult since potential carriers cannot be detected nor can susceptible animals be vaccinated. Isolation of incontact cats for a minimum of 6 months plus thorough disinfection are the only means of prophylaxis at this stage[10].

CONCLUSIONS

The natural and experimental cases mentioned in this report have exhibited many of the important clinical, clinical pathological and pathological changes described in the literature. On the basis of these findings there can be little doubt that FIP occurs as a clinical disease entity in the Republic of South Africa, and having established its presence, the disease should be considered in the differential diagnosis of all persistently febrile cats, especially those showing signs of abdominal distension. In the absence of specific diagnostic tests the clinical pathology, abdominal fluid analysis and post-mortem findings should be carefully evaluated.

ACKNOWLEDGEMENTS

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OVARIAN AUTOGRAFT AS AN ALTERNATIVE TO OVARIECTOMY IN BITCHES

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ABSTRACT: Le Roux P.H.; van der Walt, L.A. **Ovarian autograft as an alternative to ovariectomy in bitches.** *Journal of the South African Veterinary Association* (1977) **48** No. 2, 117 – 123 (En) Animal Hospital, Hermitage Terrace, Richmond, 2000 Johannesburg, Rep. of South Africa.

The literature on autotransplantation of the ovary is briefly reviewed with emphasis on the portal vein drainage area as the transplant site. An experiment is reported whereby bitches hearing such grafts were compared to entire and ovariectomised subjects with regard to endocrine status and behaviour. It is concluded that autotransplantation of the ovary to the portal vein drainage area may be a promising method of abolishing oestrus and yet avoiding the eunuchoid syndrome as is seen in ovariectomised subjects.

INTRODUCTION

If a bitch is to be kept as a family pet it is preferable that the oestrus cycle be controlled or abolished. In spite of advances made in the use of steroid hormones for this purpose, most bitches are subjected to ovariectomy at an early age or during panhysterectomy neccessitated by some uterine pathology.

The South African Police use bitches for tracking and other solo work only and have to kennel them when in oestrus. Members of the Dog Unit of the South African Defence Force hold the opinion that ovariectomy reduces aggression, keenness to work and stamina.

Amongst veterinarians there is some divergence of opinion as to the effect this operation has on the subject's wellbeing, but a fair proportion of ovariectomised bitches are likely to develop the eunuchoid syndrome. Some of these will also suffer from urinary incontinence, obesity, alopecia, seborrhoea and pruritis while all have a subclinical deficiency of female hormones. Spayed bitches kept on a maintenance dose of ethinyl oestradiol at 5–10 μg/kg daily[41] are much more alert and playful than before hormone treatment and they revert to an apathetic state when the medication is stopped.

Apart from random clinical reports on the treatment of so called "hypogonadism" in ovariectomised bitches (and female cats), the matter has received almost no attention in veterinary literature although many aspects of the effects of ovariectomy and supplementation of sex steriods have been studied in laboratory rodents. Significant changes in behaviour, body composition and endocrine status have been reported. The results of these experiments agree so closely with the above clinical observations that it is reasonable to assume that the mechanism whereby the eunuchoid state is produced will be similar in most species of mammals.

Experimentation pertaining to the behaviour of transplanted ovarian tissue spans nearly a century[36]. Early experiments proved that the ovaries of rabbits could be excised and reimplanted near their original site and full sexual function would be maintained[33]. The fate of ovarian tissue placed on various organs was reported[67 35]. It is of special interest to note that when the ovaries were transplanted to the area drained exclusively by the portal vein, oestrus was abolished[77 22]. When such an ovary was regrafted to the axilla, oestrus recurred shortly thereafter[22]. Ovariectomy and transplantation of the ovary into the spleen led to proliferation of the graft in a "neoplastic" manner[4]. The hepatic conversion of ovarian steroids abolished the feedback inhibition of gonadotropins and caused a sustained elevation of FSH and LH which was responsible for this proliferation[17 48]. When compared to ovariectomised subjects, fewer "castration cells" were evident in the pituitaries of subjects bearing an intrasplenic ovarian graft[3]. It was often reported that such grafting was not followed by such marked atrophy of the sexual organs as after ovariectomy. This was then ascribed to gonadal hormones which crossed the liver into the general circulation[3 12 28]. The survival of such grafts was studied for periods of up to 6 years in guinea pigs[28 45]. The rate of progesterone production by the graft has been studied in rabbits and the levels in the portal and systemic blood compared[40 65]. It was confidently stated by several workers that sexual behaviour was completely abolished except in those cases where post-operative adhesions created a bypass to the parietal peritoneum and general circulation[25]. Others observed that marked stimulation of the genitals could occur[3 12], especially where the graft became very large. Although subjects with splenic ovarian grafts gained more weight than intact controls, they did not become as obese as ovariectomised rats.[42] The behaviour of such transplant subjects has not been reported upon in any previous experiment.

In bitches, the results of transplantation of the ovary have been reported in experiments where the aim was to maintain sexual function[8 19 64 74 54]. No work has been published on autotransplantation of the ovary to the portal vein drainage area in dogs.

The author was of the opinion that, in bitches, autotransplantation of the ovary to the portal vein drainage area (ATOPA) would produce results similar to those reported in laboratory rodents.

Trials involving 25 subjects were conducted from 1972 until 1975. Because these cats and dogs belonged to clients of our practice and were available for laparotomy and necropsy only on a chance basis, few conclusive results were obtained and progress was slow. With the assistance of the Dog Unit of the SA Defence Force it became possible to extend our investigation. To date this has produced a great deal of information. This report is confined to those aspects which have a bearing on the possibility that ATOPA might be a practical alternative to ovariectomy of domestic pets in order that the eunuchoid syndrome could be avoided.

The Hypothesis

Autotransplantation of the ovary to the portal vein drainage area would be a feasable alternative to

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ovariectomy in bitches if the following requirements were met:

1. *The Graft* must remain viable and yet not proliferate to an exessive extent.
2. *Ovarian Hormones* must be produced by the graft in adequate quantities.
3. *Oestrus* must be abolished by the hepatic transformation of the oestradiol and progesterone produced by the graft.
4. *The Eunuchoid Syndrome* must be prevented by the ovarian hormones and their metabolites which reach the general circulation via the portal system of the liver.

MATERIALS AND METHODS

Subjects

Twelve bitches from the Dog Unit of the SADF with ages ranging from 1 to 5 years were used. The animals were kept in standard dog runs and fed a commercial dog ration. They were placed in three groups:

Group I consisted of four entire bitches, i.e. two Alsatians, one Labrador and one Doberman.

Group II consisted of three Alsatians and one Bouvier des Flanders. ATOPA was performed simultaneously on all four and laparotomy 52 weeks later for inspection and biopsy.

Group III consisted of two Dobermans and two Alsatians. Of these, three had been ovariectomised some time before and one was ovariectomised at the start of the experiment.

Surgical method of ATOPA. After bilateral ovariectomy, both the ovaries were cut into sections 1–2mm thick. A 1cm long incision was made through the serosa of the stomach to enable a pouch to be formed to either side by blunt dissection. All the ovarian fragments were pushed into these or additional pouches and the incisions were closed with fine catgut. Special care was taken to avoid dropping fragments of ovary into the abdominal cavity.

Specimens were taken for biopsy at 52 weeks by shelling out one of the masses of ovarian tissue and leaving the rest undisturbed.

Blood samples were drawn from the cephalic vein into plain evacuated tubes, allowed to clot and centrifuged. Serum was collected and kept at –20°C until assay. The frequency of sampling differed between the groups: Group I had 22 random samples collected without regard to the stage of sexual cycle.

Group II were sampled one week before ATOPA, and then weekly till week 10, every 2 weeks till week 20, every 3 weeks till week 47 and then weekly till week 53.

Group III. The subject ovariectomised at the outset was sampled as for group II till week 22, then weekly for seven consecutive weeks together with the other three in this group.

Specimens of ovarian tissue were collected at the time of surgery for ATOPA and 52 weeks later. The tissue samples were placed in Bouin's fixative, washed, dehydrated in alcohol, embedded in paraffin wax, sectioned and stained with Haematoxylin-eosin by Prof. W.H. Gerneke of the Department of Anatomy, Faculty of Veterinary Science, University of Pretoria.

Assays and estimations

Thyroid hormone: Thyroxine levels were determined by means of Tetrasorb (Abbot) and Seralute (Ames) kits. Resin T_3 uptake was assayed by Thyopac-3 (Amersham) and Trilute (Ames) kits

Steroid hormones: Oestradiol-17 beta, progesterone and testerone were estimated by radioimmunoassay[20] [71]. Cortisol was assayed employing a modified semimicro-competitive proteinbinding technique[52]. Results were computer-derived, using a probit-log transformation plot on a weighted regression analysis.

Adrenal response to ACTH: The subjects were fasted overnight. The test commenced at 9 am and proceeded with the minimum agitation of the subjects. The cortisol level was determined in samples taken before a single intramuscular injection of 0,5 mg tetracosactide (Synacthen Ciba-Geigy), then at 30 min, 60 min and 120 min thereafter. At the same time blood glucose levels and differential leucocyte counts were taken before ACTH and 120 min thereafter.

Work performance tests: All the dogs had been partially trained before entering this experiment. At approximately the 60th week of the trial they entered an intensive 6 week training period in which they performed obedience, obstacle course and attack work. The subjects were scored on the standard test schedules used by the Dog Unit of the SADF, which places particular emphasis on obedience, boldness and aggressiveness.

Body weight of the subjects in group II was recorded.

General observations

The handlers were instructed to report any signs of oestrus or unusual interest shown by male dogs.

RESULTS

When the graft sites were inspected 52 weeks after ATOPA, no adhesions were present. The ovarian tissue was seen as ovoid masses under the gastric serosa or the outer muscle layer. It was estimated that the graft had increased approximately eight times in size. The masses shelled out easily and no evidence of encapsulation was noticed (Fig. 1). Histological examination showed highly metabolic tissue with no sign of necrosis or fibrosis (Fig. 2).

From Group I a total of 22 random samples were collected. As may be expected, the mean values for oestradiol and in particular progesterone showed large

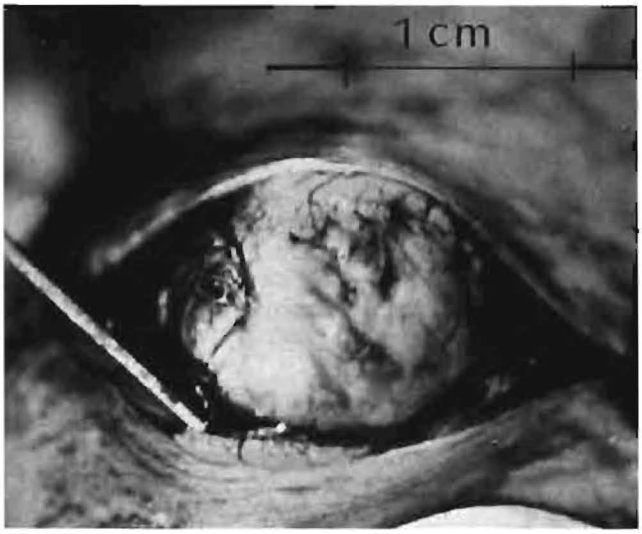

Fig. 1

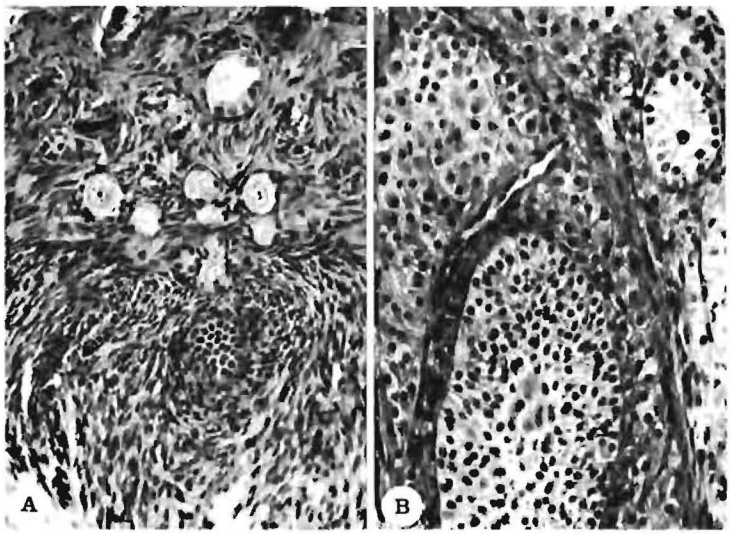

Fig. 2

tandard deviations. Our progesterone results are in greement with those reported earlier[53] while the oestradiol values were higher. This is no doubt due to the se of different methods and standards. In Table 1 the stimations for oestradiol for Groups II and III, included only the seven consecutive samplings taken at he end of the experiment.

Table 1: PERIPHERAL SERUM LEVELS OF ESTRADIOL AND PROGESTERONE

Subjects	n	Estradiol (pg/mℓ)	Progesterone (ng/100mℓ)
Group I	22	*102 ± 46	1182 ± 922
Group II	28	91 ± 28	81 ± 31
Group III	28	64 ± 19	68 ± 32

All results are means ± S.D.

When the three groups were compared, Group I obviously had the highest values for both oestradiol and progesterone. Statistical analysis of the results for the other two groups showed that the level of oestradiol was highest in Group II ($p<0,001$), Progesterone levels showed less variation although Group II was statistically elevated ($p<0,2$).

No significant alterations were observed in testosterone levels and subsequent estimates were abandoned.

The values for thyroxine and T_3 binding were inconsistent by each of the methods used and no conclusions could be drawn from the 120 results obtained.

Cortisol levels at rest were obtained in 61 samples and analysis did not disclose any differences between the groups. The values ranged from 0,4 to 8,8 µg/100mℓ.

The results of the adrenal stimulation test are given in Fig. 3 and Table 2.

Body weights were not available for groups I and III and those of group II showed a slight decrease over the 54 week period. None of the subjects in the experiment were obese at any stage.

Oestrus did not occur in any subjects in groups II and III and no pregnancies occurred in group I.

Hypogonadism was not reported in any subjects and no significant illnesses occured within these groups.

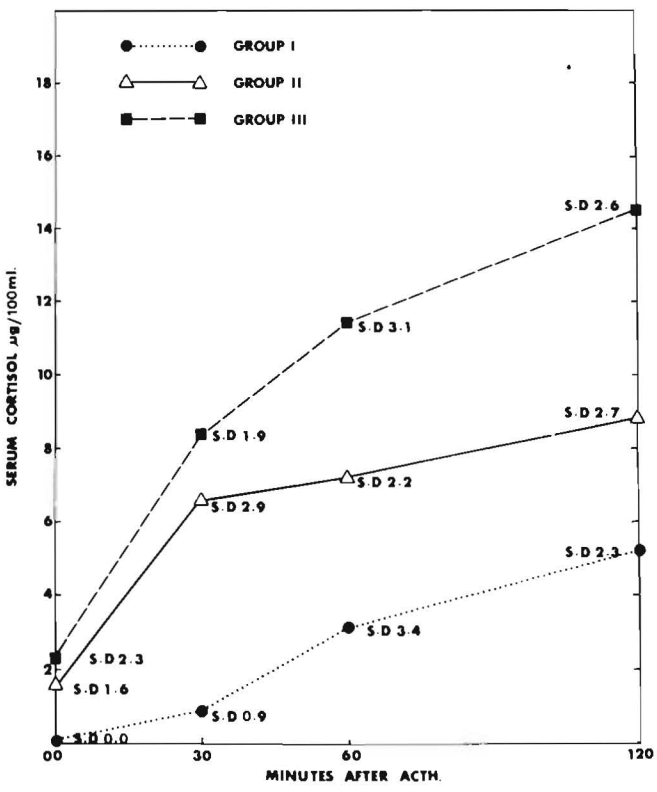

Fig. 3 Cortisol levels following ACTH stimulation.

Table 2: HEPATIC INACTIVATION OF ESTRADIOL AND PROGESTERONE

	Estradiol (pg/mℓ)	Progesterone (ng/100mℓ)	Cytology
Cephalic vein			
- S1	80	160	Anestrus
- S2	127	6	Proestrus
- S3*	107	471	Proestrus
Graft vein			
- S1	247	1144	—
- S2	894	2000	—
- S3	400	7990	—

*Possible hepatic bypass via postoperative adhesion

DISCUSSION

The ovarian graft

The ovaries of laboratory rodents are reported to survive well when transplanted whole[3 22 25 33 65]. The senior author has found that in the cat whole ovaries transplanted to the spleen or peritoneum will also survive. Cheval[8] found that when portions of the ovaries of dogs are transplanted to the abdominal muscles, only the germinal epithelium survives while the centre of the fragment undergoes necrosis. It has been reported that canine ovaries and uteri will survive when wrapped in mesentery and omentum[64]. Most contemporary reports suggest that vascular anastomosis is necessary for reliable results[19 54].

In fifteen biopsies of whole ovaries grafted to the spleen, mesentery and gastric serosa in dogs, only isolated areas escaped necrosis and resorbtion. Eight biopsy specimens from sites where the transplanted ovaries had been cut into progressively smaller fragments, showed that smaller fragments survived better. In eleven subjects where ovaries and/or fragments were grafted to both the spleen and the gastric serosa, no difference in survival relating to the locality could be shown. Luteinisation was not yet evident twelve weeks after ATOPA. The size of the graft has been found to increase during successfive phases of luteinisation to as much as ten times its original size at 42 weeks (P.H. le Roux, unpublished data).

These irregular growth spurts have been reported in mice[44] and are the result of FSH and LH surges[17]. Ovariectomy is followed by an immediate rise in FSH and a delayed but sustained rise in LH up to 70 times the resting level after 21 days[75]. The series of samples taken over 54 weeks from Group II showed irregular, fluctuating levels of oestradiol and progesterone.

It may be concluded that ATOPA with the fragmented ovaries located under the gastric serosa will consistently result in survival and considerable proliferation of the graft.

Hormone production

All the subjects in Group II, as well as the one bitch from Group III which was sampled in a similar way, showed a slight drop in oestradiol and a marked drop in progesterone in the first 3 weeks after surgery (P.H. le Roux, unpublished data). Such a drop in progesterone immediately after ovariectomy has previously been reported[14 51]. The level of oestradiol in Group II then rose significantly higher than in Group III and almost as high as for Group I. The level of progesterone was slightly higher in Group II when compared to Group III but far lower than the mean value for Group I.

Blood was obtained from veins draining the graft of three other ATOPA subjects and the oestradiol and progesterone level compared to the peripheral levels (Table 2). These figures cannot be related to those for normal ovaries[63] but similar figures for progesterone levels have been reported in ATOPA rabbits[65]. No figures were reported for oestradiol. It is clear from our figures that the hepatic clearance rate is very much higher for progesterone than for oestradiol.

Our results indicate that such ovarian grafts in bitches produce considerable quantities of oestradiol and progesterone, some of which reach the general circulation. In addition it can be expected that metabolites of these hormones would be present.

Abolition of oestrus

None of the subjects in Group II showed oestrus. This is in agreement with numerous reports on ATOPA in other species[4 22 46]. It was reported that oestrus and mating behaviour will occur only in those subjects where postoperative adhesions to the liver or parietal peritoneum provide a bypass to the general circulation[25]. In one ATOPA bitch (S_3 in Table 2) a tenuous adhesion was present between the graft site and the umbilical fat and pro-oestrus occured briefly. Another bitch (S_2) showed pro-oestrus similarly but no adhesion was present (S_1 was in "anoestrus"). When the levels of oestradiol for Group II were studied over the full postoperative period it was found that they showed frequent excursions up to 160 μg/mℓ and occasionally over 200 μg/mℓ. These levels were not however accompanied by any signs of pro-oestrus.

It has been reported that subjects bearing a splenic ovarian graft have very high levels of FSH and LH

Table 3: GLUCOSE AND DIFF. COUNTS FOLLOWING ACTH STIMULATION

	*Group	00 min	120 min
Glucose (mg/100mℓ)	I	**81 ± 5.5	82 ± 2.1
	II	72 ± 5.2	74 ± 6.1
	III	72 ± 3.8	81 ± 4.6
Neutrophils	I	67 ± 5.9	79 ± 3.1
	II	73 ± 13.6	85 ± 9.2
	III	65 ± 7.8	81 ± 4.6
Eosinophils	I	6.3 ± 2.1	7.6 ± 2.8
	II	6.5 ± 2.4	3.8 ± 4.8
	III	13.5 ± 11.1	7.0 ± 7.1
Lymphocytes	I	26 ± 5.7	12 ± 4.6
	II	20 ± 10.5	11 ± 5.2
	III	21 ± 6.6	12 ± 3.3

*Four observations in each group
**All results are means ± S.D.
Monocytes < 1% in all groups

constantly present[17 48] as is the case in ovariectomised subjects[75]. It can then be assumed that a sharp rise in oestradiol would inhibit FSH secretion[70]. The already elevated LH will cause rapid, proliferation and luteinisation of the FSH/oestradiol primed cells of the graft[61 76]. The hypothalamus and pituitary respond rapidly even to small fluctuations in oestradiol[1 6] whereas the genitalia respond slowly to large fluctuations[50]. In older grafts the predominant feature is luteinisation of all the cells[12 17] (see also Fig. 2).

From our findings it appears that, because of the fluctuating level of oestradiol, brief occasional periods of pro-oestrus may occur in a small proportion of ATOPA bitches. As these episodes do not progress to oestrus and subside spontaneously within days, they are not a serious problem; they may in fact be responsible for some of the benefits ascribed to ATOPA over ovariectomy. The problem will not be avoided by grafting only a portion of the available ovarian material. It has been reported that small grafts proliferate relatively more than larger ones to reach the same ultimate size in about the same period of time.

The Hypogonadic Syndrome

The available literature on the effects of ovariectomy should be reviewed to make some aspects of the "hypogonadic syndrome" clear. The most dramatic result of ovariectomy is the loss of sexual function and atrophy of the sexual organa since these tissues respond visibly to the large changes of oestradiol and progesterone during the sexual cycle. The receptor areas of the hypothalamus and pituitary respond to these and also to changes in the basal levels of oestradiol and to a lesser extent of progesterone[1 6 50 55]. These areas of the brain control the release of FSH, LH, ACTH and others. The "castration cells" which appear in the anterior pituitary are associated with the increased blood levels of FSH and LH[4 21 39]. The ability of the pituitary to secrete ACTH under stress is reduced[9 11] and the diurnal fluctuation of the "resting" level of cortisol[58] is reduced concurrently[57]. The adrenals accumulate cortisol precursors in the outer zona fasciculata[47] while the ability of the adrenal to synthesise cortisol *(in vitro)* is reduced[31]. Thyroglobulin accumulates in the follicles of the thyroid gland[5 10]. When oestradiol is administered to ovariectomised subjects these changes are prevented or reversed to a significant extent[21 30 68]. These endocrine changes radiate throughout the body to produce some of the hypogonadic signs.

The fat depots of the body possess receptors for specific steroid hormones so that deposition is blocked or facilitated in a regional manner in response to testosterone, oestradiol, progesterone and cortisol[34 37 38 66]. Oestradiol inhibits lipoprotein lipase in adipocytes of fat depots in general so that circulating fatty acids cannot be esterified and deposited[26].

The adrenal can synthesise only small quantities of oestradiol and progesterone. The detectable levels of these hormones in ovariectomised subjects originate from peripheral conversion of adrenal androgens and steroid metabolites[2 13 23 24 60].

Unlike the situation in humans, the normally low thyroid hormone binding capacity of dogs' blood is not affected by the level of oestradiol[18 56].

Ovariectomy is rapidly followed by a marked reduction in general activity[16 62], an altered feeding pattern, energy conservation and weight gain[29 42 72]. These changes are reversed by oestradiol and exacerbated or not affected by progesterone[15 27 29 69 73].

Virtually none of the reports reviewed deal with ovariectomy in dogs, but the clinical picture in dogs agree so closely with the known effects in laboratory rodents that one may risk some generalisations. The subjects in our three groups show significant differences in the levels of oestradiol and progesterone so that secondary differences may be expected.

The pituitary-adrenal interaction. Although the resting levels of cortisol prior to the adrenal stimulation test appear to be proportionate to the ultimate levels reached this cannot be regarded as significant, as in 61 resting samples previously assayed no group differences could be demonstrated. The steep rise in cortisol level after the single ACTH injection seen in Group III (Fig. 3) may be ascribed to availability of stored cortisol precursors and not to an increased synthetic ability in the ovariectomised bitch. A low concentration of oestradiol at the hypothalamic receptors decreases ACTH release during day to day activities, so that cortisol precursors in the adrenal cortex are not fully utilised. Repeated daily ACTH injections should deplete these stores to reveal a decreased cortisol synthetic ability[32].

From the results of our experiment it may be concluded that the ATOPA subjects of Group II show a response closest to that of the entire bitches of Group I. The changes in glucose level and differential count are parallel to the cortisol level and it is of interest to note that the eosinophil count was the least sensitive while the lymphocyte count showed the most consistent change[59].

Thyroid status. The inconsistency in the results obtained with the tests available to us agreed with reports in the literature[7]. Biopsy, TSH assay and thyroid synthetic capability after repeated TSH injection may yield further useful information.

Behaviour. One subject in Group I failed the test of work performance. Group II were all satisfactory. Of a further 3 ATOPA subjects which were trained and tested, one rated excellent (79%), one was satisfactory (56%) and one failed (50%). In Group III all 4 failed.

There was little difference between the groups as far as the obedience tests were concerned. Groups I and II did well in the obstacle course which required boldness and initiative, and Group III failed completely in this respect. Group I and II were agressive whereas none in Group III could be provoked or trained to display aggression.

The poor performance of the ovariectomised group is in accordance with previous experience in the Dog Unit of the SADF. The performances of Groups I and II seems to indicate that ATOPA does not have an adverse affect on the working ability of bitches.

CONCLUSIONS

On the basis of the results obtained in this experiment it may be concluded:

1. Canine ovaries will survive after transplantation to the gastric serosa in the manner described.

Table 4: BEHAVIOURAL ASPECTS

Subjects	Performance	Training potential
Group I		
Dog A	Timid, no aggression	58% no aptitude
Bv	Very obedient, satisfactory	68% as tracker dog
Bu	Obedient, very aggressive	82% as guard dog
D	Obedient, satisfactory	59% as tracker dog
Group II		
Dog F	Fairly obedient, satisfactory	68% as patrol dog
C	Obedient, very aggressive	70% as security dog
Do	Fairly obedient, very aggressive	81% as patrol dog
Ma	Obedient, very aggressive	70% as guard dog
Group III		
Dog An	Obedient, extremely timid	50% no aptitude
B	Very obedient, extremely timid	50% no aptitude
J	Obedient, very timid	58% no aptitude
M	Completely unstable	Discarded

2. Such a graft produces considerable quantities of oestradiol and progesterone.
3. Oestrus is abolished but brief occasional episodes of pro-oestrus may occur.
4. The levels of oestradiol in the general blood circulation of bitches which have been subjected to auto transplantation of the ovaries to the portal drainage area, are high enough to prevent development of the eunuchoid state which occurs in ovariectomised animals.

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CASE REPORT **GEVALVERSLAG**

SURGICAL TREATMENT OF AN UNUSUAL FRACTURE OF THE FIRST PHALANX OF A HORSE

G.E. FROST and H.R. DU PREEZ*

INTRODUCTION

A 3 year old thoroughbred colt in full training pulled up acutely lame during a training gallop on a dirt track.

According to the trainer no reason for the injury was evident. The horse refused to bear weight on the leg and when forced to walk back to the stables it would only touch the affected toe to the ground. The fetlock soon became swollen and was very painful. The trainer treated the animal for a sprain with ice packs and bandages. No improvement occurred in 72 hours and veterinary advice was sought.

Examination revealed the horse to be in obvious pain. He was unable to bear weight on the leg. The fetlock and pastern were swollen, especially posteriorly and painful to touch. Although no crepitation was felt, a fracture was suspected.

Anterior-posterior and lateral radiographs revealed a lateral longitudinal and slightly oblique incomplete fracture of the first phalanx extending from the metatarsophalangeal joint to the distal third of the first phalanx. The integrity of the proximal articular surface of the bone was disrupted. (See Fig 1)

As the fracture was incomplete it was felt that by means of inter-fragmentary compression, perfect realignment of the fragments was possible and surgery was advised.

SURGICAL PROCEDURE

Premedication consisted of 30mg of acetyl promazine* given intravenously. The horse was anaesthetised with 5g thiopentone sodium† intravenously and then maintained on halothane‡ and oxygen in a closed system.

The surgical site was prepared and draped to facilitate aseptic surgical technique. A transverse, curved skin incision was made over the anterior aspect of the proximal portion of the first phalanx just below the fetlock joint and in such a manner as to mobilise a skin flap which could be retracted proximally. This exposed the underlying extensor tendon and fibrous tissue covering the bone. (Fig. 2A and 2B)

A small vertical incision was made on either side of the tendon onto the bone about 1,5cm below the articular surface of P1. Using a drill guide to protect soft tissue a 4,5mm hole was drilled through the near fragment on either side of the tendon and parallel to the articular surface. A drill sleeve was then inserted into each hole in turn and a 3,2mm hole drilled into the far fragment. These holes were tapped with a 4,5mm tap and two 4,5mm cortical screws of pre-measured length were inserted and tightened. With the larger holes in the near fragment acting as glide holes, the screws compressed the fragments by the lag principle[3]. The screws were tightened sufficiently to practically obliterate the fracture line, as indicated by the post operative X-ray photographs. (Fig 3A and 3B)

The skin wound was closed with single interrupted sutures and the leg was bandaged and cast with resin impregnated glass fibre tape§ from the hoof to just below the hock.

The cast cracked when the horse stood up after the anaesthesia but it fully supported the fracture during the critical period. Twenty four hours later the cast was removed and the leg put in a heavy supporting bandage. It was immediately apparent that the patient found this more comfortable.

Post operatively 6 million units of procaine penicillin and 3 g of streptomycin were administered as well as 3000 IU tetanus antitoxin. The animal also received 20mℓ suxibuzone.¶ Post operative antibiotic and anti-inflammatory therapy were continued for 3 days.

At this time the horse walked around in its box with hardly any sign of lameness. It was at first thought that

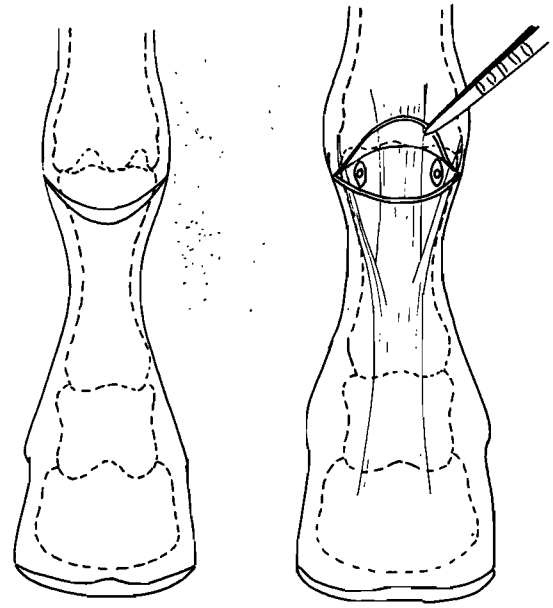

Fig. 2a Fig. 2b

3 Darnov Avenue, Bordeaux, 2194, Randburg.

*Acetylpromazine – Boots Pure Drug Co., Ltd Nottingham, England.
†Intraval Sodium – Maybaker (SA) (Pty) Ltd., Port Elizabeth.
‡Fluothane I.C.I. (Pty) Ltd., Johannesburg.
§Lightcast – M.S.D. (Pty) Ltd.
¶Suxibuzone – Chemveld Agricultural Services (Pty) Ltd.

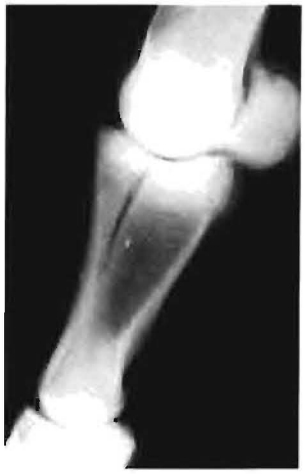

Fig. 1

Fig. 3a

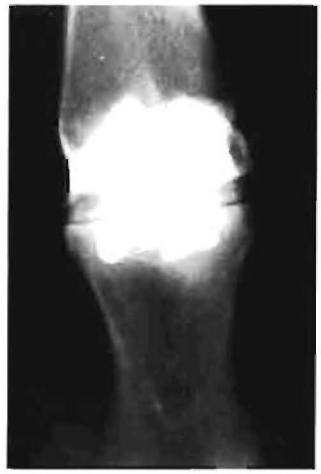

Fig. 3b

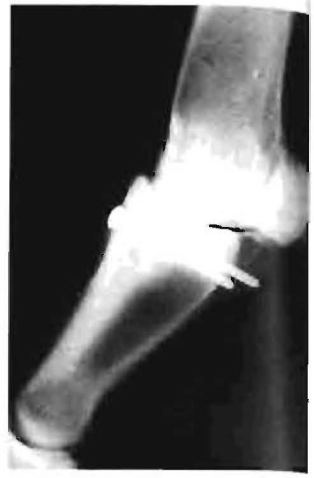

Fig. 4

the analgesic effect of the drugs was responsible for the apparent lack of discomfort but although treatment was discontinued, progress was remarkable. The patient was discharged from the hospital after 1 week when skin sutures were removed. Instructions were given for it to be confined to a loose box for 8 weeks.

Follow-up radiographs taken 9 weeks later showed excellent primary bone union with no joint deformity. The nature of the fracture allowed accurate anatomic realignment of the two fragments and modern implants and techniques ensured rigid internal fixation by interfragmentary compression. Primary bone union occurred with a fully functional joint. (Fig. 4)

DISCUSSION

Fracture of the first and second phalanges is most common among horses which are required to make very sharp turns, when most of the animal's weight becomes centred on one leg. These fractures occur in both fore and hind limbs, but are more common in the latter.[1]

Anterior chip fractures of the fetlock joint involve the anterior rim of the first phalanx, usually on the medial side. These fractures occur mainly in the front limbs.[2]

This case is unusual in that the fracture occurred on the straight and not while the animal was turning sharply. The fracture is also unusual in being in the lateral plane. These fractures are usually anterior-posterior due to the wedge effect of the sagittal ridge of the distal end of the large metatarsal bone in the intermediate groove of the proximal articular surface of the first phalanx. More commonly they are multiple or comminuted. Quite severe tortional forces must have been brought to bear on the affected bone at the moment of impact with the ground.

The two compression screws were placed as close to the proximal articular surface as possible for three reasons:

(a) The intra-articular fracture line should be obliterated to obtain most effective cartilage healing.
(b) The mechanical forces created by the weight of the animal and its movements were thought to be brought to bear primarily on that part of the fracture.
(c) The bone is thickest in that area.

As bony reaction around the screw heads was minimal, it was decided not to remove the screws.

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BOOK REVIEW — BOEKRESENSIE

DIE KLINISCHE UNTERSUCHUNG DES RINDES

GUSTAV ROSENBERGER EDITOR

2nd Ed Verlang Paul Parey – Berlin und Hamburg
pp XV + 531, Figs 478 (17 colour), 52 synopsis, Price not stated

This work is divided into the following main sections:

The first 57 pages are concerned with handling and casting of the bovine animal as well as a general review of tranquillization, analgesia, neurolepsy and briefly, control by drugs and anaesthesia.

This section is succeeded by 33 pages dealing with anamnesis, signalment and general clinical examination including bodily condition, temperature and respiratory and heart rate i.e. it describes the phase of examination during which the clinician sees the animal as a whole and gains an overall clinical impression.

The rest of the book is devoted to special examination methods. The various organs and systems are attended to in detail e.g. 18 pages are taken up individually by hair, skin, subcutis, visible mucous membranes and horns.

Examination methods are described minutely and with thoroughness. To give a few examples of the methods: extensive use is made of auscultation and percussion together with various manoeuvres to gain more information; radiological investigation is included wherever indicated; endoscopic methods like peritoneoscopy and cystoscopy are described; the various methods of biopsy with indications and interpretation are given; laboratory techniques are extensively employed.

The book therefore does not concern itself with treatment nor primarily with diagnosis but with the wide range of examination methods available to the clinician, the employment of which will vastly enhance his diagnostic acumen.

The book is most highly recommended. Particularly with the increase of large animal hospitals in South Africa, clinicians will be more and more in the position to employ from day to day the sophisticated clinical procedures described, thereby increasing their professional satisfaction and efficiency.

The illustrations, paper and print are of excellent quality.

CFBH

BOOK REVIEW — BOEKRESENSIE

THE VETERINARY ANNUAL

Ed. C.S.G. GRUNSELL and F.W.G. HILL, 17th issue

Wright-Scientechnica, Bristol 1977
pp XX + 318, Figs 92 Tabs 32, Published price £11,50

The latest edition of the Veterinary Annual contains articles by 55 authors, seven from outside the United Kingdom. It continues to fulfil its declared role of presenting to the profession, and particularly those in the field, some of the major recent advances in veterinary science. As usual, a wide field is covered with papers on all species and also six multi-species reviews on subjects which include reproduction and infertility and helminthology.

The section on cattle (9 articles) includes papers on preventive medicine in practice, current trends in infectious opthalmia, aspects of clinical differential diagnosis of degenerative myopathy (recently described in Rhodesia) and a discussion of antibiotics and mastitis by Bywater. There are seven papers on pig topics, mostly of preventive medicine importance, while two of the five papers on equine subjects are of particular interest, dealing with nutrition and the problems of long distance rides. The small animal practitioner will find eleven topical articles, including 2 papers on the canine hip, one on surgery and the other on differential diagnosis. Other subjects dealt with in this section include canine distichiasis, distortion of the distal radius – ulna, diabetes mellitus, recent advances in canine dermatology ("Demodectic mange remains one of the great unsolved mysteries of canine dermatology"), anal and perineal canine disease and intractable feline stomatitis.

The miscellaneous section contains the longest article in the book – a most informative paper on acupuncture. Other subjects dealt with here include game immobilization, disinfection techniques and serum enzymes in diagnosis. This publication is of inestimable value to busy professional people, particularly clinicians, and will widen the horizons of all who will spare the time to read it.

RKL

Cow infertility problems?

Now there's a simple and effective new treatment "Estrumate"–ICI's synthetic prostaglandin analogue

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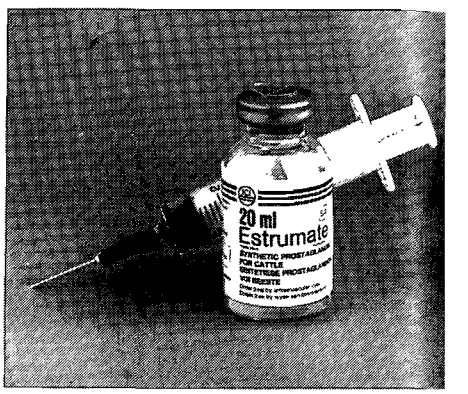

ICI **ESTRUMATE**

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THE INDUCTION OF OESTRUS IN RECENTLY WEANED SOWS WITH LOW DOSES OF GONADOTROPHINS

P.B.D. WHYTE*

ABSTRACT: Whyte, P.B.D. **The induction of oestrous in recently weaned sows with low doses of gonadotrophins.** *Journal South African Veterinary Association* (1977) **48** No. 2, 129 – 131 (En). Nat. Pig Health Scheme, Box 1357, 0001 Pretoria, Republic of South Africa.

Trials in a large scale commercial piggery indicated that use of a combination of hormones played a significant role in synchronising and expediting the onset of oestrus. The litter sizes in the treated group were significantly larger than in the controls.

INTRODUCTION

The cause of postweaning anoestrus in sows recently weaned of their litters has not been clearly defined. It would seem that sub-standard management, genetics and inadequate nutrition are individually or collectively responsible. The use of hormones for oestrus synchronisation has been introduced with a view not only to batch-weaning of piglets and batch-breeding of sows but also to effecting increased conception and farrowing rates. Various drugs including gonadotrophins[1,3] and oestrogen-prostaglandin[2] combinations, have been employed to this end.

This paper describes the results of a trial conducted in a large-scale commercial operation to determine the extent to which relatively low doses of gonadotrophin (FSH + LH) affect returns to oestrus and farrowing rates in sows which have recently been weaned of their litters.

MATERIALS AND METHODS

The trial was conducted in a large-scale commercial piggery where from 25 to 35 litters are weaned weekly. Purebred Landrace (L) and Large White (LW) sows as well as two breed (LW × L) and three breed cross females (LW × L × German Landrace) were involved.

The trial was replicated every fortnight over a three-month period from March to July 1975. On every occasion sows were randomly allotted to two groups of approximately equal numbers (Table 1) immediately after their litters were weaned. First litter sows were equally allocated to the two groups. One of the groups was designated the control group and the other the treated group.

Whereas sows in the control groups were left untreated, experimental animals received a single subcutaneous injection of 400 IU of follicle stimulating hormone (FSH) plus 200 IU of human chorionic gonadotrophin (HCG) as source of luteinizing hormone 1 day post-weaning. Litters were weaned on Thursdays and repeat-mated at 24 hour intervals after coming into oestrus 5 to 6 days after the litter was weaned. After service the sows are housed in individual stalls until farrowing. As is customary on most commercial farms, piglets are transferred between sows to equalise litter size and thus minimize pre-weaning mortality. No further data were kept after farrowing.

RESULTS

The conception rates and other data of the two groups are summarized in Table 2.

In the treated group, 86 of the 93 animals (92,4%) farrowed within 115 days of first service following weaning compared to 84 (85,7%) of the untreated sows.

Days from weaning to service

The results of the experiment are summarized in Table 3. The difference in favour of the treated group is significant at the 99% level.

There is a strong between-period difference. This is due mainly to the variability of the untreated sows in coming into oestrus, the treated groups having a much tighter distribution of days from weaning to service. The treatment-period variability shows a trend towards a decreasing number of days between weaning and oestrus in both the treated and untreated groups, with the untreated and treated groups showing significant variations. The reasons for this treatment-period variation are unclear.

Farrowing rates

The farrowing rates after the two treatments are shown in Table 4.

Table 1: TRIAL PERIODS

Trial number	1	2	3	4	5	6	7	No of sows
Number untreated	12	21	10	15	15	12	13	98
Number treated	15	15	11	15	15	13	9	93
Period totals	27	36	21	30	30	25	22	191

*Co-ordinating Veterinarian, National Pig Health Scheme, Livestock and Meat Industries Control Board, Box 1357, 0001 Pretoria.

Table 2: CONCEPTION RATES

	Number of sows	Returned to service	Not in pig	Culled	Died	Aborted	Farrowed to first service	Total
Untreated	98	7	2	3	1	1*	84 (85,71%)	98
Treated	93	2	—	3	1	1	86 (92,4%)	93
							Total	191

*One sow produced a litter of mummified foetuses.

Table 3: DAYS FROM WEANING TO SERVICE

Trial number	1	2	3	4	5	6	7	Weighted mean
Untreated	7,667	7,429	5,10	5,2	5,533	4,917	5,231	5,990
Treated	4,733	4,267	4,455	6	4,067	4,231	4,556	4,634
Weighted mean	6,037	6,111	4,762	5,600	4,800	4,56	4,955	5,33

LSD_{95} = 1,031 days LSD_{99} = 1,357 days

Table 4: FARROWING RATES AS A PERCENTAGE OF SOWS SERVED

Trial number	1	2	3	4	5	6	7	Weighted mean
Untreated	91,67	80,95	80	80	86,67	91,67	92,31	85,71
Treated	93,33	93,33	100	86,67	93,33	92,31	88,89	92,47
Weighted mean	92,59	86,11	90,48	83,33	90	92	90,91	89,01

The difference of 6,76% in favour of the treated group is not significant, probably due to the large variation in farrowing rates between periods in both treated and untreated groups.

Table 5: TOTAL NUMBER OF PIGLETS BORN PER LITTER

Trial number	1	2	3	4	5	6	7	Weighted mean
Untreated	9,909	11,353	9,375	12,417	9,769	10,818	11,00	10,762
Treated	13,357	12,643	12,727	12,077	11,143	10,667	10,00	11,919
Weighted mean	11,84	11,936	11,316	12,24	10,487	10,739	10,60	11,347

LSD_{95} = ,774/litter LSD_{99}= 1,04/litter

Table 6: NUMBER OF LIVE PIGLETS BORN PER LITTER

Trial number	1	2	3	4	5	6	7	Weighted mean
Untreated	9,364	10,765	11,417	9,077	10,273	10,25	10,19	
Treated	12,286	12,143	11,455	11,308	10,214	10,333	9,375	11,302
Weighted mean	11,00	11,387	10,474	11,36	9,667	10,304	9,90	10,629

LSD_{99} = ,968 piglets/litter

Number of piglets born per litter

The total number of piglets per litter are provided in Table 5.

The difference in favour of the treated group is again favourable at the 99% level. However, the interperiod difference and the intra-treatment variation is also significant, with the greatest variation being shown in the treated group. The reason for the inter-period variation is unknown but the clear trend in the treated group of a litter size that progressively deteriorates over the duration of the trial is suggestive of a drug deterioration with increasing length of storage time.

Number of live piglets born per litter

The differences between the number of live piglets born per litter in the treated and control groups are summarized in Table 6.

The difference in favour of the treated group is significant at the 99% level. The inter-period and intra-group variation is also significant. This indicates that a variable other than the hormone, plays a significant role in the results.

DISCUSSION

The results of the trial indicate that the hormone combination played a significant role in synchronizing oestrus and bringing the sows into oestrus more quickly than would occur naturally.

The difference in litter size, both the live born piglet and total piglet litter size, is significantly superior in the treated group. However, other variables in addition to the drug play a significant role. This is evidenced by the intra-treatment variation. These variables, operating either singly or in combination, include the fertility level of the boars, management, weather patterns, condition of the sows at the time of weaning their litters, nutritional status, genetic and breed influence, and other factors.

CONCLUSION

It would appear that the hormone is cost effective at the current drug: profit per pig price ratio, when used as routine on all sows 1 day post-weaning. The economics of this practice would, however, vary with changes in the price ratio. The piglet mortality rate from birth to weaning would also be a factor to consider. For technical reasons the comparative mortality rates were not studied.

Before a firm recommendation can be given, further work on the cause of the observed decline in litter size in the treated group is desirable.

ACKNOWLEDGEMENTS

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INFORMATION INLIGTING

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ETLIKE GEBRUIKE VAN CATOSAL® BY GROOTDIERPRAKTYKE IN SUID-AFRIKA

OPSOMMING

Spesifieke gebruike van die assimilasie-middel of sog. "metaboliese stimulant" CATOSAL® is bespreek, veral met betrekking tot wintertoestande op die hoëveld.

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Butaphosphone, die organiese vorm van fosfor wat in Catosal® teenwoordig is, het 'n gunstige invloed op feitlik al die assimilasie-prosesse in die liggaam, volgens Blum. Hy noem dus hierdie groep chemiese stowwe "assimileerders" (assimilators). Fosfor in hierdie vorm word uitgeskei kort nadat dit die metaboliese prosesse beïnvloed het, maar Sommer het bewys dat die effek nog opmerkbaar is lank nadat uitskeiding voltooi is.

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2. Spesifieke gebruike:

Dit is duidelik dat die indikasies van hierdie middel by algemene praktyk velerlei van aard is, en is op hierdie stadium glad nie almal ondersoek nie.

Spesifieke gebruike vir toestande in grootdiere in Suid-Afrika is:

(i) Metaboliese versteuringe by melkkoeie: Die hoogproduserende melkkoei is vatbaar vir 'n hele reeks metaboliese steuringe van verskillende oorsprong. Die bevordering van die normale assimilasie-proses hou voordeel in by al hierdie toestande en veral by die sog. "downer" koei. Hierdie feit het oorsprong gegee aan die gewoonte onder veeartse om Catosal "die regmaker" ("the pick-me-up") te noem. Van die veearts word dikwels verwag om wonderwerke te verrig by so 'n dier. Hoe gouer die bees weer op die been kom, hoe meer vertroue ontwikkel die eienaar in sy veearts.

(ii) Die veldkoei wat aan die einde van 'n strawwe winter swaar dragtig is. Praktisyns ken die verskynsel van die maer koei wat gaan lê om te kalf, en net eenvoudig moed verloor. Vertering van die harde ruvoer in haar rumen is klaar 'n probleem; assimilasie van verteerde stowwe is onvoldoende, en daarby is die koei aan hewige stremming blootgestel. Baie ervare praktisyns gebruik onder andere 'n groot binneaarse dosis Catosal® om so 'n koei weer in 'n positiewe balans te kry.

(iii) Wanvoeding of die voer van ongebalanseerde rantsoene by beeste gedurende die wintermaande, wat aanleiding gee tot simptome soos kroniese maagversteuring, verminderde eetlus, vertraging van melkproduksie en selfs onvrugbaarheid.

(iv) Die kronies siek dier wat simptome toon van verminderde weerstand, gewigsverlies of vertraagde groei.

(v) Die galsiekte geval. Behalwe die behoefte om rooibloedsel-vervaardiging te versnel, het die bees wat anaplasmose opgedoen het ook met 'n assimilasieprobleem te kampe, wat die herstelproses heelwat kan vertraag.

(vi) Die nie-voerder. Ossies wat nie by 'n voerdery wil aanpas nie as gevolg van 'n tydperk van wanvoeding, uitputting a.g.v. die vervoer, swakheid a.g.v. parasietbesmetting, ens.

KOMMENTAAR

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THE REGULARITY OF CYCLES AND OVARIAN EFFICIENCY IN A GROUP OF FRIESLAND COWS

C. MAREE*

ABSTRACT: Maree, C. Regularity of cycles and ovarian efficiency in a group of Friesland cows. *Journal of the South African Veterinary Association* (1977) **48** No. 2, 133 – 135 (En) Dept. Anim. Prod., Fac. Agric., University of Pretoria, 0002 Pretoria, Rep. of South Africa.

Delayed ovulation was recorded in 17,34% and anovulation in 17,66% of 623 oestrus periods in 32 Friesland cows. Cycle length was abnormal in 19,19% of 568 cycles and was associated with an increase in the incidence of defective ovulation. Inseminations failed in 24,6% of cases despite normal ovulatory function at oestrus while in nine out of 134 inseminations sperm retained its fertilizing capacity in the female tract between 24 and 72 hours.

INTRODUCTION

Functional derangements are known to play a role in the conception rate of the bovine. Van Rensburg & De Vos[8] investigated the incidence of ovulatory failure at 536 oestrus periods in 118 Friesian and 161 Afrikander cows and heifers and recorded defective ovulation at 140 periods. Delayed ovulation was recorded in 93 (66%) of these cases and anovulation in 47 (34%). Further reports are available on the incidence of silent oestrus and ovulatory failure in groups of cattle over short periods[3 4 5 7] and in which nutritional, seasonal and hereditary aetiological factors have been indicated.

In this investigation the ovulatory efficiency and cycle lengths at all consecutive oestrus periods and cycles were recorded in Friesland heifers during maturation and after calving for the first three successive breeding periods and up to an average age of 52 months.

PROCEDURE

A group of 32 Friesland heifers of comparable age were subjected to palpation of the genitalia and description of the ovaries twice weekly from the age of 7 months through maturation and insemination until pregnancy was confirmed. After calving, palpations were resumed and maintained for a further two breeding periods. Observations for signs of oestrus were carried out at frequent intervals throughout every day by an observer employed solely for this purpose. Reports of overt signs of oestrus were followed up by palpation and description of the ovaries not less than 12 hours from the onset of signs. Additional palpations were then maintained at intervals of not less than 24 hours until ovulation or regression of the follicle(s) was established. Delayed ovulation was recorded when ovulation occurred later than at the time of the second palpation at oestrus which corresponded to more than 36 hours from the onset of heat.

This criterion is justified by the length of the oestrus period (mean 19,3 range 13 to 27 hours) and the time of ovulation in relation to the end of oestrus (10,7 hours) as reported by Cole & Cupps[2]. In the investigation of Van Rensburg & De Vos[8], however, the average duration of oestrus in the dairy cow was taken as 16 hours and the average interval between oestrus and ovulation as 10½ hours.

Heifers were inseminated at a body mass of 300 kg. Cows that returned to service after the 4th insemination were removed from the group while cows that failed to re-cycle due to abnormalities like cystic ovaries or adhesions of the genitalia were naturally excluded from the records.

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RESULTS AND DISCUSSION

The ovulation record at oestrus and the corresponding cycle lengths up to confirmation of the second pregnancy in those cows that re-conceived, are summarized in Table 1.

The overall pattern of ovulatory efficiency and cycle lengths after this stage appeared unaltered and further details are excluded for the sake of brevity. Neither did delayed breeding after calving seem to have any detrimental effect on efficiency of ovulation or cycle lengths.

Of 32 cows, only 17 survived the full duration of the trial. Three animals died, three never conceived and a further nine cows were eliminated at various stages due to post partum complications, chronic laminitis etc.

Table 2 provides a comparison of the incidence of defective ovulation before and after the first calving.

Conceptions resulting from a total of 134 inseminations carried out during the entire period concerned in relation to normal or defective ovulations at oestrus, are summarized in Table 3.

The apparent ease with which normal and abnormal ovulations and cycle lengths interchange in cows of comparable age and management (Table 1) and the marked variation between individuals in the group, illustrate the precariousness of endocrine interactions in reproduction. Heifers that conceived readily (e.g. numbers 2, 5, 6, 20, 30) were characterised by a high frequency of normal ovulations and cycle lengths immediately after the onset of puberty while heifers that were destined for low fertility (e.g. numbers 11, 13, 23, 24) showed aberations at an early stage. This was confirmed when heifers removed from the group on account of a high frequency of functional disturbances before breeding, required 3,2 inseminations per calf born and in addition 20% failed to conceive as against 1,5 inseminations and 95% calving rate in the rest.

Of 623 oestrus periods 218 (35%) were subjected to defective ovulations, viz. delayed ovulation, 108 periods (17,34%) and anovulation, 110 periods (17,66%). The incidence of anovulation decreased after the first calving from 26,34% to 11,36% of oestrus periods with a consequent increase in the percentage of normal ovulations. This might have been influenced by the removal of certain individuals from the group before the first calving (e.g. numbers 11 and 23).

Table 1: THE SEQUENCE OF NORMAL OVULATION (+), DELAYED OVULATION (Θ) AND ANOVULATION (O) AT OESTRUS IN RELATION TO THE CORRESPONDING CYCLES OF NORMAL (–) OR ABNORMAL (IN DAYS) LENGTH

Cow No.	
1	$O^{4}O^{12}O^{-}\Theta^{-}+^{-}+^{-}+^{-}+//+^{-}+^{-}O^{-}+^{-}\Theta^{12}O^{-}+^{25}+^{-}+^{-}+^{-}+^{-}+^{-}\Theta^{-}+^{-}+^{-}+//$
2	$+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+//+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{27}+^{-}+^{-}+^{-}+^{-}+^{-}O^{6}O^{-}+^{-}+//$
3	$+^{28}+^{13}+^{30}\Theta^{-}+^{-}+//+^{11}+^{-}+^{30}+^{-}+^{13}+^{-}+^{-}+^{-}+^{-}+^{-}\Theta^{-}O^{-}+^{-}+^{-}+^{-}+//$
4	$\Theta^{-}+^{-}+^{-}O^{-}+^{-}+^{-}+^{25}+//+^{14}+^{-}+^{31}+^{-}+^{-}+^{25}+^{33}+^{14}+^{11}+^{-}+^{-}+^{-}+^{28}+^{-}+//$
5	$+^{-}+^{-}+^{-}+^{14}\Theta^{-}+^{-}\Theta//+^{-}O^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}O^{-}\Theta^{-}+^{-}O^{-}O^{-}+^{-}+//$
6	$+^{42}\Theta^{-}+^{-}+^{-}\Theta^{-}\Theta//+^{-}+^{4}O^{40}+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}\Theta^{-}+^{-}+^{-}+^{-}+^{-}+//$
7	$+^{10}+^{-}\Theta^{-}\Theta^{-}+^{-}+//+^{-}+^{-}+^{-}\Theta^{-}+^{-}+^{-}+^{-}+^{-}\Theta^{-}\Theta^{-}+^{-}+^{26}+^{-}\Theta//$
8	$\Theta^{-}+^{-}+^{-}+^{-}\Theta^{-}\Theta^{-}+^{-}+//\Theta^{37}O^{-}O^{-}O^{-}\Theta^{-}+^{-}O^{46}\Theta^{-}\Theta^{-}+^{-}O^{-}O^{26}+^{-}+//$
9	$+^{-}O^{-}\Theta^{-}O^{-}+^{-}+//+^{9}\Theta^{-}\Theta^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{25}+^{-}+^{-}\Theta^{-}+^{-}+^{14}+^{-}+^{-}+//$
10	$+^{47}+^{-}\Theta^{-}+^{-}+^{-}\Theta//+^{-}\Theta^{-}\Theta^{-}O^{-}+^{35}+^{-}+^{-}\Theta^{-}+^{-}\Theta^{-}+^{-}+^{-}+^{-}\Theta^{-}+//$
11	$O^{-}O^{-}O^{-}O^{-}\Theta^{11}\Theta^{11}\Theta^{-}+^{31}+^{-}O^{10}\Theta^{-}\Theta^{-}+//$
12	$O^{-}O^{-}O^{-}+^{-}O^{-}+^{-}+^{-}+^{-}O^{-}O^{-}\Theta^{-}+//+^{-}+^{-}+^{26}+^{30}+^{-}+^{-}\Theta^{32}O^{-}+^{-}+^{27}O^{-}O^{-}O^{26}\Theta^{27}+^{-}+$
13	$O^{33}O^{-}O^{-}O^{-}\Theta^{-}+^{-}O^{-}+^{-}O^{-}+//+^{-}\Theta^{-}+^{-}\Theta^{-}+^{-}O^{-}O^{-}O^{-}O^{-}O^{-}O^{9}O^{12}\Theta^{-}\Theta^{-}+^{-}+^{-}+//$
14	$O^{66}+^{-}+^{-}+^{-}+//\Theta^{25}O^{25}\Theta^{25}+^{25}+^{29}+^{28}+^{26}+^{-}+^{28}+^{25}O^{28}\Theta^{33}O^{27}+^{33}+$
15	$O^{-}O^{-}+^{-}+//$
16	$+^{-}O^{-}O^{-}+^{-}+^{-}+^{-}\Theta^{-}O^{-}\Theta//\Theta^{-}+^{-}+^{-}\Theta^{-}+^{-}\Theta^{25}+^{32}+^{-}+^{-}+^{-}+^{-}+^{-}+//$
17	$O^{-}O^{-}O^{56}+^{-}\Theta^{-}\Theta^{-}+^{-}+^{58}O^{-}+//+^{-}+^{-}+^{-}+^{-}+^{-}\Theta^{-}\Theta^{-}+^{-}+^{-}+^{-}\Theta^{-}+^{-}+^{-}+^{-}O^{-}+//$
18	$+^{-}+^{39}+^{37}+^{-}+^{-}\Theta^{-}O^{6}O^{-}\Theta//+^{-}\Theta^{-}+^{-}+^{-}+^{25}\Theta^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+//$
19	$+^{-}+^{-}+^{-}+^{-}O^{-}+^{44}\Theta^{180}O$
20	$+^{-}O^{-}+^{-}O^{-}+^{-}\Theta^{25}\Theta^{-}+//+^{31}+^{-}+^{-}+^{-}O^{-}\Theta^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{28}+^{-}+^{-}+//$
21	$O^{-}+^{25}+^{45}O^{-}\Theta^{-}\Theta^{-}+//+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}+^{-}\Theta^{-}\Theta^{-}+^{-}+^{-}+//$
22	$+^{-}O^{44}+^{53}O^{7}O^{-}+^{-}+^{26}+^{-}O^{-}\Theta^{-}+^{-}O^{-}\Theta^{-}+//$
23	$O^{-}O^{28}O^{30}O^{11}O^{6}O^{14}O^{26}O$
24	$\Theta^{-}\Theta^{-}\Theta^{-}\Theta^{-}\Theta^{-}\Theta^{-}+^{-}+^{-}+^{-}\Theta//+^{35}\Theta^{-}\Theta^{30}O^{-}+^{-}\Theta^{-}\Theta^{-}+^{-}\Theta^{-}\Theta^{-}\Theta^{-}O^{25}\Theta^{-}+//$
25	$O^{-}O^{38}+^{-}+^{-}O^{-}+^{-}+//+^{-}+^{-}+^{-}+^{-}+^{25}+^{-}O^{-}\Theta^{25}+^{-}+^{-}+^{-}\Theta^{-}+^{-}+^{-}+//$
26	$+^{-}\Theta^{-}+^{-}+^{-}+^{14}+^{8}+//$
27	$+^{-}+^{-}+^{37}O^{-}+^{-}O^{-}+^{-}+//+^{26}O^{14}\Theta^{-}+^{-}+^{27}+^{-}+^{-}+^{-}\Theta^{-}\Theta^{-}\Theta^{-}\Theta^{-}+^{-}+^{-}+//$
28	$O^{-}+^{-}+^{-}+^{-}+^{27}+^{-}O^{-}+//$
29	$O^{30}O^{7}\Theta^{53}+^{-}+^{-}+^{-}+//$
30	$+^{51}+^{-}+^{-}+^{38}+^{-}+//+^{-}+^{-}+^{-}+^{-}+^{-}\Theta^{-}+^{-}+^{-}+^{-}+^{-}+^{25}O^{-}\Theta^{29}\Theta^{-}+//$
31	$+^{37}O^{-}O^{-}+^{-}+^{-}+^{-}+^{-}+^{-}O^{-}+^{35}+^{-}+^{-}+^{-}+^{-}O^{-}O^{-}+^{-}+$
32	$+^{-}+^{30}\Theta^{-}+^{-}+//+^{-}+^{-}\Theta^{-}O^{-}+^{-}+^{-}+^{7}O^{-}O^{-}+^{-}\Theta^{-}+^{-}\Theta^{-}O^{-}+//$

// = Pregnancy

Table 2: THE INCIDENCE OF DEFECTIVE OVULATION BEFORE AND AFTER CALVING IN FRIESLAND COWS

	Normal ovulation	Delayed ovulation	Anovulation
Total oestrus periods, 623	405 (65,01%)	108 (17,34%)	110 (17,66%)
Before calving 262 periods	148 (56,49%)	45 (17,18%)	69 (26,34%)
After calving 361 periods	257 (71,19%)	63 (17,45%)	41 (11,36%)

Table 3: CONCEPTION RATE OBTAINED FROM A TOTAL OF 134 INSEMINATIONS IN RELATION TO NORMAL OR DEFECTIVE OVULATION AT OESTRUS

Conception in relation to ovulation	Inseminations No	% of total
Normal ovulation followed by conception	59	44,0
Normal ovulation without conception	33	24,6
Delayed ovulation followed by conception	9	6,7
Delayed ovulation without conception	16	12,0
Anovulation	17	12,7
Total	134	100,0

The first four oestrus periods after the onset of cyclic activity, however, were also characterised by a higher overall incidence of defective ovulations than afterwards which shows that reproductive maturation in the heifer proceeds after the attainment of puberty.

Of 568 cycle lengths recorded, 109 (19,19%) were of abnormal length (less than 15 or more than 24 days). Before calving 21,3% of cycles were of abnormal length as against 14,5% after calving, while 29 (26,61%) of 109 cycles were shorter than normal and 80 (73,39%) longer than normal.

A corpus luteum (CL) was palpable only during approximately one half of all recorded cases of prolonged cycles. In the other cycles a CL had either not developed following anovulation at oestrus or it became unpalpable at the end of a normal cycle length. At oestrus periods following on cycles where a persistent CL could be palpated, ovulatory defects were recorded in only 22% as against 35% in the total number of periods. This substantiates reports that conception rate in cows recovering from retained corpora lutea is as high as that in normal cows[6].

The interaction between factors responsible for cycle length and for ovulatory efficiency is not clear. A higher incidence of defective ovulation (50,47%) was, however, recorded at oestrus periods following cycles of aberrant length than the overall incidence (35%). Maintenance of normal cycle length however, appeared to be independent of the ovulatory efficiency at the preceding oestrus period, 75,9% of all periods with ovulatory defects having been followed by cycles of normal length.

Of a total of 134 inseminations distributed over the group of cows, 33 inseminations (24,6%) failed to result in pregnancy at oestrus periods where normal ovulation was recorded (Table 3). This indicates the possible role that tubal or uterine changes might play in clinically normal cows[1] since semen of known high fertility was used. The paradoxical incidence of conception following a delayed ovulation provides information on extreme survival times of sperm fertilising capacity in the female tract. In 9 cases quoted in Table 3, the following periods were recorded:

24 hours	5 times
36 hours	once
48 hours	twice
72 hours	once.

The question arises whether improved techniques of sperm evaluation and storage might have played a role.

Age and all extrinsic factors were comparable for the animals employed in this study. Variation between animals and within the same animal between oestrus cycles and periods can be regarded as the normal physiological variation and the regular extremes involved in the bovine reproductive process. These variations might well play a role in the unpredictability of responses that are obtained with exogenous reproductive hormone administration in the bovine.

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BOOK REVIEW — BOEKRESENSIE

BEEF CATTLE PRODUCTION IN DEVELOPING COUNTRIES

A.J. SMITH, EDITOR

University of Edinburgh 1976
pp XV + 487, Figs 50, Tabs 94, Price not indicated

More than half of the world's population of cattle (660 million head of a total of 1 150 million) is found in developing countries. These cattle are generally unspecialized in function, and have many different uses; they act as one of the main sources of power in agriculture and provide fuel (dung) for domestic use; they supply milk and meat; act as repository of wealth and fulfil numerous social and religious functions in various parts of the world. Therefore it is not surprising that the output of meat from cattle in developing countries is usually very low. The reasons for this low level of production are legion involving nutritional, sociological, economic, climatic and genetic factors and disease.

A conference, held in Edinburgh in September 1974, was organized by the Centre for Tropical Veterinary Medicine to discuss the effects of these various constraints on beef production and how the output of beef could be increased from cattle in the developing world. Thirtyfour speakers from many different disciplines took part, including geneticists, nutritionists, veterinarians, physiologists, economists, agronomists, geographers and social anthropologists. The conference was attended by delegates from 36 countries, and the discussion contributions are also included in the book.

There is no doubt that the aim of the conference was achieved i.e. to provide a comprehensive and up-to-date review of the problems of beef cattle production in developing countries. The hope is expressed that it will make a contribution towards helping the developing world to provide some of the extra food urgently needed for the world's growing population. The book can be highly recommended to South African students, veterinarians, animal scientists and even social anthropologists, because it gives a wealth of information usable in our own country.

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ISOIMMUNE BLOOD GROUP ANTIBODIES IN CATTLE AFTER THE USE OF A BLOOD VACCINE

D.R. OSTERHOFF* & A.J. DE VOS††

ABSTRACT: Osterhoff D.R.; De Vos A.J. Isoimmune blood group antibodies in cattle after the use of a blood vaccine. *Journal South African Veterinary Association.* (En) No. 2, 139 – 141 Faculty of Veterinary Science, Box 12580, 0110 Onderstepoort, Rep. of South Africa.

A total of 100 animals were inoculated twice within 21 days with the Onderstepoort babesiosis vaccine. High blood group antibody titres were recorded, especially after the second inoculation. The possibility is discussed of neonatal isoerythrolysis occuring after vaccination as a result of maternal isoimmune blood group antibodies gaining access to the circulation of the offspring which inherited the corresponding blood group factors from its sire.

INTRODUCTION

Babesiosis (redwater) and anaplasmosis (gallsickness) remain two of the most important infectious diseases of cattle in South Africa and result even today in the annual loss of thousands of animals. Measures to control these diseases vary depending on the epizootiological situation in each area and range from simple vector control aimed at eradication, to the establishment of an immune population through vaccination. Due to the present inability to culture the causative organisms *in vitro* the vaccines must necessarily consist of the blood of infected animals.

The anaplasmosis vaccine used in South Africa consists of pooled blood of up to 8 animals carrying *Anaplasma centrale,* a parasasite of low virulence which affords significant cross-immunity against the virulent *Anaplasma marginale.* Each dose, consisting of 2 mℓ whole blood, is administered subcutaneously and a single inoculation should confer life-long protection. This vaccine has been used successfully in this country for many years and the annual production reached a level of nearly one million doses during 1975/76. In a total cattle population of 13 million this represents an annual inoculation rate of approximately 7%.

The Onderstepoort babesiosis vaccine consists of pooled blood of up to 4 animals and is prepared essentially as described by Callow & Mellors[1] the only major difference being the incorporation of both *Babesia bovis* and *Babesia bigemina.* At the time of this study each dose consisted of 5 mℓ whole blood but, for purposes of standardisation, this has since been reduced to 2 mℓ. As in the case of the anaplasmosis vaccine it is also administered subcutaneously. Annual production is approximately 100 000 doses. The parasite strains used in the vaccine are not avirulent and may cause moderate or severe reactions. If this happens chemotherapy is indicated which should not interfere with the development of durable immunity to *B. bovis* but may adversely affect the degree of immunity to *B. bigemina.* If chemotherapy is necessary and imnunity to *B. bigemina* is considered desirable, a second vaccination may be necessary.

The use of blood vaccines is also practised in other parts of the world including Australia, several Latin American countries and Israel. In the United States an inactive anaplasmosis vaccine is available consisting of lyophilized killed *Anaplasma* organisms obtained from lysed erythrocytes which is administered with oil adjuvant.

As can be expected isoimmune blood group antibodies are produced in cattle after the use of whole blood vaccines[3]. If these antibodies are present in a cow and gain access to the circulation of a newborn calf which has inherited the corresponding blood group determinants (factors) from the sire, this may result in neonatal isoerythrolysis[5]. This condition has been reported from Australia where the babesiosis vaccine was incriminated as the source of antigenic stimulation[3 4] and also from the United States where the source was the inactivated anaplasmosis vaccine[2 9 10].

Despite the extensive use of blood vaccines in South Africa, confirmed cases of neonatal isoerythrolysis have not been recorded. The purpose of this work was to study the development of isoimmune antibodies in cattle in this country after inoculation of the local babesiosis vaccine.

MATERIALS AND METHODS

A total of 100 animals were selected for this study from a herd of 250 Afrikander cross cattle used in a babesiosis vaccination trial. Of these, 77 were heifers about 18 months old and 23 were calves under 6 months.

On Day 0 all the animals were bled for serum and subsequently inoculated with 5 mℓ babesiosis vaccine. Serum samples were again collected on Day 14. On Day 21 a second inoculation of 5 mℓ babesiosis vaccine was given and 2 weeks later, on Day 35, blood was again collected for serum tests.

The tests for isoantibodies in these sera were performed using the standard haemolytic blood typing test[7]. All the sera were tested undiluted against a panel of red cells from 30 animals of which the blood types were known. Of the positive reacting sera successive twofold dilutions (1/1, 1/2, 1/4 to 1/512 – were made with saline using a Cornwall Pipetting Outfit and tested with erythrocytes of the same 30 animals. The haemolytic reactions were read after 30 min, 2h and 4h and were recorded as 4 (complete haemolysis) 3,2,1, traces or 0 (no haemolysis). The corresponding scores to the six kinds of readings were given as 5,4,3,2,1 and 0 respectively and the sum of the scores (total score) given to the reaction in each tube of the titration was recorded as the strength or concentration of the antibodies. For example, a serum of which the strongest reactions were recorded at the final readings as 4 (score

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of 5) up to a dilution of 1/64, 2 (score of 3) at a dilution of 1/128 and trace at a dilution of 1/256 would have a total score of 39. (1/1 to 1/64 = 7 × 5 = 35; 35 + 3 + 1 = 39).

RESULTS AND DISCUSSION

No isoantibodies were detected in serum of these animals at the time of the first vaccination with the exception of a few trace reactions indicating the presence of weak naturally occurring anti-J-bodies.

The results obtained 14 days after the first and 14 days after the second vaccination are summarized in Table 1.

Table 1: FORMATION OF ISOANTIBODIES IN 100 ANIMALS AFTER TWO BABESIOSIS VACCINE INOCULATIONS

DIFFERENT ANTIBODIES CORRESPONDING TO ANTIGENS IN:	NUMBER OF ANIMALS SHOWING TITRES	
	After First Inoculation	After Second Inoculation
Blood group system A	2	5
Blood group system B	12	21
Blood group system C	1	8
Blood group system FV	6	11
Blood group system S	2	6
More than one system	2	31
Total (%) animals with antib.	25	82
Titre Score Range	0–12	10–47
Mean Score	3,8	26,1
Standard Deviation	1,9	10,7

Moderate antibody levels were present after the first vaccination but the mean titre score of 26,1 in 82% of the animals after the second inoculation must be regarded as very high. This is particularly so in view of a previous study[7] where it was found that only 66% of the antigenically stimulated animals produced antibodies at levels suitable for blood typing purposes.

No observations were made in this study on the persistence of these antibodies in the blood after vaccination. Blood group antibodies have, however, been observed in detectable levels for 18 months and may even persist for as long as 59 months[6]. Dimmock found that antibody levels reached a peak 3 weeks after inoculation of the Australian babesiosis vaccine and then decreased to a lower level during the ensuing 2 to 4 months[3].

It is known that cattle erythrocytes are characterized by a great variety of antigenic determinants called blood factors or phenogroups. A phenogroup is a complex of two or more blood factors which are inherited as a genetic unit and therefore appear to be controlled by one gene or gene complex. All blood factors or phenogroups that appear to be controlled by genes on the same chromosome belong to one blood group system. In cattle at least 11 genetic blood group systems are known[8], some of which are characterized by a single antigenic determinant (e.g. the L system) and some, like the B and C systems are characterized by considerable numbers of antigenic determinants. With present techniques only antigenic determinants that are on or near the surface of the cell can be detected. However, if enzymes are used to partially digest the cell walls it is likely that many more factors situated deeper in the cell wall will be found.

From Table 1 it is obvious that different antibody types were formed after inoculation of the babesiosis vaccine; the antibodies corresponding to antigenic determinants in the A-, B-, C-, FV- and S- systems were the most frequent ones, however in addition combinations of antibodies corresponding to factors in more than one system were produced. The highest titres were obtained by those antibodies corresponding to the A-, C-, F-, and S- antigenic determinants in the corresponding blood group systems and especially by Y-antibodies which correspond to one of the strongest antigenic determinants in the B-system, blood factor Y. These antigens are known to have a very high frequency in the Hereford breed[8] which forms the basis of the herd used for the production of the Onderstepoort anaplasmosis and babesiosis vaccines. The Onderstepoort babesiosis vaccine consists of pooled blood of up to four animals at a given time and explains the variation of antibody formation in the vaccinated animals. The different antibodies shown to be present in the vaccinated animals could therefor have easily been obtained as a result of the inoculation of the Onderstepoort vaccine used in this trial.

With the exception of J in the J-system, any of the determinants could theoretically be involved in the etiology of neonatal isoerythrolysis. The J antigen is not an instrinsic character of the red cell as it is a soluble glycoprotein produced in glandular tissues and acquired by the red cells on contact with that substance in blood plasma. Any J- antibody that might be obtained by J-positive calves out of J-negative dams would rapidly be bound by soluble J-substance before reaching the J-positive red cells[10].

Considering these factors it is obvious that, despite the absence to the present of proven cases of bovine neonatal isoerythrolysis in South Africa, the offspring of some of the vaccinated cows will acquire antibodies to their own red cells. The severity of this condition in calves, however, varies considerably and, according to Dimmock, is governed mainly by the amount of sensitizing antibodies to the calf's red cells ingested by it during the first few hours of life[4]. This amount is dependent primarily on the antibody levels in the dam at the time of birth which is in turn influenced by factors such as the time interval since vaccination, the degree of bloodgroup incompatibility between the dam and the vaccine received as well as the inherent ability of the dam to form and maintain antibodies[3]. In addition, there is evidence that breed differences may exist[4].

Some of these factors can be manipulated, e.g. the number of vaccinations given and the time interval between vaccination and calving. In this country general recommendations for the use of the anaplasmosis and babesiosis vaccines has been for a long time to vaccinate animals when young and to avoid vaccinating pregnant cows. The latter precaution was advised in order to avoid possible abortions in the event of clinical vaccine reactions. In general these recommendations have the effect of maximizing the interval between the dates of vaccination and calving and may well be a contributing factor to the apparent absence of clinical neonatal isoerythrolysis in this country.

As revaccination with either of the vaccines is seldom necessary, the marked effect of repeated vaccinations on the levels of blood group antibodies is therefore also eliminated. In addition, simultaneous inoculation of both the anaplasmosis and the babesiosis vaccines is advocated in those parts of the country where both diseases are prevalent. From the point of view of blood group antibody production this has the same effect as one vaccination. As approximately 75% of the lesser used babesiosis vaccine is administered concomitantly with anaplasmosis vaccine this may be an additional factor limiting the chance of neonatal isoerythrolysis occurring. (De Vos, unpublished observations).

These results show that the use of anaplasmosis or babesiosis vaccines will lead to the formation of blood group antibodies. Veterinarians should therefore be aware of the possibility of neonatal isoerythrolysis occurring albeit in a small percentage of calves. It must be stressed, however that the advantageous effects of these vaccines to the cattle industry in this country are such that they minimize the importance of possible neonatal isoerythrolysis as a factor worthy of consideration when the use of any one or both of the vaccines is contemplated. Where practical the standard recommendations discussed above should therefore be adhered to.

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THE INCIDENCE AND NATURE OF PROHIBITED ANIMAL TISSUES IN BOEREWORS ("COUNTRY-STYLE" SAUSAGE)

W.A. DE KLERK

ABSTRACT: De Klerk W.A. **The incidence and nature of prohibited animal tissues in boereword (Country style sausage).** *Journal of the South African Veterinary Association* (1977) **48** No. 2, 141 – 143 (En) Nat. Food. Res. Institute, CSIR, Box 395, Pretoria, 0001, Rep. of South Africa.

Sixty boerewors ("Country-style" Sausage) samples bought from butchers in Pretoria were examined microscopically. It was found that 97% of these samples contained animal tissues that are not permitted in boerewors under the regulations of the Foodstuffs, Cosmetics and Disinfectants Act (Act No. 54 of 1972). These regulations confine permitted tissues to fat and the musculature of cattle, sheep and pigs. Details of the tissues revealed in the survey are provided, and the implications of their presence are discussed.

INTRODUCTION

Boerewors is a spiced meat "Country-style" sausage made from the musculature and fat of the bovine, sheep or pig, or a mixture of two or more thereof. Traditionally boerewors was a home-made product but it is now mainly produced by retail butchers and wholesale meat processors. The latter have their outlets through butcheries, supermarkets and cafés. Boerewors is a product with widespread consumer acceptance in South Africa but statistics on the quantities consumed are unfortunately not available.

The requirements to be complied with in the manufacture of boerewors are specified in the regulations of the Foodstuffs, Cosmetics and Disinfectant Act (Act No. 54 of 1972). These state that:

"Boerewors" shall be made from clean, sound and wholesome musculature and fat of the bovine, sheep or pig, or mixture of two or more thereof. It shall contain not less than 90 per cent total meat and not less than 2 per cent protein nitrogen.

It may contain cereal substances, spices, harmless flavouring substances and permitted preservatives. It may contain saltpetre and sodium or potassium nitrite; provided that the finished article shall not contain more than 200 p p m of nitrite calculated as sodium nitrite.

(The meaning of this regulations is that "Boerewors" shall contain not less than 60 per cent of lean meat and not less than 90 per cent of total meat, i.e. lean meat and fat).

Boerewors, like most processed meat products, lends itself to adulteration. Finely minced constituents are not readily recognised by the naked eye while the use of spices may interfere with organoleptic detection of those tissues which are not permitted by the regulations. Finely minced animal tissues can, however, be identified microscopically. It was decided to conduct a histological survey to determine whether commercial boerewors complied with the legal definition and if not, whether the presence of any non-permitted tissues used in the manufacture of the product had any public health implications.

MATERIALS AND METHODS

Boerewors was purchased from each of 60 butchers in Pretoria and prepared for microscopical examination. Transverse sections were made into the sample to yield discs of boerewors about 1 cm thick. Four discs were removed from each sample at intervals of approximately 10 cm. The discs were immediately placed in 10% buffered formalin.

Paraffin-embedded blocks were prepared, cut into sections 5 to 7 μ in thickness, and stained with haematoxylin and eosin. One section was cut from each block so that 4 sections of tissues, lying about 10 cm apart in the sample of boerewors, were available for microscopic examination.

RESULTS AND DISCUSSION

The following animal tissues, none of which are permitted as ingredients of boerewors, were found in the specimens and are listed in Table 1.

Table 1: FREQUENCY AND DETAILS OF PROHIBITED TISSUES OF ANIMAL ORIGIN IN 60 SAMPLES OF BOEREWORS

Tissue	No of specimens positive
Spleen	39
Salivary gland	33
Liver	23
Heart muscle	20
Cartilage	20
Bone	16
Kidney	14
Udder (non-lactating)	6
Sweat gland (skin)	3
Tonsil	1
Tongue	1
Ductus deferens	1
Thymus	1
Pancreas	1

The results clearly indicate that boerewors, as made by the majority of the butchers included in the survey, is often an adulterated product which does not comply with the regulations and does not live up to the expectations of those consumers who still regard boerewors as the traditional product which should only contain beef, mutton or pork musculature mixed with fat and spices.

Organs and tissues such as spleen, salivary gland, udder, tonsil, *ductus deferens,* thymus and pancreas are aesthetically unacceptable to many if not most consum-

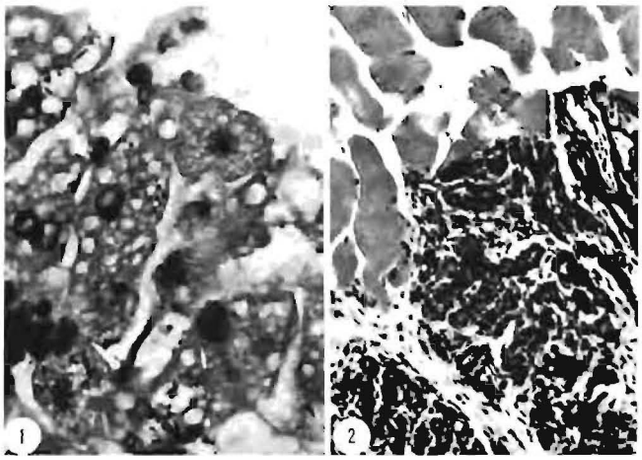

Fig. 1 Photomicrograph showing fatty changes in the liver tissue in boerewors.

Fig. 2 Histological section of boerewors with skeletal musculature (top) and splenic tissue (bottom).

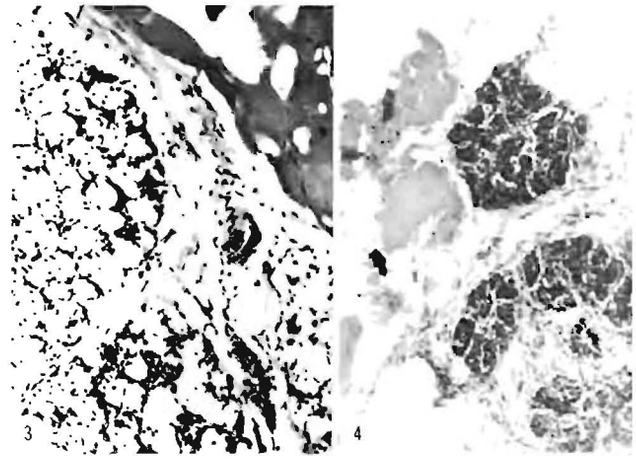

Fig. 3 Photomicrograph of boerewors specimen showing mainly salivary gland and some skeletal muscle in the top right hand corner.

Fig. 4 Histological section of boerewors containing non-lactating udder tissue (right) and skeletal muscle and fat (left).

ers. As a result of the malpractice of using such organs and tissues in the manufacture of boerewors, consumers are unwittingly eating tissues which very few would voluntarily consume.

The presence in boerewors of most of the organs listed in Table 1 is also undesirable for reasons other than aesthetic. Bacteria, for instance, establish themselves relatively easily in the parenchymatous organs before and after death. These organs therefore usually carry a greater bacterial load than muscular tissue. In contrast, living skeletal muscle is inhospitable to most bacteria[1] while post slaughter anaerobic glycolysis produces lactic acid in skeletal muscle[2]. This presence of lactic acid restricts bacterial proliferation. The muscle of beef and lamb usually attain an ultimate pH of 6,1 or below. This is unfavourable for the growth of most bacteria which grow optimally at about pH 7,0[2].

In addition the organs which are removed during dressing of the carcase in the abattoir rarely receive the same meticulous handling as the carcase itself. This will further increase the possibility of their exogenous bacterial contamination.

The presence of skin and udder in boerewors is especially undesirable as the skin is always heavily contaminated with bacteria and the bovine udder is prone to bacterial mastitis. One of the dangers of a product of this nature which is intended for human consumption and which is contaminated before (or during) manufacture is, of course, that bacteria frequently have the opportunity to proliferate when the product is displayed in butchers' shops at room temperature. A fact which may well compound this is that many members of the public are under the impression that the keeping qualities of boerewors are superior to those of many other meat products.

Yet another objection to the inclusion of organs such as liver, kidney and spleen is that they undergo *post mortem* autolysis more rapidly than does skeletal muscle. This may well lead to loss of aesthetic qualities such as flavour, texture, colour and general appearance.

Some of the organs found in boerewors samples in this survey e.g. liver and kidney, may be purchased as such over the counter. Their presence in boerewors might well mean that the butcher is using the boerewors as a means to rid himself of an inferior product which has low consumer appeal due to *ante mortem* degenerative processes like fatty changes or *post mortem* changes such as autolysis and imbition. This is illustrated by the fact that in 30% of the boerewors specimens that contained liver, the hepatic tissue showed fatty changes (see Fig. 1). Such fatty livers differ macroscopically from normal livers. They are paler, enlarged and softer and are therefore less acceptable to the consumer.

It is of course possible that small quantities of some of the non-permitted tissues found in boerewors were accidentally included but most of these tissues have a distinct anatomical appearance, colour and texture and it is unlikely that they would have been mistaken for musculature and fat. Some of the organs are not completely removed during the evisceration and dressing process, and it is not unusual to find pieces of spleen and udder still attached to the carcase when it reaches the retail butcher. This could be one of the sources of these organs to the butcher, while the practice to include the cheek musculature from cattle and pigs in boerewors probably account for the presence of salivary glands in the product. The high incidence of spleen (65%), salivary gland (55%), liver (39%), heart muscle (33%) and kidney (23%) indicates, however, that these tissues are purposely included.

The legal definition of boerewors is very specific and limiting as to the ingredients which may be used. Histological examination enables the food scientist to identify very small particles of tissue and is an accurate qualitative method for determining whether or not boerewors or other uncooked meat products are falsified. (See Figures 2, 3 and 4).

The histological survey of samples of commercial boerewors has shown that organs and tissues which are not permitted by legal definition, are in fact commonly used in the manufacture of boerewors. It may be argued that heart, liver and kidneys are nutritious and normally consumed items of edible offal and that their inclusion would not be to the detriment of the consumer. The fact remains, however, that their inclusion

is illegal and results in an adulterated product. Inclusion of spleen, salivary gland, udder, tonsil, *ductuc deferens,* thymus and pancreas must be considered as undesirable as these organs are aesthetically unacceptable.

As boerewors is normally consumed in a well cooked state, the health hazards of inclusion of the organs mentioned is probably low. Nevertheless they are likely to result in a higher initial bacterial load in the boerewors leading eventually to poorer keeping quality. Furthermore the danger of food poisoning as a result of inclusion of e.g. a piece of mastitis (staphylococcus) infected udder cannot be ignored.

ACKNOWLEDGEMENTS

Professors W.H. Gerneke and L.W. van den Heever of the Veterinary Faculty, University of Pretoria are thanked for helping with the microscopic identification of the organs and valuable suggestions respectively. Mrs M.E. van der Vyver is thanked for preparing the section and Mr N. v.d. W. Liebenberg for developing the photographs.

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1. JUBB K.V.F. & KENNEDY P.C. 1970 *Pathology of Domestic Animals,* Vol. 2. p. 466. New York: Academic Press.
2. LAWRIE R.A. 1966 *Meat Science* p. 163. Oxford: Pergamon Press.

BOOK REVIEW BOEKRESENSIE

ANIMAL AND HUMAN HEALTH: THE CONTROL OF DISEASE

G.C. BRANDER and P.R. ELLIS

Baillière Tindall, London 1976
pp V + 136, Tabs 3, Figs 13, Price not stated

This is the first of four short, inexpensive, soft cover books to be published in the series ANIMAL and HUMAN HEALTH under the general editorship of GC Brander. They are intended for medical and veterinary scientists and those who need to be aware of the crucial relationship between human and animal health.

The book is provided with a foreword by Professor Sir Michael Swann, who has chaired government committees of inquiry into the use of antibiotics in animal husbandry and veterinary practice, and on the future of the veterinary profession. The recommendations of these committees regarding the necessity of taking a broader view of the problems of animal health and their relation to human health, the parallel monitoring of animal and human disease, and the developing of preventive medicine on the farm, have served as stimulus for the writing and publishing of this series.

This volume deals with animal and human health hazards as they exist today and with the measures available to overcome them. GC Brander heads Agricultural Research and Development for Beecham's Pharmaceuticals and is Special Professor of Animal Health at the University of Nottingham. His co-author is Senior Lecturer in Animal Health at the University of Reading, Senior Scientific Adviser on Veterinary Economics to the EEC and member of the WHO's Expert Committee on the Zoonoses. Four diseases – smallpox, influenza, rabies and brucellosis – are used as models to discuss control methods. The interrelationship of animal and human health is brought out in the following chapters on host, agent and environmental relationship. Final chapters cover evaluation of disease control and likely future strategies.

The text is clear and concise, well printed and designed to provided not only factual information but also to promote a clear understanding of the subject matter. One of the more interesting chapters concerns programming, planning and evaluation and illustrates the need for study of cost-benefit relationships and cost-effectiveness analyses of disease control or eradication schemes.

This book is of undoubted value to all who work in the public health service, in food production and manufacturing, in controlling imports and immigration, in the pet animal industry or with livestock in agriculture.

L W vd H

"A CENTURY OF THE HEALING SCIENCES IN SOUTH AFRICA"

In June 1977, The Royal Society of South Africa will celebrate one hundred years of a distinguished and successful existence. It is the oldest general scientific society in Southern Africa and its roots may be traced well beyond its founding date, to meetings of learned men at the Cape in the late 1820's. The Society's history mirrors a remarkable period of growth of science. With this 'growth', the Society's ways of linking the work of scientists at the highest levels have become today an important and urgent function.

One of the highlights of the centenary celebrations of The Royal Society is a symposium entitled "A Century of the Healing Sciences in South Africa". Members of the Medical, Dental, Veterinary and Pharmacy professions have played a major role in the history of scientific development in this subcontinent. An early example is provided by the famous Dr William Guybon Atherstone who not only administered an anaesthetic as early as June 1847, but also studied the mineral springs of the Cape Province, recognised the first diamond discovered in 1867 near Hopetown on the Orange River, made observations on the Khoi-khoi or Hottentots and was a member of the Literary, Scientific and Medical Society of the Eastern Districts!

It is fitting, therefore, that the Centenary of organised science in South Africa should feature prominently the healing sciences. This unique historical event, under the distinguished chairmanship of the Minister of National Education, the Hon. Dr. P.G.J. Koornhof, was initiated and planned by Professor P.V. Tobias and Dr. J. Harington of the Transvaal Branch of The Royal Society, Dr. Cyril Adler and his wife Esther of The Adler Museum of the History of Medicine and Norman Nossel, Managing Director of Mer-National Laboratories. The event is being held in association with The University of the Witwatersrand.

THE SPEAKERS

Veterinary Science: Professor B.C. Jansen, Chief Director of Veterinary Services, Chairman of the Veterinary Board and Vice-President of the World Veterinary Association will present "One Hundred Years of Veterinary Science in South Africa". Professor Jansen's presentation will cover the animal disease position in South Africa prior to the inception of scientific control measures, and the work of the early pioneers who tried to elucidate causes of the different diseases and develop control measures. The establishment of veterinary education in South Africa and its development to the present day as well as an indication of the developments in Veterinary Science planned for the future will be of interest to the veterinarian and his colleagues in the Medical, Dental and Pharmacy professions.

Medicine: Professor J.H. Louw, Head of the Department of Surgery, University of Cape Town, author of many publications, including a major historical work entitled "In the shadow of Table Mountain — ' History of the University of Cape Town Medical School", will turn back the pages of Medicine.

Dentistry: Mrs. Vilma Grobler, wife of the Honorary Professor of Orthodontics University of Pretoria, and authoress of a thesis on the history of Dentistry in South Africa, was a natural choice to represent the Dental profession.

Pharmacy: Mr. Julius Israelsohn, member of The Pharmacy Board, Fellow of The Pharmaceutical Society of Great Britain and past President of The Pharmaceutical Society of South Africa, who has himself been a leading light in Pharmacy history-in-the-making, represents Pharmacy.

The Venue

The Great Hall, University of the Witwatersrand, was especially chosen as the venue for this unique symposium so that many interested members of the healing sciences could avail themselves of the opportunity to join the Transvaal Branch of The Royal Society and hear about earlier days of the healing sciences. The meeting commences at 2.15 p.m. on Wednesday, 29th June 1977 and all interested veterinarians are most welcome.

QUESTIONS & ANSWERS

VRAE & ANTWOORDE

Q Could you please let me know the latest information regarding *the hereditary basis of cryptorchidism in dogs.* I have a client with an imported Afgan dog that is a monorchid and they are anxious to know whether the condition is definitely hereditary or not, as this will greatly influence their decision regarding the use of this dog for stud purposes.

A: Cryptorchidism, the failure of one or both testes to descend through the inguinal canal into the scrotum, is an undesirable defect, partly because cryptorchids are temperamental, difficult to handle, and sterile, but also because they are disqualified in the show-ring. Frequently the undescended testis becomes tumorous, and the dog is feminized. Unilateral cryptorchids, often incorrectly called monorchids, are fertile. The defect occurs in swine, horses, and other mammals, but is perhaps most commonly found in dogs.

The defect occurs in many breeds of dogs, and is said by one writer to be more common in those having short skulls. In Germany, Härtl found that 23 per cent of 168 male Boxers in 57 litters were cryptorchids, and that the parents were all related. He attributed the high incidence of the defect to inbreeding and resultant concentration of the causative gene or genes. Although some reports suggest that cryptorchidism is a simple recessive, autosomal (but sex-limited) trait, the evidence is hardly conclusive to prove that only a single gene (when homozygous) is responsible. *There is no doubt that the defect is hereditary.* One difficulty preventing better understanding of the genetic basis is that genotypes of females can be determined only by progeny tests. Another is that most dog breeders are unwilling to reveal information about hereditary defects in their kennels. Expectations in various matings involving cryptorchidism are given in the following Table:

Expectations (Proportions in Litters, per cent) from Matings Involving Cryptorchidism in Dogs, Assuming it to be Monogenic, Recessive, and Autosomal, and the Sex Ratio 1:1

Parents	Progeny				
	Homozygous, free of the gene, all normal		Carriers, all normal	Homozygous for the gene	
	♂♂	♀♀	♂♂ ♀♀	Cryptorchid ♂♂	Normal ♀♀
Both normal, free of the gene	50	50	— —	—	—
Both normal, either one a carrier	25	25	25 25	—	—
Both normal, both carriers	12,5	12,5	25 25	12,5	12,5
Cryptorchid ♂* x normal and free of gene	—	—	50 50	—	—
Cryptorchid ♂* x carrier ♀	—	—	25 25	25	25
Cryptorchid ♂* x ♀ normal but homozygous for gene	—	—	— —	50	50

*Unilateral.

Veterinarians are sometimes asked to overcome this defect by surgery. Such an operation is not dysgenic (i.e., bad for the race) if done to correct bilateral cryptorchidism, because the damage done by the higher temperature of the body cavity is irreparable to the dog which would be just as sterile after the operation as before. However, correction of a unilateral case is dysgenic, because the dog would appear to be normal, and would be fertile like any unilateral cryptorchid. A better practice is to advise on ways to eliminate the causative gene.

The British Veterinary Association recommends that dog breeders should not breed from any (unilateral) cryptorchids, any litter-mates of cryptorchids, or their parents, or any normal males sired by (unilateral) cryptorchids. This advice, which is based on the mode of inheritance shown in the Table applies equally well even if later studies should show the genetic basis for the defect to be somewhat more complex.

D.R. Osterhoff
PROF & HEAD: DEPT ZOOTECHNOLOGY
Fac. Veterinary Science,
University of Pretoria
(April 1977)

FURTHER EDUCATION MATERIAL FOR THE VETERINARY PROFESSION

Introduction

In 1974 a "pilot" scheme was devised in the U.K. to assess the demand from practising veterinary surgeons for tape-slide programmes. Six programmes were originally planned; five of these are in use and the sixth will be available early in 1977; Pedigree Petfoods Ltd. provided the funds for their production.

As a result of the success of these first programmes, the Medical Recording Service Foundation (MRSF), Chelmsford, has offered to fund the production of future veterinary programmes. Several new ones are in the "pipeline" along with two left over from the original "pilot" scheme.

Production of the programmes

The first 6 programmes

The authors were responsible for the content. The tapes, slides and booklets were produced under the guidance of staff at The Royal Veterinary College (RVC), University of London. Copyright of the audiotapes belongs to The RVC.

Future programmes

The authors will be responsible for the content. Programmes will be produced under the guidance of RVC staff but the materials (tapes, slides and booklets) will be produced by the MRSF. Copyright of the audiotapes will be assigned to the MRSF.

Equipment needed

No expensive equipment is needed. Either a cassette player that takes a C90 cassette *or* a domestic tape recorder is suitable as tapes are produced in both forms. Either a slide viewer or projector is suitable for the transparencies (35mm).

Ways of using the programmes

The programmes are designed for individual use at home with the minimum of equipment. However, some practitioners may wish to use a programme as a basis for a seminar within their own practice or to show to a larger group at a local BVA meeting.

One of the important advantages of individual use is that the listener can work through a programme at his/her own speed. Inevitably, when a programme is shown to a larger group, the speed chosen will not suit all the members of the group. Programmes lasting more than 35 minutes are generally unsuitable for groups.

Each listener needs a copy of the booklet accompanying every programme; the booklet is intended to be used by the listener for making his/her own notes alongside the drawings, data, etc.

How to borrow or purchase a programme

Write to: Medical Recording Service Foundation,
P.O. Box 99,
Writtle,
CHEMLSFORD, CM1 5HL, Essex, United Kingdom.

(a) Ask for the programme by *title* and by *catalogue number*
(b) Request either a *cassette* or a *5" reel.*
(c) State how many booklets are needed.
(d) *Send no money until an invoice is received.*

Loan charge (only Inside U.K.): £2 plus VAT for 28 da including postage time.

Purchase price (inside U.K.): £5 per tape, 20p per slide pl postage and VAT; (outside U.K.): £8 per tape, 25p per sli plus postage, banking chages, etc.

Booklets: One free copy with every programme; extra copi cost between 10p and 50p each.

Programmes Available September 1976

PHYSIOLOGICAL ASPECTS of RENAL DISEASE & PHYSIOLOGICAL ASPECTS of its TREATMENT. (1974) Cat. No. VET 1
Author: Dr. A.R. Michell, The Royal Veterinary College, University of London.
Black-and-white diagrams in a booklet. Total time: 80 mins. Audience: veterinary surgeons and post-graduate research workers.

INFECTIOUS DISEASES OF SALMON and TROUT. (1974) Cat. No. VET 2
Author: Dr. R.J. Roberts, Unit of Aquatic Pathobiology, University of Stirling.
21 colour slides; small booklet. Time: 35 mins. Audience: veterinary surgeons.

POISONING IN SMALL ANIMALS. (1974) Cat. No. VET 3
Author: Professor E.G.C. Clarke, 49 Westwood Road, Tilehurst, Reading, Berks.
Booklet. Time: 35 mins. Audience: veterinary surgeons.

SWINE VESICULAR DISEASE. (First edition 1974. Revised edition late 1976). Cat. No. VET 4
Author: Mr. E.W. Hendrie, Ministry of Agriculture, Fisheries & Food, Hook Rise South, Tolworth, Surbition, Surrey.
18 slides (14 in colour); booklet. Time: 35 mins. Audience: veterinary surgeons. *Copyright of the slides and the booklet has been assigned to the CROWN.*

ABNORMAL OESTROUS CYCLES IN THE MARE. (1975) Cat. No. VET 7
Author: Dr. W.E. Allen, The Royal Veterinary College, University of London.
Black-and-white illustrations in a booklet. Total time: 65 mins. Audience: veterinary surgeons.

DISEASES IN PET & AVIARY BUDGERIGARS. (available early 1977) Cat. No. VET 5
Author: Mr. L. Arnall, 152 Bath Road, Worcester, Worcestershire.
Colour slides; booklet. Audience: veterinary surgeons.

Future Programmes

THE EYE. (1977) Part 1: Clinial Examination of the Canine Eye; Part 2: Progressive Retinal Atrophy (PRA) Cat. No. VET 6-i

Authors: Dr. P.G.C. Bedford, The Royal Veterinary College, University of London – Part 1. Dr. K.C. Barnett, A.H.T. Small Animals' Centre, Kennet, Newmarket, Suffolk – Part 2.

Colour slides; booklet. Audience: veterinary surgeons.

HIP DYSPLASIA. (1977) Cat. No. VET 6-ii

Author: Mr. D.G. Clayton-Jones, The Royal Veterinary College, University of London.

Black-and-white and colour slides; booklet. Audience: veterinary surgeons.

RADIOGRAPHY – A PRACTICAL GUIDE. (1977) Cat. No. VET 8

Author: Mr. P. Webbon, The Royal Veterinary College, University of London.

Black-and-white and colour slides; booklet. Audience: veterinary surgeons and animal nurses.

TOXOCARIASIS IN DOGS. (1977) Cat. No. VET 9

Authors: Miss E.J. Pegg and Dr. W.P. Beresford-Jones, The Royal Veterinary College, University of London.

Booklet. Audience: veterinary surgeons.

VISCERAL LARVAL MIGRANS. (1977) Cat. No. VET 10

Author: Dr. R.M. Connan, School of Veterinary Medicine, University of Cambridge.

Booklet. Audience: veterinary surgeons.

PROSTAGLANDINS & THEIR ANALOGUES IN VETERINARY PRACTICE. (1977) Cat. No. VET 11

Authors: Dr. D.E. Noakes and Dr. W.E. Allen, The Royal Veterinary College, University of London.

Booklet. Audience: veterinary surgeons.

DIFFERENTIAL DIAGNOSIS OF FIBROUS NODULES IN CATTLE. Cat. No. VET AM 1-2

Author: A.D. Osborne, University of Bristol Veterinary School.

BOOK REVIEW BOEKRESENSIE

POULTRY DISEASES

Ed. R.F. GORDON

Baillière Tindall, London 1977 pp XII 352 Figs 62 (12 colour) Tabs 16 Publ. Price £9.00

In spite of the prominent role played by poultry in human nutrition the relative involvement of our profession in diagnosis and control of poultry diseases remains low throughout the world. Similarly only few books are available on the subject. Therefore the book under review fills a noticeable gap. As an all-British effort it is of particular interest. Quite rightly the emphasis is placed on economic importance. The diseases are grouped logically under bacterial, viral, parasitic and fungal diseases, nutritional and skeletal disorders, diseases of unknown aetiology and constitutional disturbances. There follow chapters on diseases of turkeys, of ducks, of birds other than domestic poultry, a guide to field investigations, specific pathogen free poultry, the chick embryo in research, artificial insemination and poultry meat hygiene, further appendices on acts and orders applicable to poultry, poultry health scheme, stress and wellfare and other useful data.

The book is generally well written and presented. In fact I would wholeheartedly recommend it to students and practitioners if it were not for some unfortunate errors and omissions. Thus in the chapter on bacterial diseases coryza is not mentioned at all. There is no mention either of the egg transmission of *S. gallinarum*. Newcastle disease virus certainly is not agglutinated by red blood cells but *vice versa* (p. 83). Nor has the Onderstepoort Komarov virus been re-attenuated but kept in its original state (p. 92). Also some of the treatments in the parasitology section need updating. There is a large number of spelling mistakes, particularly of scientific terms. A number of the illustrations are of poor quality, and some (e.g. Figs 7 and 35) do not depict what they are supposed to show. Fig. 9 is the reproduction of a negative photograph. Most unfortunate of all is the jumble of metric and nonmetric terms in the otherwise very interesting collection of "useful data" in the last appendix. From the whole one has the impression of a rather hasty production. As it stands, the book certainly can be of great value to the worker in the poultry field. After a careful revision, however, it has the potential to become the book of choice for students as well as for general practitioners.

FWH

Cow infertility problems?

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ADMARK 1592

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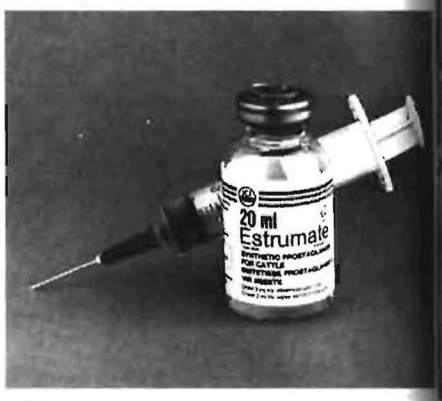

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