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EFFECT OF MONENSIN AND ITS METABOLITES IN BROILER LITTER ON SHEEP CONSUMING THE BROILER LITTER

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ABSTRACT

Two trials were conducted to determine the effect of monensin in broiler litter on sheep receiving the broiler litter in their diets. Broiler litter from chickens fed monensin as a coccidiostat, and from chickens receiving no coccidiostat, was included at a level of 30% in 2 sheep diets. In a further 2 treatments, monensin (15 mg kg^{-1}) was added to each of the 2 diets to give a 2x2 factorial experimental design. In the first trial, copper (20 mg kg^{-1} feed) was added to the diets. These lambs were fed individually at a slightly restricted level of intake. No differences between treatments were observed in feed intake, average daily gain or efficiency of feed utilisation or in the concentrations of zinc, iron and manganese in the liver, glutathione peroxidase in erythrocytes and creatine kinase concentrations in the plasma. Hepatic copper content and copper retention in the livers of the sheep receiving the added monensin were significantly higher ($P < 0,05$ and $< 0,01$ respectively) than in those not receiving added monensin. The aspartate transaminase and alkaline phosphatase concentrations in the plasma of these sheep were also higher ($P < 0,05$) than in those not consuming added monensin. In the second trial, the lambs were group-fed according to treatment and received the diets on an ad lib basis. The mean intakes of the groups receiving the diets with the added monensin, were lower than the intakes by the other groups. It was concluded that the monensin metabolites in broiler litter had no measurable effect on any of the parameters of monensin activity investigated in sheep, when such litter was included in sheep diets.

Key words: Monensin, sheep, broiler litter, hepatic copper

Van Ryssen J.B.J. **Effect of monensin and its metabolites in broiler litter on sheep consuming the broiler litter.** *Journal of the South African Veterinary Association* (1991) 62 No. 3, 94-99 (En.) Department of Animal Science, University of Natal, P.O. Box 375, 3200 Pietermaritzburg, Republic of South Africa.

INTRODUCTION

Monensin sodium (Elanco Product Company, Indianapolis, USA) is used widely as a coccidiostat in the diets of broilers, at a maximum recommended rate of inclusion of 120 mg kg^{-1} feed[6]. Monensin is also effective as an ionophor in the diets of sheep[11], at recommended levels of 15 to 22 mg kg^{-1}, and of cattle at a maximum of 33 mg kg^{-1} feed[6]. As an ionophor, the feeding of monensin can have various beneficial effects on the production of ruminants. One of its most important effects is an improvement in efficiency of feed utilisation in animals on high energy diets. This is quite often accompanied by a decrease in feed intake, without a concurrent decrease in bodymass gains[3]. Further beneficial effects are the control of coccidiosis in calves, control of feedlot disorders such as a reduction in the incidence of bloat[2] and acidosis especially during the period of adaptation to high concentrate diets[8].

Various metabolic changes due to the consumption of monensin have been observed[3]. Of relevance to the present study are the changes in the metabolism of minerals. Van Ryssen & Barrowman[18] found that monensin increased the retention of dietary copper in the liver of sheep. Anderson et al.[1] measured an improvement in the selenium status of sheep due to monensin. It has also been reported that the metabolism of sodium, potassium, magnesium, calcium, phosphorus and zinc in ruminants is altered by monensin[9 10 14].

At intakes higher than the recommended levels, monensin can have detrimental effects on animals. A reduced feed intake is one of the first symptoms of an excessive monensin intake[11 12]. At high intakes monensin can be very toxic. The LD_{50} of monensin for sheep is 11,9 mg kg^{-1} and for cattle 26,4 mg kg^{-1} livemass[12]. The LD_1 for a 400 kg bovine is estimated to be 2 210 mg monensin[12]. Symptoms of chronic toxicity are anorexia, skeletal muscle weakness, ataxia, decreased gain in mass and eventually death. Primary target tissues are the skeletal and cardiac muscles[15]. This is evident from elevated plasma concentrations of enzymes such as creatine kinase, which is usually associated with the breakdown of muscle tissues[13]. The supplementation of selenium and vitamin E to a selenium-deficient diet provided some protection against the harmful effect of high monensin intakes in pigs[19]. Deaths due to excessive monensin intake are usually due to inadvertent inclusion of excessive levels of monensin in ruminant diets, or the poor mixing and thus poor distribution of monensin in the feeds[11 12]. However, it was pointed out (M H Lowrey 1990, Private Veterinarian, P O Box 151, 3370, Umlaas Road, personal communication) that it is sometimes claimed that the mortalities which have occurred among ruminants consuming broiler litter, are caused by monensin or by its metabolites in litter which originated from the coccidiostat-treated broilers.

It was demonstrated with the use of radio-active markers that the monensin ingested by poultry, is excreted almost quantitatively via the faeces in the form of metabolites[6]. The monensin is metabolised in the body of the bird and reaches the digestive tract in the bile. The presence of these metabolites in faeces could not be detected with the use of the standard microbial test for monensin and showed a very low biological activity[6]. The question therefore arises whether the metabolites of monensin in the broiler litter could have the same detrimental effect on animals as does the unmetabolised compound.

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Two trials were conducted to establish whether excreted monensin or its metabolites in broiler litter, have the same measurable and metabolic effects on sheep as dietary monensin when sheep consume the litter. It is assumed that if litter is included in a sheep diet at a level of 30%, a relatively high level of these metabolites should still be present in the diet.

MATERIALS AND METHODS

Two groups of broilers were reared for 6 weeks in experimental rearing pens containing wood shavings as bedding material. One group received a diet containing monensin as a coccidiostat at a level of 100 mg kg^{-1} feed while the other group received no monensin. At the end of the rearing period, the litter from these pens was removed separately, sifted and sun-dried.

Two feeding trials with sheep were conducted in which these 2 types of litter were included in their experimental rations. At least 3 weeks before the onset of the trials, all the sheep were vaccinated against botulism and dosed with a wide-spectrum anthelmintic.

Trial 1: Liver biopsies were done on SA Mutton Merino wethers, (n=38) ca 9 months of age, with a mean body mass of 29 kg. Six wethers with liver copper concentrations deviating the most from the mean of the group, were slaughtered as a pre-experimental group to determine the relationship between liver mass and empty body mass. Using a 2x2 factorial design, the remaining 32 wethers were allocated to 4 treatment groups in a randomised block according to the copper concentration in their livers. The treatments were: i) a control group (C) receiving a diet containing 30% broiler litter from the broilers which did not receive the coccidiostat in their diet; ii) the control diet (as for Group C) but with added monensin (15 mg kg^{-1} feed) (C+); iii) a group on the test diet (T) including the litter (at 30%) from the chickens receiving monensin, and iv) the test diet fortified with monensin (15 mg kg^{-1} feed) (T+). The experimental diet consisted of 300 kg broiler litter, 380 kg hominy chop, 50 kg fishmeal, 180 kg veld hay, 80 kg molasses meal, 5 kg lime and 5 kg salt per 1000 kg. Copper (as copper sulphate) was added to the rations at 20 g per 1000 kg. For the first 2 weeks of the trial, monensin was included at half the intended level. This practice enabled the lambs to adapt to monensin feeding and allowed an opportunity to minimise mortalities if treatments should prove to be toxic. The sheep were fed individually. In order to keep the metabolic changes in the body as comparable as possible, it was planned to keep feed intake per sheep as constant as possible. Therefore allocations were adjusted weekly, according to the intakes of the groups receiving the added monensin.

Feed intakes and body mass changes were recorded weekly. The sheep were observed closely for ill-health and any other symptoms of toxicity. Blood samples were collected in heparin at various stages of the trial for mineral and enzyme assays. In order to minimise any effect of the liver biopsy on enzyme levels in the plasma, the first collections were only done at 36 d after the onset of the trial. On Day 77 the lambs were slaughtered, after exsanguination. Carcass and fresh liver masses were determined. Liver samples were taken, dried at 80°C and stored for further analysis.

Trial 2: In a group feeding trial, ewe lambs were allocated randomly to similar experimental treatment groups as in Trial 1. However, the added copper was omitted from the diets. The lambs (5 per group) received their diets on an ad lib feeding basis in order to measure voluntary feed intake. The trial lasted for 45 days. Feed intakes per group and individual body masses were recorded weekly.

Atomic absorption spectrophotometry was used to determine the concentrations of copper, zinc, iron and manganese in the liver and feed. Sodium and potassium concentrations in plasma were obtained using flame photometry. Glutathione peroxidase (EC 1.11.1.9) was assayed at 25°C using the coupled enzyme procedure as modified by Whanger et al.[20]. Boehringer Mannheim standard kits (Boehringer Mannheim GmbH Diagnostica, West Germany) were used to estimate the aspartate transaminase (EC 2.6.1.1), alkaline phosphatase (EC 3.1.3.1) and creatine kinase (EC 2.7.3.2) concentrations in plasma. The monensin content of the diets was estimated using an anti-microbial growth assay[6].

To calculate hepatic copper retention at the end of the trial, the following procedure was carried out:

An estimate of individual liver masses at the onset of the trial was made from the body mass of each lamb and the ratio of

Table 1: **An illustration of the interpretation of a single 2x2 factorial analysis (Copper concentration in Table 6 used as example)**

		Monensin added to broiler diet		
		No	Yes	Mean
		A		B2
Monensin added to sheep diet	No	778(C)	740(T)	759
	Yes	985(C+)	835(T+)	910
		B1		
Mean		882	788	835

Least significant differences (LSD):

	P=0,05	P=0,01
for Box A	220	297
for Boxes B1 and B2	156	210

i) Box A contains the original treatment means which each came from 8 sheep. The statistical significance of the difference between any pair of these can be calculated from the LSD's, e.g. the difference between C and C+ is 207, therefore less than 220 and not significant. These means with the respective LSD's are presented in a column in subsequent tables. Differences between 2 treatments can be compared.

ii) Both the boxes designated B, contain the means of the 2 basic treatments, viz. with or without monensin as a coccidiostat in B1, and with or without added monensin in B2. The differences within each box can be compared with the LSD's, e.g. in B2 the difference is 151 therefore less than 156 and not significant at P = 0,05.

Where the means within the basic treatments were large, being significant or approaching significance, the means are presented in subsequent tables.

liver mass to body mass as obtained from the pre-experimental slaughter group. The copper content of the livers was calculated from this estimated liver mass and the copper concentration in the biopsy samples. This was subtracted from the copper present in the liver at the end of the trial (copper concentration x liver mass) to calculate hepatic copper accumulation. The percentage dietary copper retained in the livers was obtained from total copper intake and hepatic copper accumulation.

The 2x2 factorial analyses were performed on the data with the aid of the computer programme Genstat (Lawes Agricultural Trust, 1980). To demonstrate the interpretation of results in a 2x2 factorial statistical design, a description is presented in Table 1 which can be used to evaluate the proceeding tables. In these tables the original treatment means, each from 8 sheep, are supplied and compared with the use of the LSD (least significant difference). Where combined means related to one of the 2 basic treatments were important, the means of the 16 sheep are presented with the relevant LSD's, e.g. in Tables 5 and 6.

RESULTS

Diet, feed intake and performance

The concentrations of monensin and minerals in the experimental diets are presented in Table 2.

Due to restricted feeding, no difference in feed intake among groups was observed in Trial 1. In Trial 2 the differences in

Table 2: **The concentration of monensin and minerals in experimental diets containing broiler litter (C), broiler litter with monensin added (C+), broiler litter from chickens which received monensin (T), and broiler litter from chickens which received monensin with monensin added (T+). Copper sulphate was added to all rations**

Treatment	Monensin	Copper	Zinc	Iron	Manganese
	mg kg^{-1}		mg kg^{-1} dry mass		
C	3	25,0	92	540	170
C+	26	25,6	101	612	181
T	7	25,5	100	555	175
T+	22	24,2	102	518	177

Table 3: **Mean feed intakes and performances of sheep on diets containing broiler litter (C), broiler litter with monensin added (C+), broiler litter from chickens which received monensin (T), and broiler litter from chickens which received monensin with monensin added (T+)**

	Trial 1					Trial 2		
		ADG*		EFU**		ADG*		EFU**
Treatment	Feed intake kg d^{-1}	Carcass g d^{-1}	Live g d^{-1}	Carcass	Live	Feed intake kg d^{-1}	Live g d^{-1}	Live
C	1,69	121	179	14,1	9,6	2,00	173	11,5
C+	1,68	122	192	14,0	8,9	1,68	160	10,5
T	1,70	122	197	13,9	8,7	1,93	229	8,5
T+	1,66	120	187	14,0	9,3	1,61	160	10,0
LSD*** P = 0,05	0,10	18	31	1,4	1,4	-	43	-

*ADG - average daily gain
**EFU - efficiency of feed utilisation (kg feed kg^{-1} gain)
***LSD - least significant difference

feed intake between the groups receiving the metabolites and those without the metabolites (Treatments T versus C and T+ versus C+) were negligible, while those receiving the added monensin (Treatments C+ and T+) consumed substantially less feed compared to the other 2 groups respectively (Table 3). It was not possible to compare the intakes in Trial 2 statistically, because group feeding was applied.

The mean final body masses, average daily body and carcass gains and efficiency of feed utilisation (kg feed consumed divided by kg gain) calculated for body and carcass mass gains (initial carcass mass estimated from live mass at onset of trial and dressing percentage of pre-experimental slaughter group), did not differ significantly between treatments in Trial 1 (Table 3). In Trial 2, sheep on the T treatment showed a higher ($P < 0,05$) gain in body mass than sheep on the other treatments (Table 3).

Blood analyses

The concentration of sodium (149, 148, 149 and 151 mmol l^{-1}) and potassium (4,7; 4,8; 5,0 and 4,9 mmol l^{-1}) in plasma for treatments C, C+, T and T+ respectively, did not differ significantly between treatments. The differences between treatments per collection in erythrocyte glutathione peroxidase activity and plasma creatine kinase, were not significant (Table 4). However, both glutathione peroxidase and creatine kinase levels increased between the collection on Day 36 and that on Day 77. When extra monensin was added to the diets (Treatments C+ and T+), the mean concentration of aspartate transaminase and alkaline phosphatase (Table 5) in plasma were significantly ($P < 0,05$) higher than in the 2 treatments without the added monensin.

Liver

Mean iron, zinc and manganese concentrations in the livers did not differ significantly due to treatments. These concentrations for treatments C, C+, T and T+ were: 228, 224, 241 and 224 mg iron kg^{-1} dry matter (DM); 169, 163, 182 and 159 mg zinc kg^{-1} DM and 13,9; 14,2; 13,3 and 13,9 mg manganese kg^{-1} DM respectively.

The livers of the groups receiving added monensin in the diets (C+ and T+) had a higher ($P < 0,05$) total hepatic copper content and copper accumulation (Table 6) than the groups without the added monensin. The percentage dietary copper retained in the livers of the groups receiving added monensin was significantly higher ($P < 0,01$) than for the other groups (Table 6).

The mean liver mass and liver mass expressed as a percentages of carcass mass, did not differ significantly between treatments. Fresh liver mass as a percentage of carcass mass in treatments C, C+, T and T+ was 3,2; 3,3; 3,3 and 3,6% respectively.

DISCUSSION

Accepting that metabolites from monensin cannot be detected in poultry manure[6], the concentration of unmetabolised monensin in the litter of the birds receiving the coccidiostat, can be calculated from the monensin in diet T (based on the 30% litter in the experimental diets). This amounted to 23 mg monensin per kg litter. Some of this might have originated from feed spilled onto the bedding material. Donoho[6] reported that chickens excreted less than 10% of the orally-administered monensin as "parent" monensin via the faeces. The presence of 3 mg monensin per kg feed in the Control diet, is probably a false positive reading which showed up in the anti-microbial growth assay for monensin. Such a false positive reading, plus sampling errors, might have reduced the reliability of the monensin concentrations presented.

Evidence of an effect of monensin on the sheep was observed in Trial 2, in the treatments where monensin was added (Treatment C+ and T+) to the diets. Feed intake was not restricted in this trial and voluntary feed intake in these 2 treatments was lower than in the others. This is in accordance with most other studies[3]. Monensin metabolised by the broilers, did not depress the feed intake of the sheep. In both trials, efficiency of feed utilisation did not differ between treatments. An improved efficiency of feed utilisation is considered to be one of the main beneficial effects of monensin in ruminants[3], although this is quite often not observed in sheep[18]. The significantly higher gain in mass of the sheep in Treatment T of Trial 2 is difficult to explain. In Trial 1, the average daily gain in Treatment T was also higher, though not statistically different, from that in Treatment C.

The increased copper content and copper retention in the livers of the sheep receiving the added monensin, is further evidence of an effect of monensin on mineral metabolism. This agrees with the observation by Van Ryssen & Barrowman[18] that monensin enhanced hepatic copper retention in sheep. From this study it is clear that the metabolites of monensin in the diets had no effect on copper metabolism while the added monensin increased copper accumulation in the liver. Significantly higher plasma concentrations of aspartate transaminase and

Table 4: **Mean serum concentrations of glutathione peroxidase and creatine kinase in sheep on diets containing broiler litter (C), broiler litter with monensin added (C+), broiler litter from chickens which received monensin (T), and broiler litter from chickens which received monensin with monensin added (T+)**

	Glutathione peroxidase nmol NADPH mg^{-1} Hb			Creatine kinase U l^{-1} plasma	
	day			day	
	36	58	77	36	77
C	182	267	324	118	168
C+	190	279	299	124	197
T	196	266	307	88	168
T+	230	249	318	127	187
LSD* P=0,05	41	34	54	59	64
P=0,01	55	45	73	80	86

*LSD = least significant difference

Table 5: **Plasma concentrations of aspartate transaminase and alkaline phosphatase in sheep on diets containing broiler litter (C), broiler litter with monensin added (C+), broiler litter from chickens which received monensin (T), and broiler litter from chickens which received monensin with monensin added (T+)**

	Aspartate transaminase*** U l^{-1} Day 36		Aspartate transaminase*** U l^{-1} Day 77		Alkaline phosphatase*** U l^{-1} Day 36		Alkaline phosphatase*** U l^{-1} Day 77	
	Indiv.	Mean*	Indiv.	Mean*	Indiv.	Mean*	Indiv.	Mean*
C	138		171		587		554	
		137		164		717		614
T	136		157		847		674	
C+	199		224		921		852	
		291		318		914		843
T+	383		412		903		835	
LSD**								
P=0,05	178	126	198	140	245	173	210	148
P=0,01	240	170	267	189	330	233	282	200

*means to compare with or without added monensin
**LSD - least significant difference
***means to compare with or without monensin as a coccidiostat not significantly different

alkaline phosphatase accompanied the higher hepatic copper concentrations of sheep receiving the added monensin. This may indicate that these lambs were closer to the haemolytic crisis stage of copper toxicity than those in the other two treatments. Todd & Thompson[16] reported that the concentration of enzymes which may indicate liver damage, starts to rise at about 8 weeks before the onset of a haemolytic crisis. However, in the present investigation, elevated concentrations of these enzymes were observed in all the lambs from the first blood collection on Day 36, without any further increases occurring towards the end of the trial. This does not support the suggestion that some lambs were approaching a haemolytic crisis. Van Ryssen & Barrowman[18] suggested that sheep may be close to the haemolytic crisis when their liver masses expressed as a percentage of body mass were smaller than in sheep not approaching the crisis. In the present investigation, liver mass relative to carcass mass did not differ between treatments, implying that the crisis was probably not imminent.

Various studies show that monensin alters the metabolism of sodium and potassium in the body[9 14]. Starnes et al.[14] and Kirk et al.[9] did not observe any changes in serum potassium and sodium

Table 6: **Hepatic copper concentrations in sheep which were fed broiler litter (C), broiler litter with monensin added (C+), broiler litter from chickens which received monensin (T), and broiler litter from chickens which received monensin with monensin added (T+)**

	Hepatic copper*** Concentration mg kg^{-1}DM		Hepatic copper*** Total content mg		Hepatic copper*** Accumulation mg		Hepatic copper*** Retention %	
	Indiv.	Mean*	Indiv.	Mean*	Indiv.	Mean*	Indiv.	Mean*
C	778		158		153		4,7	
		757		159		153		4,7
T	740		159		154		4,6	
C+	985		207		202		6,1	
		910		196		191		5,9
T+	835		185		180		5,8	
LSD**								
P=0,05	220	156	44	32	45	32	1,24	0,87
P=0,01	297	210	61	43	61	43	1,66	1,18

*means to compare with or without added monensin
**LSD - least significant difference
***means to compare with or without monensin as a coccidiostat not significantly different

concentrations, in agreement with the present study. However, Starnes et al.[14] quoted other studies in which monensin reduced serum potassium concentrations. Kirk et al.[10] found that monensin increased the retention of zinc in the body of sheep, although levels in the liver did not change. In the present investigation, liver zinc levels did not differ between treatments. In the present investigation, monensin did not have any effect on iron levels in the liver. This is in agreement with the observations of Elsasser[7], but not with those of Van Ryssen & Barrowman[18] who measured a reduction in hepatic iron concentration due to monensin. The observation by Van Ryssen & Barrowman[18] that liver manganese concentrations are elevated in monensin-fed sheep was not substantiated in the present study.

A glutathione peroxidase activity in excess of 40 nmol NADPH mg^{-1} haemoglobin indicates that the animal is consuming sufficient selenium to meet its requirements[1]. Therefore, the high levels of this enzyme in the present study, preclude the possibility that a selenium deficiency could accentuate the effects of monensin toxicosis[19]. Anderson et al.[1] reported that monensin improved the selenium status of sheep. On Day 36, in the present trial, the groups receiving the added monensin (C+ and T+) had higher glutathione peroxidase levels than treatments C and T respectively, although the differences were not statistically significant. On Days 58 and 77 differences between treatments were minimal, which is in agreement with Costa's[4] observation that monensin has no effect on the selenium status of animals. Whanger et al.[20] observed a continuous increase with time in the blood glutathione peroxidase concentrations of sheep receiving high levels of selenium in the diet. From the increases in enzyme concentrations between Days 36 and 77, it can be concluded that the sheep diets contained more than sufficient selenium to meet their requirements.

The fact that creatine kinase concentrations in plasma did not differ significantly between treatments may indicate that monensin, whether derived from litter or from addition to the diet at recommended levels, had no damaging or toxic effects on muscle tissue[13 15]. The relatively high creatine kinase levels measured in all treatments at the end of the trial is difficult to explain.

From this investigation, it may be concluded that the monensin fed to poultry as a coccidiostat had been metabolised to such an extent that it became metabolically inactive. It showed no effect in any of the parameters of monensin activity measured in the sheep. These results suggest that the feeding of monensin as a coccidiostat to poultry does not pose any risk to sheep consuming the litter as part of their diet. This supports the evidence of Donoho[6] that the metabolism of monensin in the body of poultry results in the destruction of most of its biological activity. Furthermore, the results suggest that statements in the popular press[5 17] that coccidiostats which are excreted in the litter of broilers can cause deaths in ruminants, especially if the same product is included in the ruminant rations as an ionophore, are too generalised. They may possibly apply to certain coccidiostats, but should exclude monensin.

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HANDBOOK ON ANIMAL DISEASES IN THE TROPICS

M M H SEWELL and D W BROCKLESBY. 4th Edition, Baillière Tindall, London. 1990. pp ix and 385. Tables 18. Price £14.95
(ISBN 0-7020-1502-4)

This is the 4th edition of the well-known text first published by the British Veterinary Association in 1962. It is divided into sections on diseases caused by arthropods, bacteria, helminths, protozoa, rickettsia, viruses and a short section on other diseases.

The book is well presented and written in a very concise manner. References are not quoted, but a few key references for further reading have been given at the end of each section.

In the preface it is stated that the aim of the book is:

"To provide a concise summary of the more important infections and conditions causing ill-health in domestic animals in tropical and sub-tropical countries, emphasizing the special features applicable in these countries in the case of diseases of a wider distribution," and that a determined effort has been made to prevent the handbook growing into a tome. This, unfortunately, has been taken too far in some cases and has resulted in a lack of certain important, practical information which would be needed for a veterinarian to cope with certain specific conditions, under field conditions.

Little information is given on the pathogenesis of diseases. In the case of canine babesiosis, it has meant that as pathogenesis is not discussed, there is no information of symptomatic treatment which would be important in the treatment of any severe case of canine babesiosis.

Dosages of drugs are not given, which in many cases is acceptable. However, in specific instances, this can cause problems. For example in the discussion on canine ehrlichiosis, it is stated that tetracycline should be used. There is no indication how long the treatment should be continued. To treat a patient parenterally for 10 days is often impractical. The practical alternative is to use doxcycline at 10 mg per kg per os.

To merely state that: "Post-immunization reactions following heartwater vaccination can be abated with tetracyclines" is probably not sufficient information to enable a veterinarian to proceed with the vaccination of valuable adult animals against this disease.

Due to the lack of detail, this book is mainly recommended for anyone wishing to obtain an introduction to animal diseases in the tropics or as a rapid source of reference for practitioners.

C G Stewart

OVERBERG RESEARCH PROJECTS. X. THE EPIDEMIOLOGY OF HELMINTHS IN EWES AND LAMBS IN THE SOUTHERN CAPE PROVINCE DURING AUTUMN

J P LOUW* and R K REINECKE*

ABSTRACT

Nematode parasite burdens of ewes grazing on grass/lucerne pasture, increased 58-fold after the first autumn rains in the southern Cape Province. Lambs were infected before the age of 8 weeks and harboured large burdens of nematode parasites before the age of 14 weeks. *Oestrus ovis* infections were present in 96% of the ewes, while 92% of the lambs above the age of 3 weeks were infected. Anthelmintic treatments in autumn, winter and spring are recommended for controlling parasites of sheep in this region.

Key words: Epidemiology, sheep, helminths, autumn.

Louw J.P.; Reinecke R.K. **Overberg research projects. X. The epidemiology of helminths in ewes and lambs in the southern Cape Province during autumn.** *Journal of the South African Veterinary Association* (1991) 62 No. 3, 101-103 (En.) Department of Parasitology, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, 0110 Onderstepoort, Republic of South Africa.

INTRODUCTION

Epidemiological studies on the nematode parasites of sheep in the Rûens region of the southern Cape were conducted in an area where no significant rain fell before April[9 10]. The dryland legume pastures in that area are unproductive during summer and autumn, and sheep on the majority of farms are grazed on harvested wheat fields until winter. Conditions on the wheat stubble were unfavourable for the survival of infective nematode larvae in the hot, dry summer. However, the eastern part of the Rûens region which forms the transition from a winter rainfall to a non-seasonal rainfall area, is known to receive substantial quantities of rain in autumn, and the dryland legume pastures can be grazed by the sheep in autumn. The epidemiology of nematode parasites of sheep grazing on these pastures in autumn, was unknown and warranted investigation after it was determined that nematode parasites were rife on similar legume pastures grazed by sheep during winter[9 10]. For this purpose a trial was conducted in ewes and lambs on the farm Vaalplaas, east of Caledon.

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MATERIALS AND METHODS

The farm Vaalplaas where the experiment was conducted, lies approximately 7 km east of Caledon (34° 15'S, 19° 28'E) in the Rûens region of the southern Cape Province. A small paddock of approximately one hectare close to the homestead, was used for the experiment. During the summer of 1987 the paddock was used as a loafing yard for dairy cows. Prior to that, it was grazed by sheep. During the autumn of 1988, the paddock was ploughed and sown to grass and lucerne and kept free from animals until the experimental animals were placed on it on 21 February 1989.

The rainfall and temperature data presented in Fig. 1 were recorded by the Agrometeorology Section of the Department of Agricultural Development at a weather recording station on an adjacent farm, Dunghye Park.

On 21 February 1989, a group of pregnant ewes (n = 18) was transferred to the experimental paddock. As the ewes lambed, each ewe and her lamb were eartagged and later slaughtered as a pair. Six ewes which had given birth to stillborn lambs, were slaughtered on 21 February 1989 and served as indicators of the helminth infection present in the ewes at the time of their transfer to the experimental paddock.

Trial schedule:

20 September - 5 October 1988 - Exposed ewes to teaser rams.
6 October - 15 November 1988 - Ewes mated.
21 February 1989 - Slaughtered 6 ewes as indicators and transferred 18 pregnant ewes to the experimental paddock.
15 March - 15 April 1989 - Lambing period.
4 April 1989 - Slaughtered 6 ewes and lambs.
16 May 1989 - Slaughtered 6 ewes and lambs.
27 June 1989 - Slaughtered 6 ewes and lambs.

The ewes were treated with ivermectin in December 1988, whereafter neither ewes nor lambs received any further anthelmintic treatments. All animals selected for necropsy were starved for 24 h, slaughtered and the heads and gastro-intestinal organs removed for processing. The abomasum, small intestine and caecum and colon were each ligated with string, separated, placed in labelled plastic bags, transported to the laboratory and processed as follows:

Abomasum: The ingesta and washings of the abomasal wall were collected in a bucket, fixed with formalin, washed on a sieve with apertures of 38 micrometer and the washed ingesta preserved in a labelled glass jar. The wall of the abomasum was then cut into small pieces and placed in a labelled glass jar containing 3% HCl, incubated at 40°C for 20 to 24 h, washed on a 38 micrometer sieve, the washings collected in a labelled jar and preserved in 10% formalin.

Small intestine: the ingesta and washings of the small intestinal wall were collected in a bucket, fixed with formalin, washed on a 38 micrometer sieve and the washings collected in a labelled jar and preserved.

Table 1: Mean worm burdens of 6 ewes slaughtered at diffetent times of the year

Slaughter date	Teladorsagia		circum-cincta	trifur-cata	Total	Nematodirus		spathiger	abnormalis	Total	Trichostrongylus colubriformis		falculatus	axei	Total	All Other	Nematode immature	Total adult	Total	Oestrus ovis	
	L3	L4				L3	L4				L3	L4								1st	2nd	3rd
21/2/89	205	259	369	0	633	19	84	0	301	224	38	22	0	135	0	0	145	1	470	531	1001	7	8	5
4/4/89	8897	15253	2945	0	27095	237	543	662	398	1182	58	1281	0	2633	6693	2395	11025	30	26190	13142	39332	14	4	12
16/5/89	1583	5585	8975	3529	19084	329	677	0	408	778	157	1609	6959	3470	3906	155	15253	0	9068	26050	35118	17	1	2
27/6/89	9803	42166	1909	416	52452	613	585	0	0	687	408	338	16141	1265	10020	866	28698	0	51428	30413	81841	19	2	7

Table 2: Mean worm burdens of 6 lambs slaughtered at different times of the year

Slaughter Date	Teladorsagia		circum-cincta	trifur-cata	Total	Nematodirus		spathiger	abnormalis	Total	Trichostrongylus colubriformis		falculatus	axei	Total	All Other	Nematode immature	Total adult	Total	Oestrus ovis	
	L3	L4				L3	L4				L3	L4								1st	2nd	3rd
4/4/89	0	30	10	0	7	0	0	10	0	2	0	0	100	0	0	17	0	5	20	25	0	0
16/5/89	192	244	998	802	1533	72	57	310	257	470	0	110	289	408	0	0	481	0	387	2097	2484	12	0	0
27/6/89	6433	26807	21621	2892	56681	498	720	1726	717	2731	776	6494	10612	20012	41053	363	78929	14	39944	98409	138354	19	2	1

Caecum and colon: The ingesta and washings of the wall of these gastro-intestinal organs were collected in a bucket, washed on a 150 micrometer sieve and the washings collected in a labelled jar and preserved in 10% formalin.

When a preliminary rough estimate of the total number of worms present in the ingesta and digesta of the abomasum and the ingesta of the small intestine indicated a worm burden in excess of 2 000 worms in a given organ, all the worms in 4 aliquots of one percent were counted under a stereoscopic microscope, otherwise 4 aliquots of 5% were processed. Approximately 30 male worms and 30 larvae were removed from each sample of ingesta for identification under a compound microscope. The ingesta of the large intestine were examined macroscopically in toto and the worms counted and identified.

For the recovery of *Oestrus ovis* larvae from the slaughtered animals, the heads were split sagitally with a saw and the nasal cavities and sinuses opened with a pair of side-cutters. The larvae were then removed and counted with the aid of a hand lens.

RESULTS

The mean monthly temperatures and the total monthly rainfall recorded on the neighbouring farm during the trial period, are presented in Fig. 1. The rainy season commenced with a precipitation of 17 mm on 13 March 1989 and 46,5 mm on 30 March 1989. The mean number of each stage of the different parasites recovered from each group of 6 animals are presented in Table 1 (ewes) and Table 2 (lambs). The geometric means of the total worm burdens of the different groups of ewes and lambs which were slaughtered, are presented in Fig. 2.

Within a period of 3 months, from 4 April to 27 June 1989, the worm burdens of the lambs increased to a geometric mean of 82 122, with the major increase occurring during the last 6 weeks (Fig. 1). The highest individual worm burden recorded from the lambs was 246 681 and from the ewes 182 437. The worm burdens of the lambs and ewes slaughtered on 27 June 1989 did not differ significantly ($P > 0,05$). Only one (4%) of the ewes in the trial, and a single lamb (8%) among those killed on or after 16 May 1989 did not harbour any *Oestrus ovis* larvae.

DISCUSSION

The group of ewes slaughtered on 21 February 1989, indicated that the ewes harboured very few worms (595) when they were initially placed on the experimental paddock. After 6 weeks, on 4 April 1989, the mean worm burden of the

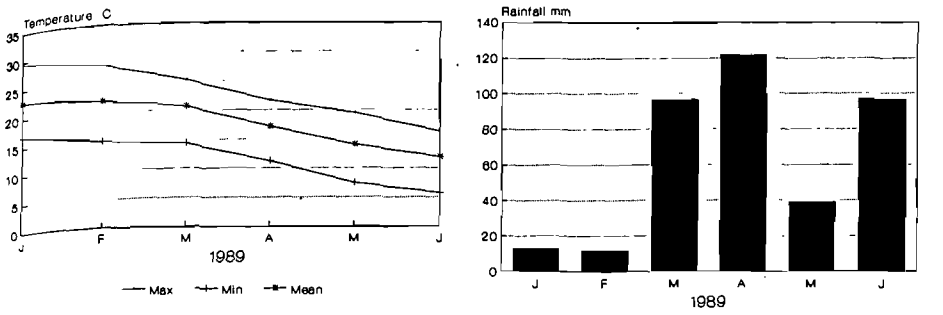

Fig. 1: **Mean monthly temperatures (°C) and total monthly rainfall (mm) recorded at Dunghye Park during 1989**

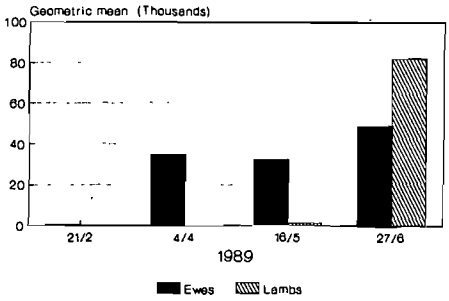

Fig. 2: **Geometric means of the worm burdens of groups of ewes and lamb slaughtered**

6 ewes slaughtered was 34 769, representing a 58-fold increase in the worm burdens of the second, compared to the first group of ewes (Table 1). The majority of these worms were undoubtedly acquired on the paddock. The lambs became infected with nematode parasites before the age of 8 weeks and harboured large parasite burdens before the age of 14 weeks.

It was not possible in the present trial to determine the source of infection responsible for the explosion in the worm burdens of the animals. The residual infection present in the ewes when they were placed on the experimental paddock, represented one probable source. Boag & Thomas[5] regarded such auto-infection as the most important source of infection to the grazing animal. Furthermore, Boag & Thomas[4] determined that worm eggs deposited during autumn, developed into infective larvae more rapidly and in larger numbers than those deposited during winter. Epidemiological studies in the Rûens region indicated that very large numbers of worm eggs were indeed deposited on the pastures in autumn[10]. In a similar Mediterranean-type climate, where pastures are naturally decontaminated by the hot and dry conditions in summer, Anderson[1] produced safe grazing for autumn and winter by treating animals early, and again late in the summer. Infective nematode larvae may, however, persist on the pasture for long periods of time. Besier & Lyon proved that nematode larvae and eggs can survive the Mediterranean summer in the faecal pellet[3]. Furthermore, Kates[8] rated infective larvae of *Teladorsagia* and *Nematodirus* the most successful (among the species tested by him) in surviving on pastures. *Teladorsagia* larvae are found in the soil[2] and can migrate to the surface through substantial layers of soil[7]. In the present study, the source of the large numbers of *Trichostrongylus falculatus* and *Trichostrongylus colubriformis* recovered from the ewes slaughtered from the experimental paddock, could not be determined (Table 1). Auto-infection was unlikely because none of the ewes slaughtered as indicators on 21 February 1989, when the remaining experimental animals were placed on the paddock, harboured any of these nematodes. If the pasture had been the source of these infections, infective larvae must have survived in the soil for approximately 18 months.

Nematodirus is a common parasite of sheep in the region, but develops into adult worms only in young animals[9 10 11]. While the results of the present study are in general agreement with those observations (Table 1 & 2), 11 of the 18 ewes slaughtered from February to May 1989 did harbour adult *Nematodirus* (Table 1). Periparturient relaxation of resistance in the ewes probably temporarily interfered with the process which usually prevents these parasites from maturing in adult animals.

Immature *Teladorsagia* have been shown to accumulate in sheep during winter and spring in this region, apparently owing to hypobiosis[9 10]. An accumulation of immature *Teladorsagia* in the sheep during winter was also noticed in the present trial and in the ewes and lambs slaughtered on 27 June 1989, respectively, 96% and 57% of the population were in the immature stage.

The results of the present study indicate that, in addition to the winter and spring treatments suggested by Louw[9], an anthelmintic treatment in autumn is necessary for all sheep in those parts of the Rûens which receive rain in autumn.

ACKNOWLEDGEMENTS

We wish to thank Mr L Fick, the farm owner, and Mr J de Wet, the farm manager, for making their facilities available for the trial. Financial support from the Foundation for Research Development of the CSIR, is gratefully acknowledged.

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THE SEASONAL ACTIVITY OF ADULT IXODID TICKS ON ANGORA GOATS IN THE SOUTH WESTERN ORANGE FREE STATE

L J FOURIE* and I G HORAK**

ABSTRACT

Adult ixodid ticks were collected at 2-weekly intervals for a period of 23 consecutive months from 15 to 20 Angora goats on a farm in the south western Orange Free State. A total of 6 ixodid tick species were recovered. *Rhipicephalus punctatus* was the most abundant and prevalent tick. It was present from spring to late summer. *Ixodes rubicundus* was the next most abundant tick and was present mainly from March or April to July with peak numbers present in April or May. The onset of this tick's activity appeared to be stimulated by low atmospheric temperatures.

Key words: Angora goats, ixodid ticks, seasonal activity

Fourie L.J.; Horak I.G. **The seasonal activity of adult ixodid ticks on Angora goats in the south western Orange Free State.** *Journal of the South African Veterinary Association* (1991) 62 No. 3, 104-106 (En.) Department of Zoology and Entomology, University of the Orange Free State, 9301 Bloemfontein, Republic of South Africa.

INTRODUCTION

Although sheep and cattle comprise the major portion of the livestock industry in the southern Orange Free State, about 60 000 Angora goats are also farmed in this province. Some research on tick infestation of Angora goats has been done in the eastern Cape Province[6 7 8] and the mortality of Angora goats caused by a paralysis-inducing tick species occurring in the southern Orange Free State has been recorded[2]. The present paper describes the seasonal activity of adult ixodid ticks on Angora goats on a farm in the south western Orange Free State. Similar studies on sheep and cattle on this farm have already been published[3 5].

MATERIALS AND METHODS

The study was conducted on the farm "Preezfontein", which is situated 10 km from the town of Fauresmith (29° 46'S; 25° 19'E) in the south western Orange Free State. The farm comprises an area approximately 6 000 ha in extent and topographically consists of flat as well as hilly ground. The vegetation in the area is defined as False Upper Karoo[1]. A fenced camp on the farm, encompassing 190 ha of vegetation typical of the region, was selected for this study.

The climate is semi-arid, with 69% of the annual rainfall occurring during the summer months. Mild to severe droughts occur periodically. Air temperatures exhibit major circadian and seasonal fluctuations with absolute temperatures varying between 39°C and -6,3°C. Total monthly rainfall during the study period was recorded at "Preezfontein" and mean maximum and minimum temperatures were obtained from a weather station at Fauresmith.

Four age and sex class categories of Angora goats were used, namely adult ewes, adult wethers, young wethers (7 months old) and kids. Each group initially consisted of 10 animals. Because tick-induced paralysis and other causes resulted in mortality, the groups of young goats and kids eventually contained between 5 and 10 animals each. The study on adult and young goats started during March 1988. During October 1988, the ewes on the farm kidded and their kids became the fourth group. During March 1989 the young wethers were excluded from the study and the kids born during the previous year were then considered as the young goat group. Angora ewes on the farm kidded again during December 1989, and the first observations on the second group of kids were made during January 1990.

Five animals belonging to each of the 3 or 4 survey groups were examined for ticks at 2-weekly intervals from March 1988 to January 1990. Each body region was carefully searched by parting the hair and visually inspecting the epidermis. All the adult ixodid ticks found were collected, placed in labelled bottles, identified and counted using a stereoscopic microscope. Burdens were expressed in terms of infestation density (numbers of ticks per kg host mass) and results presented in tabular and graphical form. To test for significant differences between sex ratios a Chi-squared test was used. Where reference is made to tick activity, it is inferred that these ticks actively quest for hosts either from the ground or from vantage points on the vegetation and then enter the parasitic phase of their life cycles.

In order to quantify seasonal abundance, the tick infestation densities of the various groups of goats were pooled. Except for *I. rubicundus* and *R. punctatus*, the numbers of the other tick species were too low to determine seasonal tendencies.

RESULTS

Total monthly rainfall and mean atmospheric temperatures are graphically illustrated in Fig. 1 & 2.

The total numbers and relative abundance of adult ixodid ticks collected from the goats are summarised in Table 1. Except for *Rhipicephalus evertsi evertsi* and *Rhipicephalus gertrudae*, where the sample sizes were too small, there were significant differences ($p < 0,05$) in the sex ratios of male to female ticks. With the exception of *I. rubicundus*, more male than female ticks were present on the goats.

The seasonal abundances of *I. rubicundus* and *R. punctatus* are graphically illustrated in Fig. 3 & 4.

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Table 1: **Adult ixodid ticks collected from Angora goats on the farm "Preezfontein"**

Tick species	Numbers recovered Males	Females	Total	Relative abundance	% goats infested
Hyalomma marginatum rufipes	33	8	41	0,69	3,6
Hyalomma truncatum	45	27	72	1,21	4,7
Ixodes rubicundus	897	1155	2052	34,34	36,2
Rhipicephalus evertsi evertsi	3	0	3	0,05	1,4
Rhipicephalus gertrudae	16	10	26	0,44	0,4
Rhipicephalus punctatus	2084	1698	3782	63,29	53,6

the long term, differentially affect the epidemiology of the toxicoses caused.

Hyalomma truncatum was the most abundant of the 2 *Hyalomma* species collected. Most (>65%) of these ticks were collected during the summer months (January - March). The relatively low number collected from goats in the present study, suggests either that these animals are poor hosts of these ticks or that Angora goats minimise contact with the ticks through behavioural patterns. Both *H. marginatum rufipes* and *H. truncatum* seek hosts from the ground and not from the vegetation and this may affect tick/host contact.

Although free-living, newly moulted adult *I. rubicundus* are already present during December or January (summer), this tick is parasitic virtually only during the winter months, and it is reasonable to assume that low temperatures stimulate the onset of tick activity. A comparison of mean minimum temperatures for 1988 and 1989 shows that those for the 3 months preceding tick activity (January-March) were lower during 1989 than during the 1988 season. During March 1989

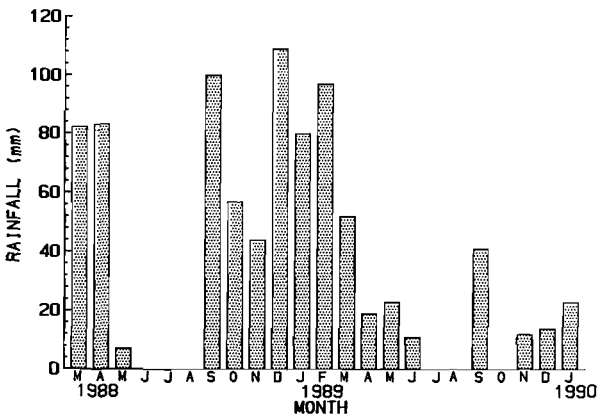

Fig. 1: **Total monthly rainfall on the farm "Preezfontein" from March 1988 to January 1990**

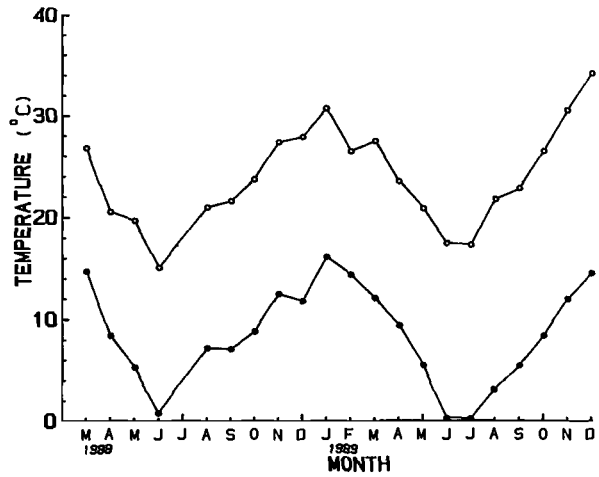

Fig. 2: **Mean monthly minimum and maximum temperatures at Fauresmith for the period March 1988 to December 1989**

DISCUSSION

There are some differences in the relative abundance of adult ticks of the various species infesting the goats and those recovered from sheep and cattle examined on the same farm in previous surveys. The relative abundances of the 4 major tick species recovered from the 3 host species on the farm, are summarised in Table 2.

Hyalomma marginatum rufipes was proportionately most abundant on cattle, *I. rubicundus* on sheep and *R. punctatus* on the goats. *H. marginatum rufipes* was least abundant on the goats, *Hyalomma truncatum* on the sheep and *I. rubicundus* on the cattle. These differences in relative abundance are probably due either to host preference of the ticks, to habitat preference of the ticks or hosts, or to behavioural differences of the hosts which may affect tick-host contacts. Accordingly, depending on the number and type of hosts frequenting a specific area, the numbers of ticks within this area may increase because of the greater availability of suitable hosts. Since *H. truncatum, I. rubicundus* and *R. punctatus* can all cause tick toxicosis[2,4], stocking densities may, in minimum temperatures fell below 10°C on 6 occasions, compared to only once during March 1988. This may explain why infestation occurred about one month earlier during 1989 than in 1988.

The variation in the commencement of tick activity and peak activity periods between years is significant. In an earlier survey on sheep *I. rubicundus* became active on "Preezfontein" during April 1987 and reached a peak during May[3]. In both surveys, tick burdens reached a peak within 4 weeks after the commencement of the activity.

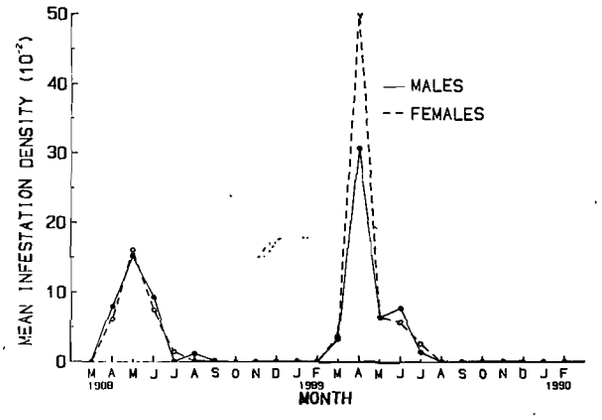

Fig. 3: **The seasonal abundance of adult *Ixodes rubicundus* on Angora goats on the farm "Preezfontein" in the south western Orange Free State (infestation density = number of ticks per kg host body mass)**

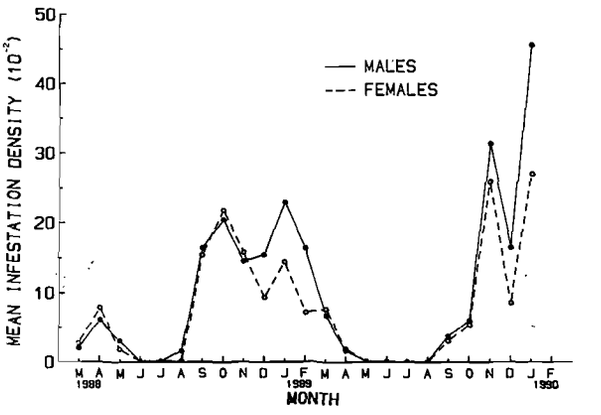

Fig. 4: **The seasonal abundance of adult *Rhipicephalus punctatus* on Angora goats on the farm "Preezfontein" in the south western Orange Free State (infestation density = number of ticks per kg host body mass)**

It is important to know when the ticks become active in order to remove stock from infested camps or apply acaracidal treatment before peak burdens are reached. On farms in the southern Orange Free State and parts of the Karoo, where these precautions were not taken during 1989, the earlier than expected activity of the ticks caused severe stock losses, with reports of up to 100 small stock mortalities on a single property not uncommon. The development of a weather-based model in order to predict the onset of tick activity is therefore regarded as a high priority and is currently receiving attention.

Unlike *I. rubicundus* the period of activity of *R. punctatus* commenced one month later in 1989 than during 1988. One could speculate that as this tick normally prefers the warmer temperatures of spring and summer, the colder winter of 1989 delayed the onset of its activity during that year.

ACKNOWLEDGEMENTS

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Table2 : **The relative abundance of the major tick species recovered from goats, sheep and cattle on the farm "Preezfontein"**

Tick species	Relative abundance (%) on: Goats	Sheep	Cattle
Hyalomma marginatum rufipes	0,7	16,3	36,2
Hyalomma truncatum	1,2	0,1	7,1
Ixodes rubicundus	34,3	82,2	3,2
Rhipicephalus punctatus	63,3	1,3	9,4
Other species	0,5	0,1	44,1

ARTHRITIS IN SLAUGHTER PIGS

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ABSTRACT

Joints obtained from 192 pig carcases were examined by means of standard microbiological and macro- and histopathological procedures. Approximately 32% of the joints were considered normal; 35,5% showed lesions consistent with osteochondrosis and a non-specific synovitis was present in 24,4%. Only 6,1% of joints were arthritic and yielded either *Staphylococcus aureus* or *Streptococcus* spp. The remainder (2,3%) had periarticular lesions such as abscesses. The study emphasises that an accurate diagnosis and correct evaluation of pig carcases showing joint lesions, is absolutely essential if a high standard of meat inspection is to be obtained and unnecessary economic losses are to be avoided.

Key words: Pigs, arthritis, osteochondrosis, meat inspection

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INTRODUCTION

Arthritis has been a common cause of the total and partial condemnation of pig carcases in the Republic of South Africa (RSA)[3]. *Erysipelothrix rhusiopathiae* was found to be responsible for 48% of the cases of arthritis and *Streptococcus* spp., *Actinomyces (Corynebacterium) pyogenes* and *Staphylococcus aureus* for 20%, 4% and 2% respectively, whilst in 26% of the arthritic joints, no micro-organisms could be cultured in spite of marked pathological changes in the joints[3]. Consequently, farmers were advised to vaccinate their pigs against swine erysipelas and it was considered imperative that both the *Lnn. iliac mediales et laterales* and the *Lnn. axilares primae costae* be examined during the meat inspection of pig carcases if the correct diagnosis of arthritis was to be made. In subsequent years, the incidence of arthritis, when measured against abattoir condemnation figures, decreased[5].

In 1987 and 1988 there was an increase in the number of total and partial condemnations of pig carcases, which were recorded by personnel of the meat inspectorate as being due to arthritis. In one instance, the condemnation rate for arthritis was as high as 55% of the total number of pig carcases condemned at an abattoir in a particular month (Table 1)[5]. The economic and pig health implications associated with these excessive condemnations, prompted an investigation into the problem.

The aim of this study was to determine which micro-organisms were involved in infectious arthritic lesions and to describe the patho-anatomical nature of the joint lesions.

MATERIALS AND METHODS

A total of 262 joints which had been condemned as being arthritic by the meat inspectorate, were obtained from 192 pig carcases from 4 Grade A abattoirs in the Pretoria/Johannesburg area. Each joint was opened and examined and macroscopic changes were recorded. Specimens for histopathology were taken from articular surfaces and synovial membranes. Swabs were taken aseptically from joints and plated on tryptose blood agar and incubated aerobically at 37°C for 72 h.

RESULTS

No changes in the joint cartilage or capsule were noted in 31,7% of the joints, and these were considered to be normal. Macroscopic lesions of osteochondrosis were recorded in 35,5% of the joints examined. Articular cartilage in these joints demonstrated buckling, full thickness cartilage flaps and ulcers, while synovial villi showed feathery hypertrophy. Microscopically, the synovial villi contained occasional lymphoid cuffs, haemosiderin-laden macrophages and embedded cartilage chips. A non-specific synovitis, characterised by mild to severe hyperaemia of the joint capsule, with or without periarticular bruising, and by the histological presence of embedded cartilage chips in many of the synovial membranes, was found in 24,4% of the joints. Only 6,1% of the joints were found to have pathology typical of infectious arthritis[3]. *Staphylococcus aureus* or *Streptococcus* spp. were isolated from these joints and no *E. rhusiopathiae* organisms were found. These joints showed a progressive subacute to chronic sero-fibrinous to fibrinopurulent arthritis with marked hypertrophy of the synovial villi and fibrous thickening of the joint capsule with or without pathology of the articular cartilage. The increase of synovial fluid varied from negligible to copious and varied from yellowish-brown to red. Periarticular lesions, mainly abscesses, were found around 2,3% of the joints otherwise diagnosed as being normal.

DISCUSSION

The reason why many of the joints in this study were regarded as being arthritic at meat inspection, was because of the increased and discoloured synovial fluid and prominent synovial villi. A significant number of joints (24,4%) showed a synovitis which was possibly traumatic in nature with the inflammation of the synovial membranes being induced by

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Table 1: **Pig carcase condemnations due to arthritis for months January-August 1988 at 4 Grade A abattoirs[6]**

Abattoir	Month	Number pigs slaughtered	Number carcases condemned	% carcases condemned due to arthritis
A	January	5 575	155	15
	February	5 565	180	19
	March	7 073	170	21
	April	4 628	79	27
	May	*	*	*
	June	5 879	170	25
	July	*	*	*
	August	6 189	161	25
B	January	4 115	150	38
	February	4 824	121	37
	March	5 429	116	24
	April	4 750	144	55
	May	5 243	277	41
	June	4 780	232	43
	July	4 805	201	46
	August	5 510	106	46
C	January	*	85	20
	February	*	92	29
	March	*	122	16
	April	*	72	21
	May	12 176	123	12
	June	12 345	139	36
	July	10 496	100	27
	August	12 123	76	38
D	January	10 629	69	42
	February	12 589	59	34
	March	14 539	68	44
	April	11 156	61	54
	May	12 690	32	44
	June	13 577	39	49
	July	12 360	65	23
	August	13 765	66	41

* = Figures not obtainable

damaged articular cartilage. It was considered that early stages of osteochondrosis played a significant role in many of these cases. Since many of these joints also showed periarticular bruising, trauma from loading, transport or preslaughter handling probably also contributed to the increased synovial fluid.

Osteochondrosis, a non-infectious condition which is usually bilaterally symmetrical, is characterised by focal disruptions of articular cartilage at the chondro-osseus junction resulting in full-thickness cartilaginous folds, buckles, flaps or ulcers[2]. The regional lymph nodes associated with joints showing osteochondrosis are normal[2].

In some cases this could have been as a result of mild peri-articular bruising and joint concussion with a concomitant increase of joint fluid, which may have been blood-stained. With these joints being opened at the abattoir, any increased synovial fluid had possibly drained away by the time they were examined in the laboratory.

The incidence of infectious arthritis was relatively low. In contrast to previous findings[3], *E. rhusiopathiae* did not appear to be an important aetiological agent in this study. Joints with infectious arthritis characteristically contain an exudate, have thickening of the synovial membranes, articular erosions and possibly pannus[2]. Synovial villi show moderate to severe polypoid hypertrophy and inflammation with occasional infarction of villus tips. A most important finding is that the regional draining lymph nodes are usually enlarged due to lymphoid hyperplasia and possible suppuration[2]. It is essential that the relative lymph nodes be examined when judging whether a carcase has infectious arthritis or not. This includes the *Lnn. axilares primae costae,* the main lymph nodes draining the fore-limb, which are not routinely examined at abattoirs[3]. A high percentage of the joints (75,0%) in this study were elbow joints.

It was felt that the non-infectious joint conditions (91,6%) noted in this study did not, in most cases, warrant partial condemnation of the limb. This led to unnecessary mutilation of the carcase and economic loss to the producer as a result of carcase mass loss and downgrading. The downgrading of arthritic pig carcases that have been subjected to partial condemnation of the forelimb, appears to be unrealistic. This aspect of grading pig carcases needs to be investigated further.

Pigs likely to have osteochondrosis are those that walk on their toes with a stilted gait, have carpal and elbow joints which are flexed, rear limbs which are hyperextended, an arched back or walk with excessive lateral swaying[1,2]. Further research should be conducted into the occurrence and economic implications of this condition in the national pig herd. However, it is imperative that both the *Lnn. iliaci mediales et laterales* and the

Lnn axilares primae costae be examined when inspecting pig carcases and that this should play an integral role in the differentiation of infectious and non-infectious joint lesions. The functions of the meat inspectorate should also include protecting the producer from unnecessary economic losses, by not carrying out unjustified partial and total condemnations of carcases at the abattoir. This study confirms previous recommendations concerning condemnations at abattoirs[3 4]. The accurate diagnosis and correct evaluation of a porcine carcase showing joint lesions, is absolutely essential if a high standard of meat inspection and meat hygiene is to be obtained. This must be performed by a suitably qualified veterinarian. In addition, data on economically important conditions diagnosed at abattoirs, should be utilised in disease surveillance programmes to the best advantage of the livestock industry in the RSA.

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Book review/Boekresensie

FELINE PRACTICE THE "IN PRACTICE" HANDBOOKS

E BODEN (EDITOR)

1st Edition. Balliere Tindall, W.B. Saunders, 24-28 Oval Road, London, NW1 7DX. 1991. pp VIII and 220, 31 tables, 7 diagrams and 78 plates.

As noted by the authors of the chapter on feline nutrition, "feline medicine has (until recently) been the poor relation of veterinary science". The publication is an attempt to address the dearth of information relating to the domestic feline, and while it can not be described as being an authoritative text, it offers a comprehensive review of a number of syndromes and conditions that are likely to confront most small animal practitioners.

Contained in this book, are a number of excellent reviews on syndromes including anaemia, hepatopathy and dyspnoea, where emphasis is placed on diagnostic procedures and differential diagnoses. A number of useful algorithms designed to facilitate diagnosis are provided.

The chapter on feline nutrition is particularly informative, in that a host of idiosyncrasies relating to diet are highlighted. The nutritional management of medical cases such as diabetes mellitus and chronic renal failure are described, and the authors provide some sound advice on the feeding of the debilitated cat.

An extensive and decidedly welcome review on ocular diseases in the cat, with numerous high quality colour plates will enable both student and practitioner alike, to improve their knowledge in this field. As with the other topics discussed, each chapter concludes with a reference list should the reader require further more specific information.

While other organ systems and syndromes still require reviews of the quality offered herein, this handbook will be an invaluable addition to the library of the feline practitioner and undergraduate student alike.

R.D. Kirkpatrick

THE EFFICACY OF ULTRASONIC PEST CONTROLLERS FOR FLEAS AND TICKS

C R BROWN* and B D LEWIS*

ABSTRACT

Two ultrasonic pest controllers, a pet-collar unit and a large unit for household use, were tested for their efficacy in repelling fleas and ticks in a choice chamber. Neither unit had any affect on the distribution of fleas or ticks in the choice chamber up to 24 h exposure, and activity of fleas, ticks and cockroaches was unimpaired. The study extends and supports previous findings that ultrasound is ineffective as a means of controlling common pests of households and pets.

Key words: Ultrasound, fleas, ticks, pest control

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INTRODUCTION

Ultrasound generally refers to high frequency sound inaudible to the human ear (above approximately 20 kHz). Although inaudible to humans, some insects are capable of detecting ultrasound. In particular, some moths respond, by evasion, to ultrasound in the 20-40 kHz range, the range used for prey detection by many insectivorous bats[1 13]. Such observations provided an early stimulus for investigating the use of ultrasound for the control of agricultural insect pests. Results of field trials, mainly on cotton bollworm, tobacco budworm and cabbage looper moths, are conflicting, some showing promise[6 12] and others no effect at all[1 2]. In contrast, there is no a priori reason to suggest that ultrasound will be effective in repelling other insects, in particular common household pests (mainly cockroaches and fishmoths) and pests of domestic pets (fleas and ticks). There is little evidence that domestic insect pests have receptors capable of detecting ultrasound, although fleas may be capable of detecting ultrasonic frequencies in the region of 100 to 10 000 kHz[3]. This is far above the 20-60 kHz output of commercial ultrasonic pest repellers. Nevertheless, the idea of non-chemical control of household pests is an attractive one, and a wide range of ultrasonic pest controllers claiming to repel insect pests in the domestic environment, is available in the United States and Europe. The efficacy of some of these devices has been the subject of several investigations, both in the laboratory[4 5 10 14] and under more natural conditions[8 10 15]. Most of these studies suggest that ultrasonic devices are ineffective in controlling domestic pest populations, although there is still some controversy on the matter[7].

It is only relatively recently that ultrasonic pest controllers have become available on the South African market, mainly through mail order companies advertising in newspapers and magazines. Subjective reports from purchasers that these devices are effective, led us to test the repellent effects of 2 such devices on fleas and ticks in a choice chamber.

MATERIALS AND METHODS

Adult cat fleas *(Ctenocephalides felis)* were collected from domestic cats and dogs. Fleas were either used on the day they were collected, or kept overnight in a glass jar with animal hair. Fleas were not fed and fresh fleas were used for each trial.

Adult *Rhipicephalus simus* ticks were supplied by the Tick Research Unit at Rhodes University, (Grahamstown, Republic of South Africa) and supplemented with adult ticks recovered from domestic dogs in the Grahamstown area. Because only limited numbers were available, some ticks were used in more than one trial, but none more than 3 times over the entire period of the experiments and not in successive trials.

Two ultrasonic devices, purchased from mail order companies, were tested. The smaller unit, a flea and tick collar unit for pets is made in Taiwan but bears no brand name. It is designed for attachment around the neck of a cat or dog, or to be placed in a kennel or pet basket. The instructions claim that the high frequency sound will work by repelling pests within a range of 4 feet (1,2 m). It further claims that fleas within this range will stop jumping within seconds and so will not jump onto pets fitted with the device.

The larger unit also carries no brand name, nor is there any indication of its country of origin. It is designed for household use and is powered by two 9V batteries or supplied mains adapter. The specifications claim that the unit sweeps continuously over 30 to 65 kHz, has a sound pressure level of 130 dB, and is effective in an area of 2 000 to 2 500 square feet (180-225 m^2). The rate at which the device sweeps its frequency range is adjustable by the user. Both devices were tested before and after the experiments to confirm that they were producing ultrasound.

The test chamber comprised a Y-shaped plywood box with a broad base (16 x 10 x 8 cm) and two narrow arms (30 x 8 x 8 cm). The broad base of the Y was partly divided by a cardboard baffle, effectively dividing the chamber into a left and right side. A 3-piece perspex lid allowed for observation and easy access to the chamber. Linen-covered rectangular holes (6 cm^2) cut in the ends of the arms allowed the ultrasonic devices to be placed immediately outside the chamber with their transponders facing into the chamber. The chamber was lined with dressmaker's batting, covered with white linen, to absorb the ultrasound and restrict it, as far as possible, to one arm of the chamber. Tests with an ultrasonic bat detector (QMC Mini Bat Detector)

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Table 1: **Effect of the pet-collar ultrasound unit on the distribution of *Ctenocephalides felis* in a choice chamber. NS = not significant**

Trial No.	No. of fleas	Initial distribution quiet arm	Initial distribution unit arm	Final distribution quiet arm	Final distribution unit arm	χ^2	Significance
1	19	11	8	8	11	1,349	NS
2	21	12	9	9	12	1,215	NS
3	28	5	23	5	23	0,061	NS
4	17	8	9	8	9	0,059	NS
5	33	16	17	17	16	0,030	NS
6	29	11	18	5	24	4,431	$P<0,05$
7	20	6	14	7	13	0,060	NS
8	30	16	14	23	7	5,659	$P<0,05$
Pooled	197	85	112	82	115	0,129	NS

established that no ultrasound from the pet-collar unit penetrated to the end of the "quiet" arm of the chamber. With the large unit in position, ultrasound in the "quiet" arm of the chamber was still detectable, but was substantially less than in the arm with the unit, attenuation being estimated at 80 to 90%. Trials were carried out in a constant environment room at 24°C with a 12L 12D light cycle.

Fleas or ticks were introduced into the chamber at the base of the Y. The number of insects used varied depending on availability, but was never fewer than 11 fleas and 10 ticks. Fleas and ticks were left for at least 60 min to distribute themselves in the chamber and their distribution (left or right arm) noted (initial distribution). The ultrasonic unit being tested was then placed at the end of one of the arms of the chamber and switched on. Initially, the unit was placed at the left and right arms at random based on odd (left) and even (right) numbers generated by a random number generator. In a second trial of the large unit with fleas, the unit was sometimes deliberately placed in the arm containing the most fleas.

After the unit was switched on, the chamber was left undisturbed for 24 h. At the end of the trial, the number of fleas or ticks in each side was again counted (final distribution). Overall, 8 trials were carried out on fleas with the pet-collar unit and 7 on ticks. Fourteen and 6 trials were carried out with the large unit on fleas and ticks, respectively.

Chi-square (χ^2) tests, corrected for continuity[16], were carried out for each individual trial to establish any significant differences between initial and final distributions of fleas and ticks in the chamber and, where χ^2 values were homogeneous, pooled χ^2 were obtained by summing the initial and final distributions.

RESULTS

Electronic analysis showed that the pet-collar unit produced pulsed ultrasound at a frequency of 35 kHz, giving a 2 millisecond (ms) tone burst every 40 ms. The sound pressure level (SPL) of the unit could not be measured, but output from the unit was virtually undetectable with the bat detector at ranges > 30 cm.

The larger unit produced modulating sound which cycled between 20 and 37 kHz with no break in modulations. SPL was not measured, but the unit was detectable with a bat detector for at least 10 m.

Of 8 trials with the pet-collar unit against fleas, 6 trials showed no significant difference in the distribution of fleas before and after the unit had been switched on, one trial showed a significant change in distribution towards the unit and one trial away from the unit (Table 1). Overall, there was no significant change in the distribution of fleas after 24 h exposure to ultrasound (pooled $\chi^2 = 0,129$, $P > 0,50$).

Ticks also showed no response to ultrasound generated by the pet-collar unit, all 7 trials showing no significant differences in their initial and final distributions (pooled $\chi^2 = 0,006$; $P > 0,75$) (Table 2).

Four out of 6 trials on fleas using the large unit, showed a significant change in distribution after 24 h exposure. However, the movement was towards the ultrasound (Table 3). χ^2 values for individual trials were not homogeneous and were therefore not pooled. A further series of 8 trials with substantially more

Table 2: **Effect of the pet-collar ultrasound unit on the distribution of *Rhipicephalus simus* in a choice chamber**

Trial No.	No. of fleas	Initial distribution quiet arm	Initial distribution unit arm	Final distribution quiet arm	Final distribution unit arm	χ^2	Significance
1	20	8	12	10	10	0,469	NS
2	24	13	11	12	12	0,040	NS
3	20	8	12	9	11	0,052	NS
4	20	9	11	10	10	0,273	NS
5	39	22	17	21	18	0,026	NS
6	24	14	10	12	12	0,386	NS
7	20	11	9	10	10	0,051	NS
Pooled	167	85	82	84	83	0,006	NS

fleas showed no significant difference between initial and final distributions after 24 h exposure to ultrasound in any individual trial (Table 4), or overall (χ^2 = 0,082; P > 0,75).

There was no significant difference in the initial and final distributions of ticks in any of the 6 individual trials (Table 5) or overall (χ^2 = 0,219; P > 0,50).

strongly suggesting that the devices are ineffective for repelling fleas and ticks. These results are consistent with previous studies on ultrasonic pest controllers. For example, several studies have shown that cockroaches are unaffected by a wide range of ultrasonic frequencies[4 5 9 10]. More specifically, Rust & Parker[14] found no movement of fleas away from an

evidence that they are not adversely affected by ultrasound. Similarly cockroach nymphs have been found inside ultrasonic pest repellers after trials in apartment buildings, showing that cockroaches were even using the devices for harbourage[10].

The claim in the instructions accompanying the pet-collar unit used in the pre-

Table 3: **Effect of the large ultrasound unit on the distribution of *Ctenocephalides felis* in a choice chamber during the first series of trials**

Trial No.	No. of fleas	Initial distribution		Final distribution		χ^2	Significance
		quiet arm	unit arm	quiet arm	unit arm		
1	24	21	3	8	16	59,520	P < 0,001
2	34	18	16	8	26	10,655	P < 0,001
3	17	3	14	2	15	0,010	NS
4	18	9	9	1	17	12,500	P < 0,001
5	42	21	21	18	24	0,595	NS
6	11	6	5	1	10	7,425	P < 0,001

Table 4: **Effect of the large ultrasound unit on the distribution of *Ctenocephalides felis* in a choice chamber during the second series of trials**

Trial No.	No. of fleas	Initial distribution		Final distribution		χ^2	Significance
		quiet arm	unit arm	quiet arm	unit arm		
1	43	31	12	32	11	0,029	NS
2	42	30	12	33	9	0,729	NS
3	39	30	9	31	8	0,036	NS
4	21	11	10	16	5	3,866	P < 0,02
5	33	6	27	3	30	1,273	NS
6	45	6	39	5	40	0,048	NS
7	37	5	32	2	35	1,445	NS
8	46	34	12	34	12	0,028	NS
Pooled	306	153	153	156	150	0,082	NS

DISCUSSION

Ultrasonic sound is rapidly attenuated by distance and is diffracted by solid objects. In the present study, absorption and attenuation was such that either no or very little ultrasound was present in the "quiet" arm of the choice chamber. If ultrasound generated by the devices repelled insects as claimed, one would expect a significant movement of fleas and ticks away from the ultrasonic units into the sound shadow of the "quiet" arm. Such movement was not observed, ultrasonic device in a cardboard tube. Furthermore, Dryden et al[8] and Schein et al[15] showed that pet-collar devices were ineffective in reducing flea numbers on cats and Schein et al[15] found no difference between numbers of fleas and ticks initially placed on dogs with ultrasonic pet-collars and on control dogs, even after 14 d exposure.

In the present study, fleas and ticks were observed on the linen at the end of an arm of the choice chamber within one cm of the transponder, supporting

sent study that fleas will cease jumping within seconds of exposure to the collar, is also unfounded. Fleas in the chamber were regularly observed to jump and previous studies have demonstrated that ultrasound has no effect on fleas' jumping or on their normal circadian rhythm of activity[11 14]. Rust & Parker[14], however, showed that bursts of CO_2 did elicit increased activity, as might be expected of insects that rely on CO_2 concentration and thermal and visual cues to locate hosts. Ticks in the present study, on the

Table 5: **Effect of the large ultrasound unit on the distribution of *Rhipicephalus simus* in a choice chamber**

Trial No.	No. of fleas	Initial distribution quiet arm	unit arm	Final distribution quiet arm	unit arm	χ^2	Significance
1	20	9	11	11	9	0,455	NS
2	20	10	10	10	10	0,050	NS
3	14	4	10	6	8	0,788	NS
4	10	4	6	6	4	0,938	NS
5	30	18	12	17	13	0,035	NS
6	20	12	8	10	10	0,469	NS
Pooled	114	57	57	60	54	0,219	NS

other hand, showed little movement after initially distributing themselves in the chamber, even when they had settled within one cm of the ultrasonic devices. Gently exhaling in their vicinity, however, did elicit movement showing that they were not immobilised by the ultrasound.

The leaflet accompanying the large unit used in the present study also claims that the unit will stun larger insects such as moths, bees and cockroaches, rendering them immobile and allowing them "to be swept away at leisure". To test this claim, a single trial with 7 cockroaches (*Periplaneta americana)* was carried out. The trial was conducted as described for fleas and ticks, but cockroaches were provided with food and water and a cardboard tube was placed at the end of each arm of the chamber as harbourage. Ultrasound from the large unit had no noticeable effect on cockroach activity, cockroaches at night being especially active with no signs of immobility. Although there were too few cockroaches for statistical purposes, there was also no change in their distribution after 24 h exposure to ultrasound, but after 48 h all 7 cockroaches were clustered in the tube immediately in front of the ultrasound unit, but immediately moved when disturbed.

In addition to activity, ultrasound has also been shown to have no effect on reproduction in either cockroaches[10] or fleas[8 10], the latter despite claims that the use of ultrasonic pet collars inhibit flea population growth[7].

The present study demonstrates that the 2 ultrasonic devices tested fall short of claims in their specification and instruction leaflets with regard to their performance. Furthermore, the study has failed to substantiate that these ultrasonic devices have any efficacy in repelling common household pests. On the contrary, this and other studies have shown such devices to be ineffective for controlling fleas, ticks or cockroaches.

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A PRELIMINARY INVESTIGATION INTO THE IMMOBILISING POTENTIAL OF A TILETAMINE/ZOLAZEPAM MIXTURE, METOMIDATE, A METOMIDATE AND AZAPERONE COMBINATION AND MEDETOMIDINE IN OSTRICHES (Struthio camelus)

J VAN HEERDEN* and R H KEFFEN**

ABSTRACT

Ostrich chicks (n=34) were successfully immobilised with intramuscular injections of a tiletamine/zolazepam mixture at dosages of 5, 10, 15 and 20 mg kg^{-1}; with metomidate at dosages of 15 and 20 mg kg^{-1} and with a metomidate/azaperone combination at respectively 20 and 6,6 mg kg^{-1}, and 10 and 3,3 mg kg^{-1}. Unsatisfactory immobilisation with violent body movements and self traumatisation were observed in an adult ostrich after the intramuscular administration of a tiletamine/zolezepam mixture. Anaesthesia was achieved by the administration of metomidate in combination with azaperone. Medetomidine administered at a dosage rate of 0,1 mg kg^{-1} did not result in immobilisation of ostrich chicks (n=4). Findings in ostrich chicks should not necessarily be extrapolated to adult birds.

Key words: Ostrich, *Struthio camelus,* immobilisation, tiletamine, zolazepam, metomidate, azaperone, medetomidine

Van Heerden J.; Keffen R.H. **A preliminary investigation into the immobilising potential of a tiletamine/zolazepam mixture, metomidate, a metomidate and azaperone combination and medetomidine in ostriches *(Struthio camelus)*.** *Journal of the South African Veterinary Association* (1991) 62 No. 3, 114-117 (En.) Department of Companion Animal Medicine and Surgery, Faculty of Veterinary Science, Medical University of Southern Africa, 0204 Medunsa, Republic of South Africa

INTRODUCTION

Successful field immobilisation of free-ranging ostriches (*Struthio camelus)* by chemical means would require the intramuscular or subcutaneous administration of an immobilising agent. The intramuscular administration of drugs in ostriches has met with variable success; large dosages of drugs, and difficult induction and recovery periods being some of the recorded problems[3 4 6 7]. A tiletamine/zolezepam combination has been used either intramuscularly (4-12 mg kg^{-1}) or intravenously (2-8 mg kg^{-1}) as an induction agent[6]. Tiletamine has also been used in combination with ketamine to restrain ostriches[3]. As yet, an effective immobilising drug for intramuscular administration has not been identified.

Tiletamine hydrochloride, a cyclohexamine anaesthetic agent, in combination with zolazepam hydrochloride, a benzodiazepine tranquilliser, results in cateleptoid anaesthesia and analgesia in mammals[8]. The intramuscular administration of the drug usually results in smooth induction and recovery from anaesthesia, good skeletal muscle relaxation as well as a retention of palpebral and pharyngeal reflexes[8]. In mammals, the clinical effects of medetomidine, a potent selective and specific agonist of pre- and postsynaptic alpha 2-adrenoceptors, include sedation, anxiolysis and analgesia[5] Metomidate, an imidazole derivative, alone or in combination with azaperone, a butyrophenone tranquilliser, has been used as an immobilising agent in mammals and birds[1]. Metomidate has strong central muscle relaxant properties, but no analgesic activity and is often used in combination with azaperone. Azaperone is one of the butyrophenone tranquillisers[1].

This paper reports on an evaluation of the immobilising potential of intramuscular injections of a tiletamine/zolazepam mixture, metomidate, a metomidate/azaperone combination and medetomidine in ostriches.

MATERIALS AND METHODS

Ostrich chicks (n=39) of both sexes, ranging in body mass from 6,4 to 22,5 kg (Table 1) and one adult male ostrich were used in this investigation. These partially tame, apparently healthy birds were kept under semi-intensive conditions on a commercial ostrich farm. All birds were subjected to a single treatment each.

Prior to the administration of the test drug, the birds were manually restrained and body mass, rectal temperatures as well as heart and respiratory rates were recorded. Birds were sexed by cloacal inspection.

Birds received one of the following treatments: a tiletamine/zolazepam combination (Zoletil 50, Reading) (250 mg ml^{-1}) at 5, 10, 15 or 20 mg per kg; metomidate (Hypnodil, Janssen) (50 mg ml^{-1}) at 15 or 20 mg per kg, metomidate at 20 mg kg^{-1} in combination with azaperone (Stresnil, Janssen) (40 mg ml^{-1}) at 6,6 mg kg^{-1}, metomidate at 10 mg kg^{-1} in combination with azaperone at 3,3 mg kg^{-1} and medetomidine (Domitor, Farmos Group) (1 mg ml^{-1}) at 100 ug kg^{-1} (Table 1).

Following intramuscular administration of the drug, the birds were carefully observed. Times to immobilisation, any visible reactions to the drug and time to recovery were recorded. Rectal temperatures, pulse and respiratory rates were also recorded at regular intervals. Pedal reflexes were evaluated at the same time. Birds were regarded as immobilised when they assumed lateral or sternal recumbency. Birds were considered to have recovered from the effects of the drug when

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Table 1: **Number, sex, body mass of ostriches immobilised with either a tiletamine/zolazepam mixture, a metomidate/azaperone combination, metomidate or medetomidine. Time to immobilisation and time immobilised are also given**

Drug	n	Sex m	Sex f	Body mass (range)kg	Dosage mg kg^{-1}	Time to immobilisation ($\bar{x}$,SD)	Time immobilised ($\bar{x}$,SD)
Tiletamine/	5	4	1	6,6-10,7	5	1 min 56s (± 34s)	33 min 10s (± 9 min)
zolazepam	5	2	3	6,4-9,2	10	3 min 15s (± 2 min 19s)	79 min (± 33 min 12s)
	1		1	12,2	15	1 min 25s	106 min
	4	2	2	9,7-16,1	20	1 min 9s (± 15s)	126 min 24s (± 48 min)
	1	1		estimated 130	650*	15 min 42s	160 min
Metomidate	5	4	1	8-15,1	15	3 min 23s (± 40s)	44 min 38s (± 15 min 12s
	5	3	2	11-14,1	20	2 min 20s (± 58s)	58 min 36s (± 18 min 48s
Metomidate/	5	2	3	8,4-22,5	20/6,6	1 min 50 (± 18 min 24s)	119 min (± 18 min 54s)
asaperone	5	1	4	10,8-13,3	10/3,3	2 min 58s (± 1 min 35s)	45 min (± 9 min 48s)
Medetomidine	4	2	2	11,8-18,3	0,1	not immobilised	

*total dosage
SD = standard deviation

they were able to stand on their feet unassisted.

Time to immobilisation and time immobilised were examined by regression analysis against dosage of drug or drug combinations.

RESULTS

The body mass and sex of the ostriches as well as the average times to immobilisation and the average time the ostriches remained immobilised are presented in Table 1. Prior to the injection of test drugs, rectal temperatures, respiratory and heart rates were respectively 39,9°C (n = 39; SD = 0,54; range = 39-41,1) 25 cycles min^{-1} (n = 39; SD = 39,9 range 12-60) and 121 beats min^{-1} (n = 39; SD = 23; range 80-164). The adult male ostrich could not be examined prior to administration of the drug. Average respiratory and heart rates after the administration of the test drugs are presented in Table 2. Increased heart rates were recorded in birds treated with the tiletamine/zolezepam combination at a dosage rate of 20 mg kg^{-1}, at 15 min after the injection of metomidate at 15 mg kg^{-1}, and after the administration of metomidate (20 mg kg) and the metomidate/azaperone combinations (Table 2).

The administration of the relatively lower dosages of the metomidate/azaperone combination resulted in an average increase in cloacal temperature of 0,6°C whereas the higher dosages caused an average drop in cloacal temperature of 0,8°C after 75 min. Minimal changes in cloacal temperatures were observed with administration of the other drugs.

All birds injected with the tiletamine-/zolazepam combination, metomidate or with the metomidate/azaperone combinations were immobilised. Immobilisation was often preceded by forward and backward staggering until the birds collapsed in either sternal or lateral recumbency. Birds injected with metomidate, showed a progressive limp in the leg in which they were injected before going down. In chicks immobilised with the different dosages of the tiletamine/zolazepam combination, metomidate and with the metomidate/azaperone combination (10 and 3,3 mg kg^{-1}), intermittent kicking movements, intermittent head and neck movements, occasional yawning and, in some birds, regurgitation of a greenish fluid were observed. Body, leg and neck movements were precipitated by handling, by insertion of a thermometer into the cloacae and by testing pedal reflexes. Pedal reflexes remained intact.

The adult ostrich immobilised with the tiletamine/zolezepam mixture displayed violent kicking movements and flung its head, neck and body around. This resulted in a serious damage to both eyes of the ostrich.

The ostriches immobilised with the metomidate/azaperone imxture (20 and 6,6 mg kg^{-1}) remained very still throughout the period of immobilisation. Salivation and regurgitation were observed in 4 of the birds. Pedal reflexes disappeared and were only elicited 75-90 min after administration of the drug. Profound respiratory suppression occurred in 2 birds; one bird became completely apnoeic and in another, the respiratory rate dropped to 2 cycles per min. Rhythmic chest compression and the intravenous administration of 10 mg of doxapram hydrochloride (Dopram, Continental Ethicals) resulted in adequate ventilation of the bird. All birds made very smooth recoveries.

Droopings of the wings, mild drowsiness, occasional drooping of the head and slight ataxia were observed in the birds treated with medetomidine. The birds were not immobilised. All showed a drop in heart rate.

Increased dosages of the tiletamine/zolezepam mixture and the metomidate/azaperone combination were significantly positively correlated with time immobilised (r = 0,77; r = 0,94). The correlation between drug dosage and time to immobilisation was statistically insignificant.

DISCUSSION

Tiletamine in combination with zolazepam, metomidate and metomidate in combination with azaperone successfully immobilised ostrich chicks. Although the birds were immobilised, full anaesthesia and analgesia were apparently achieved with the metomidate/azaperone combination only. Apart from fluid regurgitation in some birds, increased heart rates with higher dosages of drugs and respiratory

Table 2: **Average respiratory and heart rates of ostrich chicks at times after the administration of different dosages of a tiletamine/zolazepam mixture, metomidate, and a metomidate/azaperone combination**

No of ostriches	Drugs and dosage rate; average respiratory and heart rates	Time in min after administration of drug						
		5	10	15	30	45	60	75
5	Tiletamine/zolazepam; 5 mg kg^{-1}							
	respiratory rate	26	22	22	21	-	-	-
	heart rate	131	117	121	102	-	-	-
5	Tiletamine/zolezepam; 10 mg kg^{-1}							
	respiratory rate	23	21	26	22	22	21	-
	heart rate	125	122	131	110	110	92	-
1	Tiletamine/zolezempam; 15 mg kg^{-1}							
	respiratory rate	26	20	20	16	16	12	-
	heart rate	132	112	120	126	100	88	-
4	Tiletamine/zolazepam; 20 mg kg^{-1}							
	respiratory rate	40	46	43	41	31	25	-
	heart rate	186	153	140	125	111	101	-
5	*Metomidate; 15 mg* kg^{-1}							
	respiratory rate	-	25	21	31	115	-	-
	heart rate	-	111	133	108	115	-	-
5	Metomidate; 20 mg kg^{-1}							
	respiratory rate	-	33	20	43	56	-	-
	heart rate	-	166	166	147	130	-	-
5	Metomidate/azaperone; 10 and 3,3 mg kg^{-1}							
	respiratory rate	17	14	16	16	18	-	-
	heart rate	184	186	171	144	126	-	-
5	Metomidate/azaperone; 20 and 6,6 mg kg^{-1}							
	respiratory rate	16	24	19	21	28	24	16
	heart rate	193	196	192	179	185	160	145

- respiratory and heart rate not taken

suppression in 2 birds, no other untoward clinical side-effects were observed in these ostrich chicks.

The tiletamine/zolezepam combination, however, resulted in unsatisfactory immobilisation (associated with serious self-traumatisation) of the adult ostrich and should thus possibly not be used for immobilisation of free-ranging ostriches. This should also caution against the extrapolation of our relatively favourable findings in chicks to mature ostriches.

The metomidate/azaperone mixture resulted in smooth induction, maintenance and recovery from the immobilised state. Birds appeared to have been fully anaesthetised. A dosage of approximately 15 mg kg^{-1} metodomidate and 4 mg kg^{-1} azaperone should be considered for the immobilisation of ostriches in the field. Further investigation into the use of this drug combination under field conditions, should be undertaken.

The administration of medetomidine did not result in immobilisation of ostrich chicks. Medetomidine, primarily an anxiolytic drug, should therefore probably be used in combination with other immobilising agents such as ketamine hydrochloride. This would be in agreement with its reported use in other species[2].

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Book review/Boekresensie

DIAGNOSTIC PARASITOLOGY FOR VETERINARY TECHNICIANS

EDITOR: JOANN COLVILLE

American Veterinary Publications, Inc., 5782 Thornwood Drive, Goleta, California 93117, USA. 1991 pp 266, paperback binding, numerous photographs, line drawings and diagrams, price US $24.50 plus US $4.00 shipping, (ISBN 939674-32-7).

This neatly produced book will be of great value to practices and diagnostic laboratories.

In the short introductory chapter the various groups of parasitic organisms are described. This is followed by a comprehensive chapter on common procedures for diagnosing ectoparasitic, helminthic and protozoal infections. Specific procedures, which are described in detail, are presented in blocked format for easy reference.

The remainder of the book is taken up by chapters on parasites of dogs and cats, horses, food animals, rabbits and rodents, and pet and aviary birds. The latter 2 chapters are especially helpful, as this information is not readily available.

The line drawings and most of the photographs are excellent aids in identification of the parasites. Some photographs, eg. *Onchocerca* microfilariae (p 133) and a *Giardia* trophozoite (p 250) are indistinct and could have been omitted altogether. The severe cropping, in which parts of some of the photographed specimens are lost, is irritating.

Although the index is fairly complete, there are some glaring omissions. Although the preparation of Lugol's Iodine Solution warrants its own block (p 27), it is not indexed. The hosts are not indexed as such.

This manual is obviously aimed at the American market. A South African technician or veterinarian will search in vain for *Cowdria* or *Encephalitozoon,* for example.

On the whole, this is an excellent laboratory manual which should enjoy steady sales.

B.L. Penzhorn

Book review/Boekresensie

DOGS & CATS — A HEALTH GUIDE
HONDE & KATTE — 'N GESONDHEIDSGIDS

JOHANNES ODENDAAL

1st edn. Tafelberg Publishers LTD, 28 Wale Street, Cape Town 8001 1990 pp 136, illustrations 60 Price R19-95 (ISBN 0624030024).

This book (available in English and Afrikaans), should be a useful source of reference for the concerned pet owner. When I first read the book I was very critical. However, having tried to review the book from a layman's point of view, I believe the information it contains will be a useful guide to pet owners in South Africa. The book contains a number of inaccuracies and is ambiguous at times, but I believe it will achieve its objective. The text is divided into 3 parts. Part 1 deals with basic information on dogs and cats, part 2 deals with conditions of the body systems and organs, while part 3 deals with a few miscellaneous conditions affecting dogs and cats. Over 200 conditions affecting dogs and cats are discussed.

G.N. Eckersley

THE EFFECT OF PREMEDICATION ON THE INDUCTION DOSE OF PROPOFOL IN DOGS AND CATS

JUDITH K GEEL*

ABSTRACT

The effect of premedication on the induction dose of propofol was determined in 15 cats and 25 dogs undergoing elective surgical procedures. The induction dose of propofol in dogs younger than 8 years old was 6,9 ± 0,9 mg kg^{-1} (n = 4) without premedication and 4,3 ± 1,4 mg kg^{-1} (n = 12) with premedication with acetylpromazine maleate. The induction dose in cats younger than 3 years old was 7,8 ± 1,1 mg kg^{-1} (n = 8) with atropine alone and 7,1 ± 0,9 mg kg^{-1} (n = 7) with the inclusion of acetylpromazine maleate. The reduction in the induction dose of propofol was statistically significant in dogs, but not in cats. When atropine was used together with a fentanyl-droperidol combination or pethidine and acetylpromazine maleate in dogs, the mean induction dose of propofol was reduced to 2,1 ± 0,1 mg kg^{-1} (n = 4) and 2,4 ± 0,3 mg kg^{-1} (n = 5), respectively. Propofol was also evaluated as an induction agent in patients undergoing non-elective surgical procedures.

Key words: Propofol, dogs, cats, premedication, dose, induction, anaesthesia

Geel J.K. **The effect of premedication on the induction dose of propofol in dogs and cats.** *Journal of the South African Veterinary Association* (1991) 62 No. 3, 118-123 (En.) Department of Experimental and Clinical Pharmacology, University of the Witwatersrand Medical School, 7 York Road, 2193 Parktown, Republic of South Africa.

INTRODUCTION

Propofol [Diprivan Injection 10 mg ml^{-1}, Stuart Pharmaceuticals, (South Africa) (Pty) Limited] is an alkyl phenol (2,6 diisopropylphenol), which is marketed as a white sterile oil-in-water emulsion. It has anaesthetic properties following intravenous administration and is currently registered for use in man. The original solvent, Cremophor-EL (polyoxyethylated castor oil), caused a release of histamine in dogs which prevented its use as an anaesthetic agent in this species[19]. It was also believed to be associated with a significant incidence of anaphylactoid reactions in man[2 3 10 15]. The current formulation is a soya-bean emulsion and exhibits some loss of potency in man when compared to the Cremophor-EL formulation[16 19]. Although the frequent occurrence of a cutaneous flush was noted with the use of the emulsion formulation for the induction of anaesthesia in man, no tendency to develop anaphylactoid reactions was observed, as determined by measurement of immunoglobulin levels, complement C3 levels and plasma histamine concentrations[3]. No allergic-type reactions have been recorded in dogs or cats with the emulsion formulation[1 6 15].

In man, propofol causes a rapid induction of anaesthesia and is extensively distributed from the blood into the tissues following intravenous administration[16]. Metabolism is rapid and occurs via hepatic and extrahepatic mechanisms with inactive metabolites excreted by the kidney[15 16]. It lacks cumulative properties[15]. Recovery from anaesthesia is smooth and rapid and is associated with minimal postoperative confusion[15]. Thus, propofol is used in man as an alternative to methohexitone for the maintenance of general anaesthesia during brief outpatient procedures[4].

The potential of propofol for use as an anaesthetic in veterinary science was recognised from clinical trials performed on experimental animals during the development of propofol for use in man[6]. Its use in animals was associated with a rapid smooth induction which was of short duration and a smooth recovery[6 15 19].

Induction doses of propofol for dogs and cats with and without premedication, using acetylpromazine maleate, have been reported[1 12 19]. The effect of tranquillising premedication on the induction dose for cats, produced conflicting results[1 12]. One study examined the effect of tranquillising premedication on the induction dose in dogs and cats, but did not appear to differentiate between the different types of tranquillising premedication when the induction doses were statistically analysed[12]. The induction dose of propofol for dogs and cats, when an opioid was used in the premedication, had not been reported. This has been shown to decrease the induction dose of propofol in man[4].

The purpose of this investigation was to establish if the induction dose of propofol for dogs and cats was affected by the different types of routine premedication drugs used in the small animal section of the Department of Surgery, Faculty of Veterinary Science, University of Pretoria. Propofol was also assessed as an induction agent in patients considered to be anaesthetic risks.

MATERIALS AND METHODS

Propofol was used as an intravenous induction agent in cats (n = 15) and dogs (n = 25) undergoing elective surgical procedures and assigned an American Society of Anaesthesiologists (ASA) status of one[5]. The decision of whether to give premedication and the type of premedication was determined on a random basis. If premedication was given prior to induction, it was administered as outlined in Tables 1 & 2.

Propofol was also used as an intravenous induction agent in cats (n = 7) and dogs (n = 18) that were to undergo non-elective surgical procedures and that had been assigned an ASA status greater than one. These cases either received no premedication or premedication was given as determined by the condition of the animal and the surgical procedure to be performed (Tables 3 & 4).

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An over-the-needle Teflon catheter (Jelco, Critikon) was inserted, using an aseptic technique, in the cephalic vein of the animal prior to the induction of anaesthesia. The amount of propofol drawn up in the syringe was based on an approximate dose of 7,0 mg kg^{-1} for dogs and 8,0 mg kg^{-1} for cats which had previously been established for unpremedicated patients[12]. The initial amount of propofol in the syringe was recorded. A bolus intravenous injection was given intravenously at 2,0 mg kg^{-1} with incremental doses titrated within 15 to 20 sec to the stage where there was sufficient relaxation of the jaw and suppression of protective laryngeal reflexes to allow endotracheal intubation. The amount of propofol not used was noted and the total dose of propofol used was recorded.

Lignocaine [Xylocaine, Astra (Keatings)] was sprayed onto the larynx of cats as a routine procedure prior to intubation. Anaesthesia was maintained with halothane (Fluothane, ICI) in all cases except dental procedures and thoracotomies. In these cases, enflurane (Ethrane, Abbott) and isoflurane (Forane, Abbott) were used, respectively.

Post intubation, the patient was connected to an inhalation anaesthetic system, and the vaporiser setting was increased in increments of 0,5% with every

Table 1: **Induction dose of propofol in dogs younger than 8 years undergoing elective surgical procedures (ASA status I)**

Premedication (*)	Species	Surgical procedure	Age	Sex	Body mass (kg)	Dose (mg kg^{-1})	Mean induction dose (mg kg^{-1})
No premedication	Maltese	ovariohysterectomy	7 months	F	3,75	8,0	6,9 ± 0,9
	Pug	nictitating membrane flap (corneal ulcer)	2 years	M	7,10	6,3	
	GSD	ovariohysterectomy	young adult	F	22,00	5,9	
	GSD	ovariohysterectomy	young adult	F	27,50	7,2	
Acetylpromazine maleate (1) 0,10 mg kg^{-1} SC	ST	ovariohysterectomy	2,3 years	F	11,00	3,6	4,3 ± 1,3
	GSD	ovariohysterectomy	young adult	F	20,00	3,3	
	GSD	ovariohysterectomy	young adult	F	17,50	2,6	
	Mongrel	ovariohysterectomy	1,5 years	F	5,50	3,6	
	Mongrel	ovariohysterectomy	6 months	F	5,00	5,2	
	Mongrel	ovariohysterectomy	1,5 years	F	6,40	2,0	
	Rottweiler	caudectomy	3 months	F	10,50	4,8	
	Bullterrier	ovariohysterectomy	6 months	F	18,00	5,1	
	Dachshund	ovariohysterectomy	8 months	F	6,80	6,8	
	BT	ovariohysterectomy	11 months	F	8,70	4,6	
	Fox terrier	orchidectomy	1,3 years	M	12,00	5,8	
	Poodle	ovariohysterectomy	1 year	F	4,75	4,1	
Acetylpromazine maleate (0,05 mg kg^{-1} SC); Atropine (2) 0,05 mg kg^{-1} SC and Pethidine (3) 2,00 mg kg^{-1} IM	Maltese	front leg amputation (radial paralysis)	3 years	M	10,50	2,9	2,4 ± 0,3
	Bulldog	congenital elbow luxation repair	2 months	M	6,50	2,2	
	Chihauhua	orchidectomy	1 year	M	1,60	2,4	
	GSD	vasectomy	young adult	M	31,50	2,3	
	Labrador	stifle arthrotomy	7 years	?	36,00	2,1	
Fentanyl 0,4 mg ml^{-1} droperidol 20 mg ml^{-1} (4) IV (**) 1 ml 60 kg^{-1} and atropine 0,05 mg kg^{-1} SC	GSD	vasectomy	young adult	M	32,50	2,0	2,1 ± 0,1
	GSD	ovariohysterectomy	young adult	F	28,06	2,1	
	GSD	vasectomy	young adult	M	35,00	2,0	
	GSD	ovariohysterectomy	young adult	F	28,50	2,3	

(*) Given one hour before induction unless otherwise indicated
(**) Given 15 min prior to induction

GSD = German Shepherd dog ST = Staffordshire terrier BT = Boston terrier
? = Unknown M = male F = female
IM = intramuscular SC = subcutaneous IV = intravenous

(1) ACP 2 mg ml^{-1}, Centaur
(2) Atropine 0,5 mg ml^{-1}, Centaur
(3) Pethidine 50 mg ml^{-1}, Centaur
(4) Innovar vet, Jansen Pharmaceutica

3 or 4 breaths of the animal. The patient was initially maintained on a halothane concentration of 3,0% on a closed circuit system or 2,5% on a semi-open system. Once the animal was stabilised, a vaporiser setting of 2,0% for a closed circuit system and 1,5% for a semi-open system on halothane was used.

The patient was placed in a recovery room after the surgical procedure and monitored during recovery. Recovery times were not recorded.

The reduction in the induction dose with the use of premedication in dogs and cats undergoing elective surgical procedures was analysed, using the Student's *t* test with $p < 0,05$ taken as the minimal level of statistical significance. The doses of propofol given in animals undergoing non-elective surgical procedures were not analysed statistically.

RESULTS

The induction doses which would allow endotracheal intubation for animals undergoing elective procedures, using no premedication and different types of premedication, are shown in Tables 1 & 2. There was a significant reduction ($p < 0,05$) in the mean induction dose of propofol with the use of premedication in dogs. When acetylpromazine maleate (ACP 2 mg ml^{-1}, Centaur) was used, there was a 38% reduction in the mean induction dose of propofol of dogs. When an opioid was included in the premedication in dogs, there was more than a 60% reduction in the mean induction dose of propofol. Use of acetylpromazine maleate in cats however, did not cause a significant reduction ($p > 0,05$) in the mean induction dose of propofol.

Induction doses of propofol used in dogs and cats undergoing non-elective surgical procedures are shown in Tables 3 & 4. The induction dose was administered too slowly in 2 dogs undergoing non-elective surgical procedures (Table 3). This resulted in further incremental doses of propofol being given before endotracheal intubation could be achieved.

Transient apnoea accompanied by a slightly blue discolouration of the tongue was occasionally seen at the time of endotracheal intubation in cats undergoing elective surgical procedures. Respiration returned spontaneously in each case.

The induction of anaesthesia in the English Bulldog (Table 1) with propofol was smooth and intubation was easily achieved. The geriatric patient undergoing a short dental procedure (Table 3) recovered from the anaesthetic with a return to an alert habitus within a short period of time.

DISCUSSION

This study found that the mean induction dose of propofol in dogs was significantly reduced ($p < 0,05$) when premedication was used. The affect of acetylpromazine maleate on the induction dose of propofol in dogs was found to be similar to that of other work which reported a mean induction dose of $5,95 \pm 1,86$ mg kg^{-1} in unpremedicated dogs and $3,81 \pm 2,07$ mg kg^{-1} in dogs given acetylpromazine maleate[19]. Inclusion of an opioid in the premedication in dogs undergoing elective surgery, reduced the mean induction dose by over 60%.

The inclusion of acetylpromazine maleate in the premedication of cats, was not shown to significantly affect ($p > 0,05$) the induction dose of propofol. This supported the results of another study which established that the mean induction dose of 6,8 mg kg^{-1} in cats was not significantly affected by prior administration of acetylpromazine maleate[1].

The effect of atropine alone on the in-

Table 2: **Induction dose of propofol for cats younger than 3 years undergoing elective surgical procedures (ASA status I)**

Premedication (*)	Surgical procedure	Age (months)	Sex	Body weight (kg)	Dose (mg kg^{-1})	Mean induction dose (mg kg^{-1})
	orchidectomy	8	M	3,2	6,3	
	ovariohysterectomy	6	F	3,3	8,8	
	ovariohysterectomy	young adult	F	3,1	7,7	
Atropine (1)	ovariohysterectomy	36	F	2,5	6,8	$7,8 \pm 1,1$
0,05 mg kg^{-1}	dental procedure	24	?	3,2	9,4	
	orchidectomy	12	M	3,8	7,9	
	ovariohysterectomy	8	F	2,5	7,0	
	orchidectomy	18	M	4,1	8,8	
Acetylpromazine	orchidectomy	9	M	4,0	7,5	
maleate (2)	ovariohysterectomy	9	F	3,0	6,2	
0,10 mg kg^{-1}	ovariohysterectomy	18	F	2,8	6,1	
and	orchidectomy	24	M	4,8	6,9	
atropine	ovariohysterectomy	young adult	F	3,2	8,8	$7,1 \pm 0,9$
0,05 mg kg^{-1}	orchidectomy	9	M	3,5	6,7	
	ovariohysterectomy	12	F	3,3	7,3	

(*) Given one hour prior to anaesthesia by subcutaneous injection
M = male F = female ? = unknown

(1)Atropine 0,5 mg ml^{-1}, Centaur (2)ACP 2 mg ml^{-1}, Centaur

Table 3: **Induction doses of propofol for dogs undergoing non-elective surgical procedures**

Premedication (*)	Species	ASA status	Surgical procedure	Age	Sex	Body mass (kg)	Dose (mg kg^{-1})
	Rottweiler	III	diaphragmatic hernia repair	3 months	F	7,5	5,2
No premedication	Boxer	III	laparotomy	5 months	M	14,0	2,9
	Mongrel	III	diaphragmatic hernia repair	Adult	?	4,5	3,6
	Poodle	III	dental scaling and tooth extraction	19 years	M	6,6	4,6
	Maltese	II	laparotomy	2 years	M	4,0	(+)10,0
Acetylpromazine maleate (1)	Mongrel	II	fracture femur/open reduction	4 months	M	2,5	4,8
0,10 mg kg^{-1} SC	Chow	II	rectopexy	8 weeks	F	3,2	3,8
	Scottish terrier	II	laparotomy	5 years	M	14,0	2,0
Acetylpromazine maleate	Staffordshire terrier	II	thoracotomy	3 years	F	10,0	3,0
0,05 mg kg^{-1} SC;	Maltese	II	laminectomy	4 years	F	4,5	2,0
	Pug	III	femur luxation/open reduction	4 years	M	8,9	4,3
Atropine (2) 0,05 mg kg^{-1} SC and	Mongrel	II	slot decompression	12 years	M	10,0	(+)6,0
	Maltese	II	laminectomy	3 years	F	3,9	4,1
pethidine (3) 2,00 mg kg^{-1} IM	Staffordshire terrier	II	ulna fracture/open reduction	Adult	?	21,5	1,4
Fentanyl 0,4 mg ml^{-1} droperidol 20 mg kg^{-1} (4) 1 ml 60 kg^{-1} IV (**) and atropine 0,05 mg kg^{-1} SC	Maltese	IV	perineal herniorrhaphy	10 years	M	4,7	4,3
	Pomeranian	IV	inguinal herniorrhaphy	14 years	M	3,4	1,2
	Bull Mastiff	II	humeral fracture/open reduction	Adult	M	31,4	3,2
	Red Setter	III	patent ductus arteriosus ligation	12 weeks	F	11,5	2,5

(*) Given one hour prior to induction of anaesthesia unless otherwise indicated
(**) Given 15 min prior to induction of anaesthesia
(+) Drug administered too slowly

SC = subcutaneous IM = intramuscular IV = intravenous
M = male F = female ? = unknown

(1) ACP 2 mg ml^{-1}, Centaur (2) Atropine 0,5 mg ml^{-1}, Centaur
(3) Pethidine 50 mg ml^{-1}, Centaur (4) Innovar vet, Jansen Pharmaceutica

duction dose of propofol was not evaluated in this study. Atropine has not been found to significantly affect the induction dose in dogs[19]. However, it may cause a prolonged recovery in cats[12]. Other factors, not evaluated in this study, that could affect the induction dose of propofol, include the effect of sex and age on induction dose. Male dogs have been found to require slightly larger induction doses than females[12 19]. In man, elderly patients have been shown to require a lower induction dose of propofol than young patients[4 11 13 15].

The incidence of apnoea after induction, was not monitored in this study. However, propofol is a profound respiratory depressant and a high incidence of apnoea post administration of propofol is reported in man[14 15]. It has been found to cause a significant decrease in minute volume immediately after administration and in patients receiving an opioid premedication[15]. Thiopentone and propofol were not shown to differ significantly in the percentage of patients experiencing apnoea post induction[15]. A similar clinical impression was obtained on the incidence of apnoea in dogs induced with propofol when compared with the induction of anaesthesia using thiopentone and methohexitone[19]. However, the low blood oxygen tensions, found in animals anaesthetised with propofol and breathing air, indicate the necessity of the routine use of endotracheal intubation and the possible need to support respiration with intermittent positive pressure ventilation[19].

The speed of administration of propofol in this study affected the dose requirement and rapidity of onset of anaesthesia in dogs. Propofol was administered slowly, over approximately 60 sec, on 2 occasions (Table 3) which resulted in larger doses being used and a longer time before endotracheal intubation could be achieved. In man, the administration of a 2 mg kg^{-1} induction dose of propofol is reported to produce a satisfactory level of anaesthesia more

rapidly and more reliably if injected over 5 sec than over 60 sec[14 15]. The speed of injection has not been found to affect the incidence or duration of apnoea or the degree to which blood pressure dropped[15]. Transient apnoea with cyanosis was occasionally seen on endotracheal intubation of cats undergoing elective surgical procedures in this study. This was not considered to be associated with too rapid an administration of propofol. Respiration returned spontaneously in each case.

Induction of anaesthesia with propofol in man, causes a decrease in arterial pressure which is more severe in the elderly[4 15 18]. The cardiovascular depression obtained with propofol is greater than that obtained with thiopentone and methohexitone and may be dose-related[4 14 18]. As the diastolic pressure falls to a proportionally greater degree than the systolic pressure, it was suggested that there may be a decrease in peripheral resistance[15]. Recent work has shown that propofol may have a direct venodilatory effect, causing a reduction in preload with a secondary decrease in cardiac output[7]. If preload is maintained, cardiac output and arterial pressures are well preserved at normal anaesthetic blood concentrations[7].

It must still be established whether the reduced induction dose of propofol in dogs given premedication is associated with less cardiovascular and respiratory depressant effects.

Thiopentone is still the most popular, cheap and widely used intravenous anaesthetic for dogs. It is often used as the sole anaesthetic agent for short minor surgical procedures. The solution for injection is alkaline and can produce tissue necrosis if injected perivascularly, and arterial spasm if injected into an artery[8]. It has a short induction time and has anti-convulsant properties following anaesthesia[6 8 15]. It is not rapidly metabolised and has a cumulative effect which can lead to prolonged anaesthesia and unconsciousness[8]. This can be a serious hazard postoperatively[8]. Propofol also has a short induction time but lacks anti-convulsant properties[6 15]. No tissue damage occurs after perivascular or intra-arterial injection[6]. One of the desirable characteristics of propofol is its lack of cumulative properties when compared with thiopentone[12]. This allows it to be used for continuous intravenous infusion for maintenance of anaesthesia[1 4 10 15 17 19]. Emergence from anaesthesia is rapid with a rapid return to an alert habitus[15]. However, one study on dogs premedicated with acetylpromazine maleate and atropine and induced and maintained on propofol, found that although cardiovascular and respiratory effects were similar to those in dogs anaesthetised with halothane and nitrous oxide, maintenance on propofol was associated with a higher incidence of vomiting in the recovery period[9].

An initially higher halothane concentration was used after induction with propofol than was normally used after induction with thiopentone. This was found to be necessary to prevent lightening of anaesthesia between induction and sta-

Table 4: **Induction dose of propofol in cats undergoing non-elective surgical procedures**

Premedication (*)	ASA status	Surgical procedure	Age	Sex	Body mass (kg)	Dose (mg kg^{-1})
	III	Diaphragmatic hernia repair	5 months	M	3,4	5,9
Atropine (1) SC 0,05 mg kg^{-1}	III	Pelvic fracture/open reduction	2 years	F	2,6	6,5
	III	Penile urethrostomy	Adult	M	5,0	3,2
Acetylpromazine maleate (2) 0,10 mg kg^{-1} SC and atropine 0,05 mg kg^{-1} SC	II	Bite wounds	8 months	?	3,5	7,7
	I	Pinnectomy-squamous cell carcinoma	8 years	M	3,5	6,7
Acetylpromazine maleate 0,05 mg kg^{-1} SC;	II	Femur fracture/open reduction	5 months	M	2,7	4,8
Pethidine (3) 1,00 mg kg^{-1} IM and atropine 0,05 mg kg^{-1} SC	II	Maxilla fracture/open reduction	3 years	F	2,0	6,0

(*)Given one hour prior to anaesthesia
(1)Atropine (0,5 mg ml^{-1}, Centaur) (2)ACP (2 mg ml^{-1}, Centaur) (3)Pethidine (50 mg ml^{-1}, Centaur)
SC = subcutaneous IM = intramuscular
M = male F = female ? = unknown

bilisation with halothane.

As it causes a rapid, smooth induction and is rapidly metabolised, propofol may be a useful induction agent in the anaesthetic management of diaphragmatic hernias, where rapid access to the airway is needed, and in the anaesthetic management of the English Bulldog. In the latter case, a rapid access to the airway and a rapid recovery with early return of protective reflexes is desirable as a result of this breed's potential to develop an upper airway obstruction syndrome.

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Book review/Boekresensie

EQUINE PRACTICE THE "IN PRACTICE" HANDBOOKS

E BODEN (EDITOR)

1st Edition. Bailliere Tindall, W.B. Saunders, 24-28 Oval Road, London, NW1 7DX, 1991. pp X and 300, 34 tables, 21 diagrams and 149 plates.

The compilation of articles in this book offers an update on a variety of topics in equine medicine, surgery and theriogenology.

While many of the articles are descriptive in nature, they introduce the reader to valuable diagnostic aids such as palpebral nerve blocks for the examination of the blepharospastic eye.

In those articles where surgical techniques are outlined, the step-by-step description is coupled to good quality plates and diagrams which capably qualify the technique under discussion.

A major proportion of the book is reserved for the different aspects of forelimb lameness and the author provides a concise summary of the approaches to lameness diagnosis and treatment. Where possible, different conditions are illustrated by good quality radiographs and the anatomical landmarks associated with the different nerve blocks receive appropriate attention.

The section dealing with cardiology, highlights the general theme of the book, in that a sound clinical evaluation will remain invaluable for a correct diagnosis, and while ECG tracings would have been a valuable adjunct to the conditions described, not all practitioners have access to such facilities and hence have to rely on their own skills to formulate the correct diagnosis and an appropriate treatment strategy.

For the above reason in itself, the book is to be recommended for its review of established procedures and as an insight into current developments in the various fields. Both potential and current equine practitioners will benefit from this publication.

R.D. Kirkpatrick

LACK OF ANTIBODIES TO CORONAVIRUSES IN A CAPTIVE CHEETAH *(ACINONYX JUBATUS)* POPULATION

JENNIFER A SPENCER*

ABSTRACT

Cheetahs *(Acinonyx jubatus)* (n=40) were tested by means of an immunofluorescent test (IFT) for the presence of antibodies to the feline coronavirus group. All cheetahs tested negatively and this was further confirmed by virus serum neutralisation.

Key words: Cheetah, *Acinonyx jubatus*, coronavirus, immunofluorescent test.

Spencer J.A. **Lack of antibodies to coronaviruses in a captive cheetah *(Acinonyx jubatus)* population.** *Journal of the South African Veterinary Association* (1991) 62 No. 3, 124-125 En.) Department of Infectious Diseases, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, 0110 Onderstepoort, Republic of South Africa.

Feline infectious peritonitis (FIP) is a sporadic, highly lethal coronaviral disease of Felidae[10]. Recent sero-epidemiological surveys have demonstrated that infection with feline coronaviruses is prevalent in 20 to 30% of domestic cats in the general population worldwide, and in multi-cat households, this rate may even be as high as 100%[10]. The disease has been recognised in Europe, North America, Australia, Japan and South Africa. The natural host range of FIP virus (FIPV) includes domestic and non-domestic felids such as cheetah *(Acinonyx jubatus)*, lion *(Panthera leo)*, serval *(Felis serval)*, caracal *(Felis caracal)* and leopard *(Panthera pardus)*[1 2 3 8 9].

The relatively low numbers of cheetah in the wild[5] has led to the establishment of various captive breeding programmes where the animals may come into contact with feral cats. This, as well as the fact that cheetahs have been proposed to lack genetic variability, make them highly susceptible to infectious diseases[6]. Subclinical coronavirus infection[4] as well as epidemics of FIP[3 8] have been reported in captive cheetah populations. It is important to monitor the immune status of the cheetah, as well as to be in a position to certify a group of cheetahs free of coronaviral infection, as this may have important management implications. For this purpose, a rapid serological test was developed for the detection of antibodies to the coronaviruses in cheetahs kept at the De Wildt Cheetah Breeding Centre of the National Zoological Gardens of South Africa at De Wildt.

Forty cheetahs which were housed in small groups in wire enclosures were investigated. They were separated from other species at the Centre by these fences, but the camps were accessible to feral cats. The age range of the cheetahs varied from a few months to 14 years. There has not been any evidence of clinical FIP in this group to date.

Two samples of 10 ml of blood were drawn into plain tubes from the medial saphenous vein, one month apart, from each cheetah during 1989 (total of 80 samples). The blood samples were then centrifuged and the serum drawn off and frozen until tested.

An indirect IFT, using foetal cat whole foetus (FCWF) cells (obtained from Dr Pedersen, UC, Davis, in 1984), infected with a field isolate of FIP virus (strain CAC 230 obtained from Wellcome Research Laboratories, Kent, England), fixed onto multi-well test slides, was used to detect the presence of antibodies in the serum samples of the cheetahs. Monolayers of virus-infected cells were harvested by trypsinisation when about 40% of the cells showed cytopathic changes. The cells were washed 3 times with phosphate-buffered saline (PBS), mixed with equal amounts of uninfected cells and air dried onto the slides (3×10^3 cells per well). The slides were fixed in chilled acetone for 10 min, dried and stored at -20°C until used. The cells were covered with 20 µl volumes of a 1:10 dilution of either test serum or positive control. The positive control consisted of peritoneal fluid and serum obtained from a clinical case, and having a fluorescent response of 3+ at 1:400 dilution. The slides were incubated for one hour at 37°C in a humid chamber and subsequently washed 3 times in PBS and once in distilled water. After air-drying, 20 µl volumes of a 1:50 dilution of fluorescein isothiocyanate (FITC) labelled rabbit anti-cat IgG (Zymed laboratories, San Francisco, CA) were applied to the cells. The slides were incubated, washed and dried as before and mounted in buffered glycerol (pH 7,8). The cells were examined for specific reactivity using a fluorescence microscope.

FCWF cells (0,2 ml) were seeded in 96 well microtitre plates. When the cells were confluent, the medium was removed and replaced with 0,1 ml fresh medium, 50 µl of virus and 50 µl of a 1:5 dilution of test serum or positive control. The virus was seeded in 6 dilutions: 10^{-2} to 10^{-7}. The plates were examined daily for cytopathic effect (CPE).

None of the 80 samples tested showed specific reactivity when examined with the fluorescence microscope. The positive control, on the other hand, showed strong fluorescence when examined at the same dilution. This absence of specific fluorescence, indicated a lack of antibodies to the feline coronaviruses, including FIP, in this cheetah group. This was further confirmed by virus neutralisation, as, by Day 1 there was a 4+ CPE in all the samples, showing a lack of antibodies in the sera.

Reports of FIP disease in cheetahs have been scarce and clinical outbreaks have rarely been reported. Colly[2] reported a loss of 2 adult cheetahs on their intro-

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duction to the Johannesburg zoo. Evermann et al.,[3] and Pfeifer[8] reported on an outbreak in Oregon in which serious losses occurred. However, the presence of coronavirus antibodies with no clinical FIP disease, was reported in a group of wild-caught cheetahs by Horzinek & Osterhaus[4].

No antibodies to coronaviruses could be detected in the study group. These animals therefore constitute a FIP-free population. This has important implications; firstly they serve as a population from which animals can safely be introduced into established breeding groups and, secondly, they constitute a highly susceptible population which emphasises the need for screening of all newcomers to the group.

Feline infectious peritonitis is an immune mediated disease[7]. It is therefore largely dependent on the state of the cell mediated immunity (CMI) of the animal.

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Book review/Boekresensie

ATLAS OF DIAGNOSTIC RADIOLOGY OF EXOTIC PETS

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1st Edn. Schluetersche Wolfe, Hannover and W.B. Saunders, Philadelphia. 1991, pp 224, 328 radiographs and 51 line drawings. ISBN (0-7234-1642-7).

The editors of the atlas selected a representative international team of clinicians and radiologists, specialists on diseases and breeding of exotic and domestic pet animals. The objective of the volume was to fill the gap in knowledge on radiological interpretation of a large variety of small to medium-sized mammals, birds, reptiles and amphibians. The book is primarily destined to meet the needs of veterinary practitioners.

A short, well structured chapter describes radiographic techniques relevant to each group of animals. Methods of physical and chemical restraint are briefly described, as well as the standard radiographic positioning.

Normal anatomy is demonstrated on several radiographs in each species. The accompanying texts depict the unique anatomic features of single species of their groups, such as the os penis or the intra-abdominal position of the testicles in the male guinea pig. These data are interesting from the point of view of the comparative anatomy and indispensable for the proper radiologic interpretation of radiographs.

The following chapter covers abnormalities. As in the previous section, descriptions of the excellent radiographs are concise and accurate. Line drawings are used effectively to augment some radiographs by outlining important landmarks. Some abnormalities are indicated on radiographs with the help of arrow markers. Only certain diseases/abnormalities are presented. However, the selection is sufficiently representative to cover the more common conditions.

The book is well laid out and easy to read. It indicates the diagnostic value of radiology in exotic and domestic pets, for conditions in which the clinical and many paraclinical methods of investigation could be risky to the patient and non-rewarding for the clinician. The atlas can be recommended to veterinary practitioners dealing with these particular species and for veterinarians in zoological gardens as well as to animal rescue and breeding centres. Research workers in the laboratory animal sciences, comparative anatomy and physiology will also find this atlas highly interesting.

J. Still

THE RESPONSE OF ANIMALS TO SUXAMETHONIUM (SUCCINYLDICHOLINE) AND SUCCINYLMONOCHOLINE

J HATTINGH*, N I PITTS*, V DE VOS**, D G MOYES* and M F GANHAO*

ABSTRACT

The time which elapses before cessation of breathing, and blood pressure and blood gas changes after the intramuscular administration of suxamethonium, or a mixture of suxamethonium and hexamethonium, is compared in immobilised African elephants *(Loxodonta africana)* and buffaloes *(Syncerus caffer)*. In addition, the respiratory responses of elephants and other animals to intravenous administration of suxamethonium and succinylmonocholine are reported on, as are the effects of darting animals with succinylmonocholine. The results show that respiration is affected in a similar fashion in all species investigated. However, the characteristic gradual decrease in respiratory rate seen in elephants during culling, using suxamethonium, resembles the effects observed when succinylmonocholine is administered. It is suggested that elephants are killed by this first breakdown product of suxamethonium during culling and/or that unique acetylcholine receptors may be involved.

Key words: Elephants, *Loxodonta africana*, culling, suxamethonium, succinylmonocholine, stress.

Hattingh J.; Pitts N.I.; de Vos V.; Moyes D.G.; Ganhao M.F. **The response of animals to suxamethonium (succinyldicholine) and succinylmonocholine.** *Journal of the South African Veterinary Association* (1991) 62 No. 3, 126-129 (En.) Departments of General Physiology and Anaesthesiology, University of the Witwatersrand, 2050 Johannesburg, Republic of South Africa.

INTRODUCTION

The blood biochemical responses of wild animals to suxamethonium during culling have been well documented[5 6]. Although the blood compositional changes found in elephants are similar to those seen in the blood of buffaloes, impalas, rabbits, etc, the elephants take much longer to die than do any of the other species investigated. African elephants collapse on average 2,6 min after being darted with suxamethonium, but often make attempts at voluntary movement and may remain conscious for up to 25 min. All other species studied, show iso-electric electroencephalographic recordings soon after collapse[6] which occurs within 2 to 3 min of darting.

The reason for this apparent insensitivity of the respiratory muscles of elephants to suxamethonium is not clear. In these animals there is a gradual decrease in respiratory rate after the administration of suxamethonium, in contrast to the more dramatic cessation of breathing seen in other species. The exact comparative time taken for the cessation of breathing, cardiac activity, etc. for elephants and buffaloes is reported here. In addition, the responses of animals killed with suxamethonium are compared to those killed with a mixture of suxamethonium and hexamethonium, the latter compound having been introduced in an attempt to decrease the physiological response to culling[7 8]. Furthermore, because suxamethonium (succinyldicholine) is broken down to succinylmonocholine in the body, the effects of the latter compound administered intra-venously and intra-muscularly were investigated. The sample sizes in the present study are small because of the cost of the animals and carcasses could not be used for human consumption due to the presence of drug residues. A series of different experiments were thus conducted (in some cases on single animals) in an effort to better understand the action of suxamethonium on a variety of species.

MATERIALS AND METHODS

Buffalo (*Syncerus caffer*) (n=15) impala *(Aepyceros melampus melampus)* (n=8), elephants *(Loxodonta africana* (n=11), wildebeest *(Connochaetes taurinus)* (n=10), zebra *(Equus burchelli)* (n=4) and warthog *(Phacochaerus aethiopicus)* (n=5) were darted as described previously, immobilising them with succinylmonocholine[9] or the appropriate dose of carfentanil (Jansen Pharmaceutical, Beerse, Belgium)[5 6 7 8]. The methods used for measuring arterial PO_2, PCO_2, pH, arterial blood pressure, respiratory rate, haematocrit and osmolality in immobilised animals and the concentration of plasma glucose, total lipid, total protein, lactate, cortisol and total catecholamines have also been described[2 4 6]. After completion of all procedures in immobilised animals, they were allowed to stabilise for about 20 min, control blood samples were taken and they were given an intra-muscular or intra-venous dose of either suxamethonium (Scoline, Glaxo, London, England) or succinylmonocholine[9] with or without physostigmine (Roche, Baste, Switzerland), neostigmine methyl sulphate (Kompani Ultramar, Hamburg, West Germany) or hexamethonium chloride[7 8] (Sigma, United Kingdom). Recordings continued until a zero blood pressure was recorded at which stage a further blood sample was taken in some cases. For the comparison of the effects between suxamethonium only or suxamethonium and hexamethonium, blood samples were taken at 2 min intervals until death. Results are reported as means ± S.D. or as percentage of control value where relevant.

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RESULTS

Effects of intra-muscular suxamethonium and a suxamethonium/hexamethonium mixture in animals immobilised with carfentanil.

Regular respiratory movements stopped after 5,3 ± 2,4 min in buffaloes (n=3) and after 2,1 ± 0,1 min in impalas (n=3) killed with suxamethonium and after 3,4 ± 0,7 min in buffaloes (n=3) and 1,4 ± 0,5 min in impalas (n=3) killed with the mixture. Respiratory effort ceased after 12,6 ± 4,0 and 8,0 ± 0,7 min in buffaloes and impalas killed with suxamethonium respectively, and after 5,1 ± 1,1 and 3,7 ± 1,0 min respectively in animals killed with the mixture. In all animals killed with suxamethonium. Blood pressure also tended to fall faster in the mixture group and by plotting PO_2 versus blood pressure, it was found that in all animals, blood pressure decreased only when arterial PO_2 was between 25 and 20 mmHg. The changes in concentrations and values for the blood variables investigated (see above) from the pretreatment control samples to those taken immediately after no blood pressure was recorded, were similar to the ones reported earlier[5 7 8]. The only difference observed between the suxamethonium and suxamethonium/hexamethonium groups was a decreased total catecholamine response in the latter (significant, $P < 0,05$, in impala and buffaloes). Of the 3 elephants which received the mixture, one died quickly (see above) whereas the other 2 showed no effects other than a slight initial decrease in blood pressure. These animals were then given suxamethonium (60 min later) and died in the usual way.

Table 1: **Change with time from control for blood gases and blood pressure in elephants and buffaloes after the administration of suxamethonium (S) and suxamethonium/hexamethonium (S/H). Results are expressed as percentage of control value (means). Drugs were administered intramuscularly at time zero (To)**

Variable and treatment		Time (min)										
		0	2	4	6	8	10	14	18	22	26	30
Buffaloes												
PO_2	S(n=3)	100	74	35	20	18	18	-	-	-	-	-
	S/H(n=3)	100	68	28	18	18	18	-	-	-	-	-
PCO_2	S(n=3)	100	102	110	130	-	-	-	-	-	-	-
	S/H(n=3)	100	124	144	158	-	-	-	-	-	-	-
Blood	S(n=3)	100	102	104	55	20	0	-	-	-	-	-
pressure	S/H(n=3)	100	102	94	64	33	0	-	-	-	-	-
Elephants												
PO_2	S(n=3)	100	94	87	80	75	69	51	35	25	19	18
	S/H(n=1)	100	86	70	60	55	49	24	18	18	18	18
PCO_2	S(n=3)	100	105	110	116	124	132	185	228	252	260	264
	S/H(n=1)	100	125	150	172	195	218	226	242	250	250	250
Blood	S(n=3)	100	104	109	111	111	111	105	70	44	30	0
pressure	S/H(n=1)	100	98	96	92	84	76	15	0	-	-	-

Effects of darting animals with succinylmonocholine

A number of animals from different species were darted with succinylmonocholine. The results are shown in Table 2. In animals that were immobilised and which recovered subsequently, respiration was regular, mucous membranes were pink, muscle fasiculations were observed and a marked hyperaemia of the conjunctival mucous membranes was evident. In one of the buffaloes investigated (No. 3, Table 2), 0,02 mg kg^{-1} physostigmine was administered 32 min after darting and the impression gained was that succinylmonocholine acts as a neuromuscular blocker in a fashion similar to that of suxamethonium (i.e. depolarising), but that the respiratory muscles are affected less.

Effects of intra-venous suxamethonium and succinylmonocholine in animals immobilised with carfentanil:

The results obtained are shown in Table 3. In all animals investigated, the intra-

elephants, regular respiratory movements continued until a few minutes before blood pressure was zero, with a gradual decrease in rate and depth throughout the time period (n=3 for suxamethonium and n=1 for suxamethonium/hexamethonium). The percentage change in PO_2, PCO_2 and blood pressure with time was proportionately similar for impala and buffaloes, for both the suxamethonium and mixture groups. For clarity, only the buffalo and elephant results are presented in Table 1. The results show that all responses in elephants are delayed compared to those of buffaloes and that in animals exposed to the mixture, PO_2 decreased and PCO_2 increased sooner than in

venous administration of either suxamethonium or succinylmonocholine resulted in a change in respiration after a lag period (A in Table 3); cessation of breathing in the case of the former compound and deep breaths (sighs) in the case of the latter. Respiration then usually returned to normal (B in Table 3) with no further effects. In some cases, however, respiration gradually decreased in rate and depth and finally stopped (C in Table 3). The dosages of the 2 substances used, never resulted in quick and complete irreversible cessation of breathing, although it is postulated here that this would occur with greater amounts. Two responses were thus observed with intravenous

suxamethonium, depending on dosage: cessation of breathing, followed by a return to normal and cessation of breathing, followed by a return to normal with subsequent gradual decrease in respiratory rate and depth until all activity ceased. When physostigmine was administered together with suxamethonium, respiration was completely inhibited at a dose level which the animal had previously survived (Table 3). When an elephant was given neostigmine before receiving a low dose of intramuscular suxamethonium, decreased rate and depth of respiration were observed for a short while, followed by a more prolonged inhibition (Table 3). Succinylmonocholine always produced sighs and when given in sufficient amounts, resulted in a gradual inhibition of the rate and depth of respiration.

Table 2: **Effects of darting animals with succinylmonocholine**

Species	Dose	Time (min) from darting: Time to recumbancy	Time to up	Time to cessation of respiration
Buffalo *(Syncerus caffer)*				
Adult female	3g	7,20	38,29	-
Adult female	5g+	4,11	-	6,30
Adult female	3g	8,02	55,00	-
Adult female	5g+	5,50	-	28,07
Sub adult female	1,5g	No effect		
Adult male	5g	7,40	46,30	
Sub adult female	3g	No effect		
Impala *(Aepyceros melampus melampus)*				
Adult male	0,5g	about 15	53,30	-
Adult male	3g	0,50	-	4,15
Adult male	1g	3,30	-	10,25
Adult male	0,8g	No effect		
Sub adult male	0,8g	7,42	38,36	
Adult male	0,7g	21,02	55,54	
Adult male	0,6g	No effect after 36 min		
Wildebeest *(Connochaetes taurinus)*				
Adult male	1g	No effect after 50 min		
Adult male	2g	8,06	Death due	to regurgitation
Adult male	1,35g	25,5	35,35	-
Adult male	1,5g	No effect after 40 min		
Adult male	1,5g	No effect after 40 min		
Adult male	1,8g	29,16	44,38	-
Sub adult male	2,0g	No effect after 30 min		
Adult male	2g	6,22	-	24,55
Zebra *(Equus burchelli)*				
Adult female	2,5g	No effect after 30 min		
Adult male	3,5g	67	9,56	-
Adult male	3,5g	7,56		16,34
Adult male	2,5g	No effect		
Warthog *(Phaccohaerus aethiopicus)*				
Adult male	0,6g	No effect		
Adult female	0,7g	14,06	102	-
Adult female	0,5g	7,42	-	25,30
Sub adult male	0,5g	No effect		
Adult male	0,5g	No effect		
Elephant *(Loxodonta africana)*				
Adult male	25g	No effect		

DISCUSSION

Elephants showed a different response to intra-muscularly administered suxamethonium during culling compared to buffaloes[5]. Although the blood constituent and cardiovascular changes were similar in both species, the time course was markedly different. Elephants showed a gradual and slow inhibition of respiration. This cannot be explained by differences in dosage, because in this study both species received about 3 mg kg^{-1} suxamethonium. In addition, the same response was observed, irrespective of route of administration, i.e. intra-venous or intra-muscular, although time differences were evident (see also above). These results may indicate a difference in the affinity of the nicotinic acetylcholine receptors of the respiratory muscles of *elephants* for suxamethonium, or a different mode of action of suxamethonium in these animals. A combination of these effects is possible. It is known that hexamethonium has an antagonistic effect at certain skeletal neuromuscular junctions during a competitive blockade[1]. Suxamethonium is a depolarising neuromuscular blocker, but its effect was seemingly antagonised in 2 of the 3 elephants investigated, although not in one of the animals of the other species studied. Furthermore this effect was not seen in any conscious elephants culled with the mixture[8]. The anaesthetic used is not known to have any effects on the action of neuromuscular blockers, but could possibly have influenced the combined action of suxamethonium and hexamethonium in elephants. Collectively, these observations may point to unique acetylcholine receptors in the respiratory muscles of these animals.

Hexamethonium in combination with suxamethonium, when administered to immobilised impalas and buffaloes, resulted in more rapid changes in PO_2, PCO_2 and blood pressure than when only suxamethonium was used. These effects were also seen in the one elephant which died when the mixture was given. The results indicate that the effects of hexamethonium at autonomic ganglia and other areas where transmission may be blocked (e.g. chemoreceptors), are similar in the different species. It is thus only the action of suxamethonium and the possible antagonistic effect of hexamethonium on the respiratory muscles in elephants which differed from those in other animals investigated. Another possible explanation is that it is not suxamethonium which blocks neuromuscular transmission in the respiratory muscles of elephants, but one of its breakdown products. Succinylmonocholine is one such substance and 25 g was administered to an elephant during the culling operation (Table 3). This dose is in excess of the amount of succinylmonocholine which would result from the breakdown of suxamethonium used during culling. No effects at all were observed, but the experiment was stopped after 14 min. From the results for immobilised animals, it is clear that this

Table 3: **Effects of intravenous suxamethonium and/or succinylmonocholine on immobilised animals. With repeat treatments, the time interval was 20-25 min**

Species	Drug	Dose	Time *(s) A	B	C
Buffalo					
Adult male	M	0,5 and lg	No effect		
	M	2g	235	290	366
Adult male	S	10mg	50	248	-
	S	20mg	40	136	312
Impala					
Adult male	M	0,5g	26	80	147
Wildbeest					
Adult female	S	50mg	15	37	552
Adult female	S	10mg	98	120	484
Elephant (body mass estimated from shoulder height,. National Parks Board tables)					
1200 kg male	S	900mg	76	244	-
	S	1,8g	52	264	528
240kg female	S	180mg	60	108	-
	S	360mg	36	84	-
		550mg	52	268	470
1650kg female	S	1,3g	34	60	650
400kg male	S	300mg	25	76	-
	M	600mg	21	80	-
	S*	600	95	very slight respiratory effort up to 2695	
290 male	S	200mg	33	48	-
	S	200mg	53	111	-
	M	200mg	34	67	-
	M	400mg	14	23	-
	M	600mg	104	150	1374
260kg female	S**	100mg	102	242	-
	S	100mg i.m.	248	371	3112

*Time A: From time of injection of drug until a change in respiratory pattern was observed

Time B: From time of injection of drug until respiration returned to normal

Time C: From time of injection of drug until respiration stopped

S = suxamethonium
M = succinylmonocholine
* also received 8,8 mg of physostigmine
** also received 6 mg of physostigmine

substance may block respiration (at sufficient dosage), but that it takes some time. In addition, the results obtained with intra-venously administered suxamethonium, seem to indicate that this substance does cause an initial block from which the animal may recover, but if sufficient amounts are used, this is followed by a gradual inhibition of respiration in a manner similar to the effects of succinylmonocholine. In in vivo cat sciatic-gastrocnemius preparations, succinylmonocholine has been shown to have a definite but weak blocking action[3]. When the dose was repeated after apparent recovery of the endplate transmission, a more intense block was induced. A similar result was obtained after recovery from a prior suxamethonium dose. This suggests that succinylmonocholine may have a low affinity for the nicotinic acetylcholine receptor, but that at a critical dose an effective block is induced. It is therefore possible that cholinesterase activity in elephants (true and/or pseudo) is such that at the dosages used, only succinylmonocholine reaches the motor endplates of respiratory muscles, with the resultant slow inhbition when suxamethonium is used during culling. If this were the case, it would also appear that receptor affinity is less for succinylmonocholine than for suxamethonium. Receptor affinity for these substances and cholinesterase activity therefore require further investigation on a comparative basis.

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ENCEPHALITOZOON INFECTION IN A STILL-BORN FOAL

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ABSTRACT

A stud Clydesdale foal was still-born near full term. Macroscopic examination revealed a normal placenta, pulmonary atelectasis and faint white mottling of the kidneys. Microscopically there was severe lymphoplasmacytic interstitial nephritis. Numerous organisms resembling *Encephalitozoon cuniculi* were present in the affected kidneys. The organisms occurred in the areas of inflammation as well as in the renal glomeruli and intracellular cysts in the renal tubular epithelial cells and exhibited Gram positive staining. Ultrastructurally the organisms posessed a polar vacuole and a spiral filament typical of Microsporidia. The organisms were not detected in sections of the other organs examined.

Key words: *Encephalitozoon cuniculi*, microsporidia, abortion, still-birth, equine.

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INTRODUCTION

Encephalitozoonosis is a fairly common, ubiquitous latent infection of laboratory animals such as mice, rabbits, guinea-pigs, golden hamsters and rats[5]. It is also a fairly common infection in the Arctic fox where these are bred in Scandinavian countries[17]. Vávra et al. also reported yearly epidemics of encephalitozoonosis amongst suricates *(Suricata suricata)* in the Prague zoo in Czechoslovakia[17]. In South Africa this condition has been reported in laboratory rabbits, mice[6], wild dogs[15], dogs[1 14] and a kitten[16]. Although in some of these cases the parasite was identified as *Nosema*, subsequent clarification of the taxonomic position of the parasite by Cali, clearly indicates that these were in fact cases of encephalitozoonosis[4].

In South Africa canine encephalitozoonosis is generally sporadic but serological evidence indicates a prevalence of 18% in sera collected from Pretoria and Durban and 65-70% in certain breeding establishments[12]. The position in wild rodents in this country is unknown. Serum samples from laboratory rabbits showed a prevalence of 40%[13].

Although the canine disease was originally described by Plowright[10] as an "encephalitis-nephritis"-syndrome, in some instances the organisms disseminate more widely, affecting organs such as the liver, lungs and myocardium. The renal lesions in dogs usually comprise a severe, progressive widespread, sub-acute plasmalymphocytic interstitial nephritis accompanied by the presence of *Encephalitozoon* parasites in glomeruli and tubular epithelial cells in intracellular cystic forms, or freely in areas of inflammation, or in tubular lumens after rupture of such cysts[1 7 10].

Although Waller et al[18] found serological evidence of *Encephalitozoon* in horses no morphological description of lesions due to *E. cuniculi* infection in equines could be traced in a search of the literature. This report deals with an isolated case of a dead-born foal, showing renal lesions and organisms identical to those encountered in canine encephalitozoonosis.

A stud Clydesdale mare from the Clarens district in the Orange Free State delivered a still-born foal, near full term. The mare had aborted the previous season and this was the third foetal loss in the stud out of 9 pregnancies during that season. The mare showed no pyrexia or any other obvious clinical signs of systemic disease before, during, or after delivery of the foal.

The foal and placenta were rapidly cooled down in a deep freeze, but not frozen, and despatched to the Faculty of Veterinary Science, University of Pretoria, where a post mortem examination was conducted approximately 24 h after birth.

No placental abnormalities were identified. Foetal examination revealed mild hydrothorax, total atelectasis of both lungs, mild haemoperitoneum and congestion of the abdominal organs. The kidneys were congested and had an indistinct whitish mottled appearance of the cortex.

Specimens of the brain, spleen, liver, kidneys and intestines were fixed in 10% buffered formalin for histopathological examination. Sections were prepared according to standard procedures.

The only lesion of significance was encountered in the kidneys which consisted of a severe diffuse, interstitial lymphoplasmacytic nephritis (Fig. 1). Haematoxylin- and eosin-(HE) stained sections revealed numerous parasites morphologically resembling *E. cuniculi.* These parasites characteristically do not stain well with HE. However, application of Gram's stain using the Goodpasture as well as the Brown-Hopps methods, highlighted the distinctly Gram positive nature of the organisms which were morphologically indistinguishable from *E. cuniculi* (Fig. 2 & 4). Numerous intracellular cysts, most of which occurred in tubular epithelial cells (Fig. 3), were observed. Numbers of free-lying organisms were also seen in areas of cell infiltration, as well as in tubular lumens. Organisms were also encountered within endothelial cells of the glomerular tuft or lying freely in Bowman's space (Fig. 2). The organisms measured 2,4 x 1,3 μm. No parasites or signs of inflammation were

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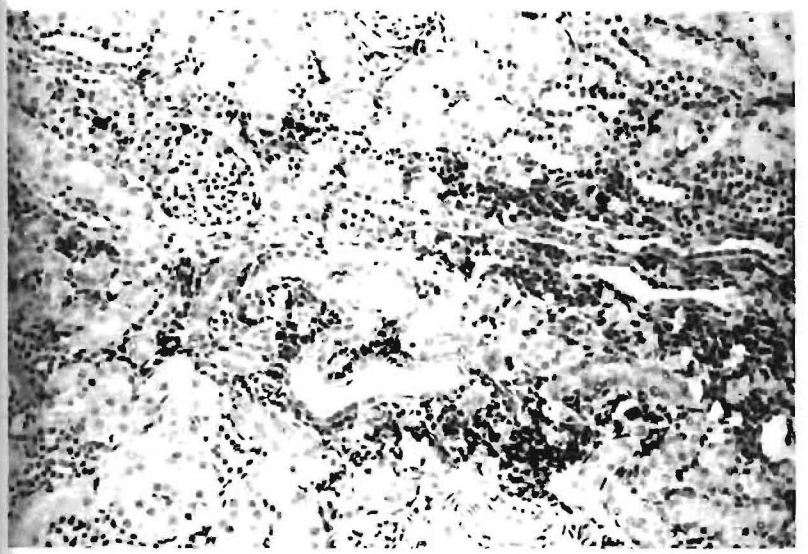

Fig. 1: **Diffuse lymphoplasmacytic interstitial nephritis in a stillborn foal. HE X 100**

seen in sections of the brain, liver, spleen or intestinal tract.

Formalin-fixed specimens of the kidney were transferred to 4% glutaraldehyde in Millonigs phosphate buffer, and postfixed in buffered one per cent osmium tetroxide. Thin sections were stained with uranyl acetate and lead citrate.

Microsporidial spores with the characteristic morphology of *E. cuniculi* were present in glomerular spaces as well as in renal tubular epithelial cells. The spores were generally electron-dense but in some a polar vacuole, and in others 5-7 coils of the polar filament, were visible.

Another mare of the stud was induced to foal at 370 d gestation by injecting 20 i.u. oxytocin (Oxytocin, Ciba-Geigy) intravenously. Foetal manipulation was necessary to correct an abnormal presentation. In spite of intensive supportive therapy, the foal died 3 d later. A post mortem and histopathological examination did not reveal any signs of *Encephalitozoon* infection.

Uterine biopsies were performed on the 2 mares which gave birth to the abovementioned foals. Histopathological examination of the samples did not reveal significant lesions.

Serum from the mare as well as from the rest of the stud was checked for antibodies against *Encephalitozoon* using fluorescein labelled antihorse IgG (FITC-GOAT X HORSE IgG, Zymed, California). These tests yielded negative results.

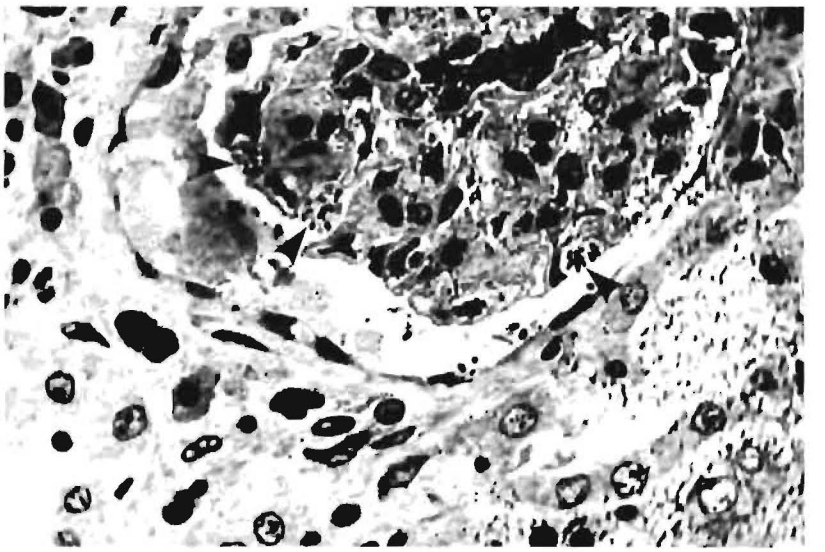

Fig. 2: ***Encephalitozoon cuniculi* parasites in the glomerular endothelium and some free-lying organisms in Bowman's space (arrow). Toluidine Blue X 1000**

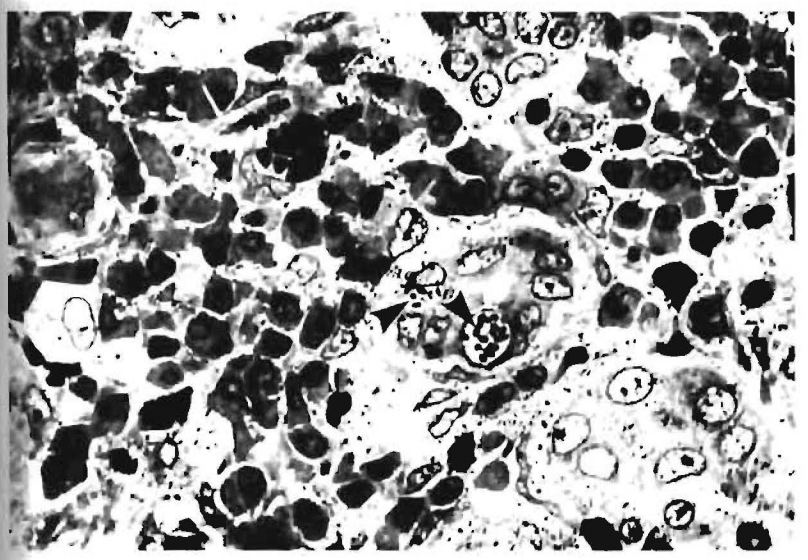

Fig. 3: **Intra-epithelial parasitovorous cysts in renal tubule surrounded by inflammatory cells. Gram's X 1000**

E. cuniculi is a parasite of warm-blooded vertebrates. It was originally confused with *Nosema* spp. which affects invertebrates such as bees as well as cold-blooded vertebrates[5]. It is commonly associated with laboratory animals, but several cases have been reported in dogs[1,3,7,14]. This case probably represents the first report of this parasite in equines. However, in 1988 a case of placentitis from a mare revealing numerous *Encephalitozoon* parasites (as well as Chlamydia) was diagnosed and taken up in the FIP collection in a conference organised by Parker[9]. Unfortunately such an unusual diagnosis was not suspected in the foal reported on in this paper, at the time of the necropsy, with the result that a urine sediment ex-

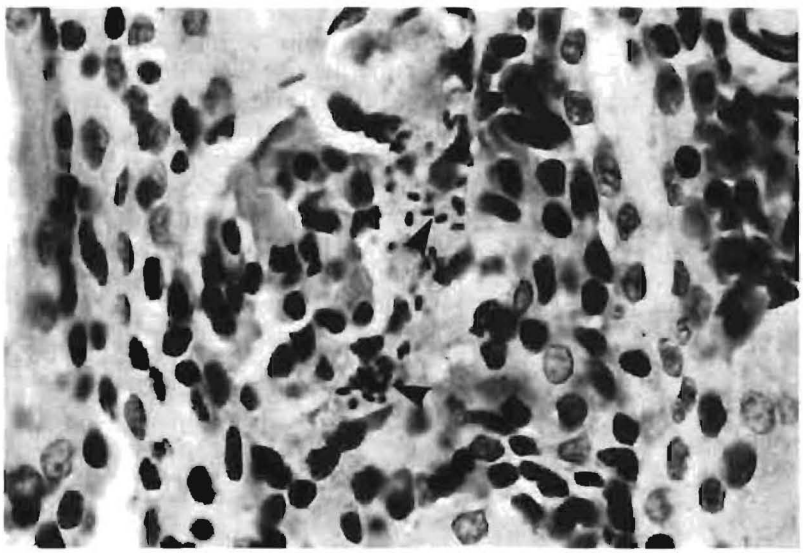

Fig. 4: **Free-lying *Encephalitozoon* organisms in an area of lymphoplasmacytic nephritis (arrow). Gram's X 1000**

amination was not carried out, nor were attempts made to culture or transfer the parasite to a laboratory animal.

The only evidence which substantiates the diagnosis of *Encephalitozoon* infection, is the histopathological and electron microscopical finding of numerous organisms in the kidneys of the subject which morphologically[2 3 5 14], as well as in its staining characteristics, resemble *E. cuniculi*. The ultrastructural demonstration of a single nucleus and the coils of the spiral polar filament are strong evidence of a Microsporidian. The presence of 5 to 7 coils, is indicative of *Encephalitozoon*[2 5 7 8]. The nature of the resultant inflammatory process is also consistent with *Encephalitozoon* infection, as it resembles very closely the pathology described in cases of canine encephalitozoonosis[1 3 7 10]. From a differential diagnostic point of view the authors are confident that the organisms are not *Toxoplasma, Besnoitia, Klosiella, Hepatozoon, Sarcocystis* or botryomycotic *Staphylococci*. The severity and diffuse nature of the ensuing nephritis caused by the infection, was beyond doubt the cause of death in this still-born foal.

The absence of antibodies against *E. cuniculi* in the mare is difficult to explain. One can only speculate that this may have been a different strain, or that the mare was only transiently infected or that the infection involved the foetus only. It is suspected that the parasite in man is not identical to *E. cuniculi*[11].

Transplacental infection is well documented[5] and this must explain the intra-uterine infection in this foal. The more common route in nature is the oral route of infection from infected litters or urine contamination of the feed and is probably the route by which the mare had picked up the organism. The intra-uterine death of this foal near term is also consistent with the manifestation of *E. cuniculi* infection in other species where neonatal mortality is a common finding[1 3 7 10 17].

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ERYTHEMA MULTIFORME IN TWO HORSES

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ABSTRACT

Erythema multiforme is reported for the first time in 2 South African horses. Both horses displayed a sudden, fulminant outbreak of raised, non-alopecic and non-pruritic plaques over the dorsolateral aspects of the neck and trunk. In both cases the distribution of the lesions was bilaterally symmetrical. Histopathological findings included hydropic degeneration of basal epidermal cells, eosinophilic necrosis of individual or groups of keratinocytes, intra-epidermal and sub-epidermal cleft formation and mixed, dermal, perivascular infiltrates. An initiating cause could not be identified in either case. Both horses underwent gradual spontaneous remission within 3 months.

Key words: Erythema multiforme, horse, skin disease.

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INTRODUCTION

Erythema multiforme is an acute, self-limiting dermatosis which has been described in man[1-3], dogs[4-7] and cats[6 8]. Two cases have been reported in horses[9 10]. The aetiology of erythema multiforme is unknown[1-3]. However, in man it has been associated with infections (viral, bacterial, fungal and mycoplasmal), drugs, neoplasia, collagen-vascular disorders, contact reactions, sunlight, cold and endocrine factors. In approximately 50% of cases the precipitating factor is unknown[2]. Cases in dogs have been associated with infections (staphylococcal folliculitis, anal sacculitis)[6 7] and drugs (aurothioglucose, cephalexin, chloramphenicol, diethylcarbamazine, gentamicin, levamisole, l-thyroxine, trimethoprim-sulphadiazine)[4-7]. Some cases have been idiopathic[6]. In cats, erythema multiforme has been associated with drug therapy (penicillin, aurothioglucose)[6, 8]. In the 2 cases of erythema multiforme reported in horses, a precipitating factor was not identified[9 10]. In man, there is no sex predilection for the disease, however, patients are most commonly in their second or third decade of life[2]. No age, sex or breed predilection can be identified among cases described in dogs, although more males are represented than females[4-7]. The incidence of the disease is relatively common in man[2], uncommon in the dog and rare in the cat[6].

The exact pathogenesis of the disease is unknown. In man, immunological studies of lesions less than 24 h old show deposits of IgM and C3 in the walls of the superficial dermal microvasculature. In addition, circulating immune complexes have been demonstrated[2 3]. It may thus be hypothesised that immune complex formation and subsequent deposition in the cutaneous microvasculature may play a role in the pathogenesis of the disease.

In man, acute attacks usually resolve within 3 to 6 weeks. Some patients show a tendency toward periodic recurrence and in most instances are free of lesions for long periods between episodes. Occasionally, however, attacks occur with such frequency that lesions persist from one episode to the next[2 3]. In animals the condition undergoes resolution within one to 12 weeks following removal of the precipitating factor or simply due to the natural self-limiting course of the disease[5-11]. In severe cases, scarring may remain in places where ulceration has occurred[4]. Permanent ocular sequelae include conjunctival scarring, corneal perforation, opacities and uveitis[2 4].

The disease in man and dogs has variable clinical and histological manifestations, depending on the severity of the primary lesions[1-3 6]. Mild lesions include erythematous macules, patches, papules and plaques. Target lesions are characteristic, representing macules or papules which have expanded peripherally and cleared centrally (usually within 12 to 24 h). Severe lesions include vesicles and bullae with or without ulceration. Lesions may be seen on the skin and/or mucous membranes (notably the oral, nasal and conjunctival mucosae and mucocutaneous junctions). In mild cases the lesions are usually asymptomatic, the overlying hair coat is normal and the condition is referred to as erythema multiforme minor. Erythema multiforme major or Stevens-Johnson syndrome is a more severe form of the disease involving the skin and mucous membranes with variable degrees of visceral involvement and significant constitutional symptoms. Vesicles and bullae appear suddenly on the skin and mucous membranes and rupture, to leave painful erosions and ulcers. Ocular lesions (keratitis, corneal ulceration, purulent conjunctivitis, anterior uveitis and panophthalmitis), fever, anorexia and depression are commonly seen. Diarrhoea, paronychia, onychomadesis, polyarthritis, otitis media, pneumonia and renal failure are less common. Erythema multiforme major may be fatal.

Histological findings vary according to the severity of the lesions[1 3 6]. Epidermal changes include hydropic degeneration of basal cells and eosinophilic necrosis of individual or groups of keratinocytes in mild lesions. Mononuclear cells and neutrophils may invade such areas of necrosis, and the necrotic cells subsequently lose their nuclei and coalesce. In severly affected areas, hydropic degeneration of the basal cells may result in subepidermal separation, and all keratinocytes appear necrotic, with only the horny layer remaining preserved. In other areas, however, there may be severe damage to the upper epidermal layers and less severe damage to the lower layers, resulting in intra-epidermal cleavage. Extensive epidermal necrosis in bullous-necrotic le-

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sions may be indistinguishable from that found in toxic epidermal necrolysis.

Dermal changes include a mild to pronounced perivascular infiltrate, involving mostly lymphocytes and histiocytes. Eosinophils may also be present. In some cases the infiltrate may assume a lichenoid pattern, which may be severe enough to obscure the dermo-epidermal junction. There is often pronounced oedema of the superficial dermis. In severe forms, the oedema may lead to the formation of a bulla within the epidermis with the basement membrane zone forming its roof. In some instances, the oedema may lead to epidermal spongiosis and multiple intra-epidermal vesicles associated with exocytosis. Pigmentary incontinence, vasodilatation and erythrocyte extravasation are often present in the upper dermis[3].

Treatment of erythema multiforme may be unnecessary as the disease usually runs a mild course and spontaneously regresses within a few weeks[2 6 7]. An underlying cause should be sought and corrected whenever possible. Severe cases require supportive care in order to prevent secondary infection of open lesions and to correct fluid and electrolyte imbalances when a significant proportion of the cutaneous surface is involved[2 4]. The use of corticosteroids is controversial and probably not effective in changing the course of the disease, although it is often used in severe cases in an attempt to minimise sequelae[2 4].

The purpose of this paper is to report the occurrence of erythema multiforme in 2 horses, the first cases to be reported in South Africa.

CASE REPORTS

Case 1: An 8-year-old grey Arab mare was presented with a history of a sudden, fulminant onset of "ring-like" skin lesions one week previously. The existing lesions were becoming more prominent, but no new lesions had developed. Clinical examination revealed the presence of raised, non-alopecic, annular plaques over the lateral aspects of the neck (Fig. 1) and shoulders, and the dorsal aspects of the back and rump. The lesions showed a bilaterally symmetric distribution and varied between about 5 and 8 cm in diameter. They were nonpruritic but sensitive to the touch. The early lesion was firm and non-alopecic but later became eroded, moist and covered by a yellowish crust. Affected skin was thickened and lacked extensibility. The horse was otherwise normal.

Skin scrapings, hair-pluckings, bacterial and fungal cultures were negative. Histopathology revealed mild epidermal spongiosis with pronounced individual keratinocyte necrosis (Fig. 3). In some areas the keratinocyte necrosis was extensive enough to cause intra-epidermal cleft formation (Fig. 4). Hydropic degeneration of basal epidermal cells was mild to marked and in some areas there was sub-epidermal cleft formation (Fig. 5). The dermis showed a marked superficial dermal oedema and a moderate perivascular infiltrate involving lymphocytes, histiocytes and eosinophils.

A diagnosis of erythema multiforme was made. The owner was again consulted regarding the horse's possible exposure to an inciting agent e.g. drugs, contact reactants, physical factors. No initiating factor could be identified.

Treatment involving the use of topical fly-repellents (Fly-Away, Centaur) and once daily bathing of eroded lesions with chlorhexidine solution (Hibitane, ICI) was instituted. The condition remained static for a few weeks and thereafter underwent gradual, spontaneous remission within 3 months. The horse has remained normal for 18 months.

Case 2: A 5-year-old bay Hannovarian mare was presented with a history of a previous episode of "urticaria" of sudden onset, involving the dorsal aspects of the animal from the withers to the tail-base. The horse had exhibited hyperaesthesia over the affected areas. Treatment with antihistamines had been given and the horse had recovered over an unspecified period of time. Three weeks after remission, the lesions had suddenly reappeared and were first noticed when the horse was brought in from the paddock in the evening. No lesions had been noted during the morning grooming session before the horse was put out to graze. Treatment involving antihistamines, glucocorticoids, antifungal and insecticidal washes, had been ineffective.

Clinical examination revealed skin lesions on the dorsal aspect of the animal with a bilaterally symmetric distribution, which extended from the withers to the tail base and from 5 to 7cm on either side

Fig. 1: **Non-alopecic, annular plaques on the lateral aspect of the neck of a horse with erythema multiforme (Case 1)**

Fig. 2: **Multiple plaques arranged in a "crazy-paving" like pattern on the lumbosacral area of a horse with erythema multiforme (Case 2)**

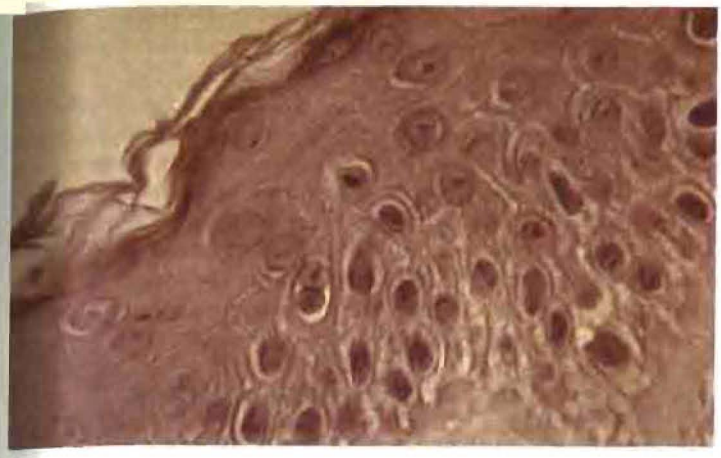

Fig. 3: **Photomicrograph of skin biopsy (Case 1). Note mild acanthosis, epidermal spongiosis and eosinophilic necrosis of a single keratinocyte (H:E, X 400)**

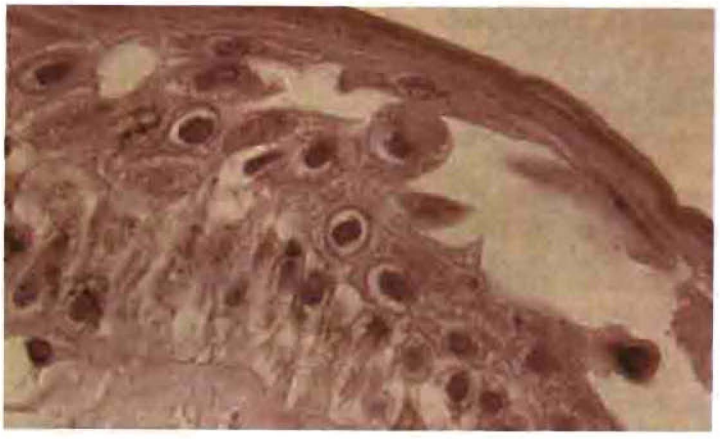

Fig. 4: **Photomicrograph of skin biopsy (Case 1). Note hydropic degeneration of basal keratinocytes, eosinophilic necrosis of keratinocytes and intra-epidermal cleft formation (H:E, X 400)**

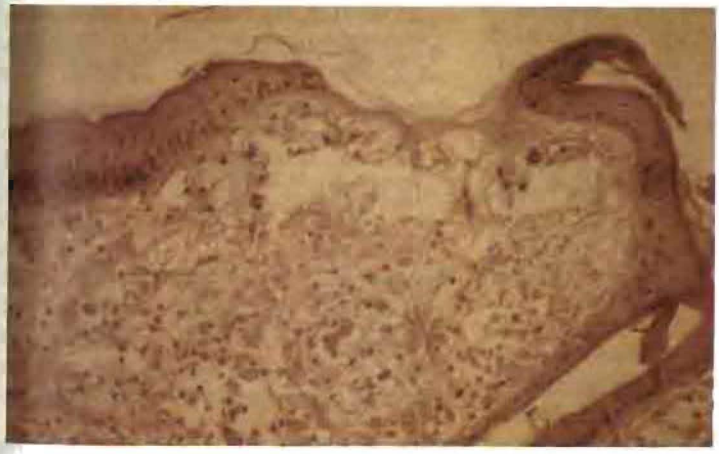

Fig. 5: **Photomicrograph of skin biopsy (Case 1). Note marked superficial dermal oedema with infiltration of mononuclear cells and eosinophils. Also note sub-epidermal cleft formation and necrosis of the overlying epithelium which has been retracted to the right (H:E, X 100)**

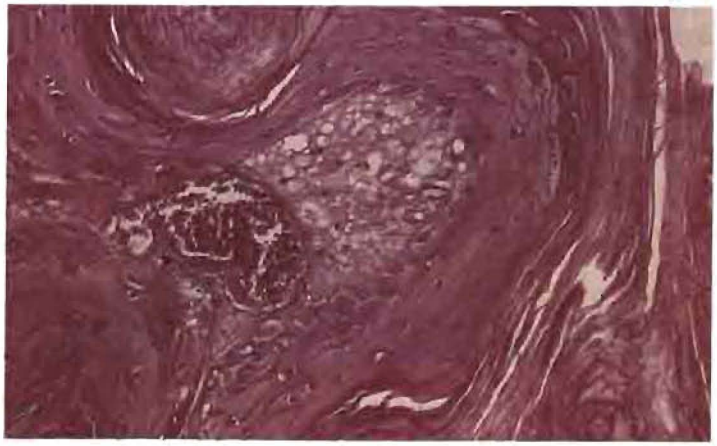

Fig. 6: **Photomicrograph of skin biopsy (Case 2). Note marked orthokeratotic hyperkeratosis and acanthosis. Also note the necrosis of individual keratinocytes, superficial dermal oedema and dilatation of dermal blood vessels (H:E, X 100)**

of the midline. The lesions were firm, raised, non-alopecic plaques arranged in a "crazy-paving" like pattern (Fig. 2). The affected skin exhibited a decrease in elasticity and extensibility. Secondary lesions included thick crusts overlying some plaques, which could easily be removed together with the overlying hair, leaving an oozing erosion below. Pruritus was absent, but the horse was hyperaesthetic over affected areas to the extent that she could no longer be saddled. The horse was otherwise normal.

Skin scrapings, hair pluckings, bacterial and fungal cultures were negative. Direct smears made from the undersides of crusts, revealed only keratinised cells. Histopathology revealed a marked orthokeratotic hyperkeratosis and acanthosis with individual keratinocyte necrosis and focal areas of hydropic degeneration of basal keratinocytes (Fig. 6). Superficial dermal oedema was present, with dilatation of the superficial dermal blood vessels and extravasation of red blood cells in places. A moderate perivascular infiltrate involving mostly lymphocytes, histiocytes and a few eosinophils was present in the dermis, along with a moderate, diffuse pigmentary incontinence.

Erythema multiforme was diagnosed but investigation for the inciting cause was again unsuccessful. The crusts were removed during grooming and the lesions disappeared within 6 weeks. No treatment was given. The horse has remained normal for 9 months.

DISCUSSION

Very few cases of erythema multiforme have been reported in horses[9] [10]. However, an increasing awareness of the clinical and pathological features of the disease will probably result in the recognition of a greater incidence of the disease in our domestic animals.

In previous reports of erythema multiforme in the horse, no inciting cause could be identified[9] [10]. In the 2 cases reported here, we were unable to find precipitating factors in that there was no history of previous drug administration, illness or change in feed or environment.

Clinical lesions encountered in horses have been mild and self-limiting, in that spontaneous remission has occurred within 3 months. The onset has been acute and the distribution of lesions symmetric. In all cases the primary lesions include papules and plaques which assume an annular, arciform or serpiginous configuration. The lesions are nonpruritic, although they may be sensitive to the touch, and nonalopecic. Secondary lesions involving erosions and crusting may be encountered. The lesions of erythema multiforme can be differentiated from the wheals seen in urticaria in that they are firm, do not pit on pressure and are more persistent (lasting weeks to months as opposed to hours or days). Similar lesions to those seen in erythema multiforme, are seen in cases of allergic contact dermatitis and arthropod reactions.

Severe cases of erythema multiforme have not been reported in the horse, however, in other species these must be differentiated from toxic epidermal necrolysis (considered by some workers to be a maximal expression of severe erythema multiforme, and often histologically indistinguishable), other bullous skin diseases, staphylococcal scalded skin syndrome, fixed drug eruptions, systemic lupus erythematosis, acute graft-versus-host disease and epidermotropic lymphoma.

Histological findings in horses[9-11] are much in accordance with the findings in man[1-3], dogs and cats[4-8]. Epidermal changes include orthokeratosis, focal parakeratosis, mild to moderate spongiosis, focal hydropic degeneration of basal keratinocytes and necrosis of individual keratinocytes. Sub-epidermal and intra-epidermal cleavage has been encountered. Dermal changes include pigmentary incontinence, superficial dermal oedema, dilatation of dermal vessels, extravasation of erythrocytes and perivascular accumulation of mononuclear cells (and sometimes eosinophils).

The histological picture seen in mild forms of erythema multiforme, must be distinguished from that seen in allergic urticarial eruption. The latter involves papillary oedema with a mixed inflammatory-cell infiltrate and is not associated with histologic alterations of the dermo-epidermal interface. Other histologic differential diagnoses include acute fixed drug eruption, toxic epidermal necrolysis and graft-versus-host reactions, from which erythema multiforme cannot always be distinguished.

Specific treatment of erythema multiforme is unavailable and probably not necessary in mild cases as they have been seen in horses. However, one should attempt to establish a precipitating factor which should be removed if possible. Treatment of secondary lesions aimed at minimising fly-worry and preventing secondary infection is advisable.

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POX VIRUS INFECTION IN CAPTIVE JUVENILE CAIMANS *(CAIMAN CROCODILUS FUSCUS)* IN SOUTH AFRICA

MARY-LOUISE PENRITH*, J.W. NESBIT**, and F.W. HUCHZERMEYER*

ABSTRACT

Light grey macules developed on the skin and in the mouths of juvenile caimans, *(Caiman crocodilus fuscus)* (n=8), kept in the quarantine section of the reptile park at the National Zoological Gardens, Pretoria, Republic of South Africa. The gross, histopathological and ultrastructural features of the lesions were commensurate with pox virus infection. This outbreak closely resembled the disease described elsewhere in 3 juvenile captive caimans.

Key words: Pox virus, caiman, *Caiman crocodilus fuscus*

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In the class Reptilia, disease caused by pox viruses has only been recorded in crocodilians. Banks[2] described skin lesions in the Arafuran file snake, *(Acrochordus arafurae)*, which he suggested resembled caiman pox lesions, but no confirmatory histopathology was done.

Poxlike skin lesions in juvenile captive caimans *(Caiman sclerops = Caiman crocodilus fuscus)* were first reported in Florida[6]. The authors stated that similar lesions were considered by veterinarians and importers to be "not uncommon" in caimans, but apparently had not been encountered outside Florida.

An outbreak of poxviral disease in farmed Nile crocodiles, *(Crocodilus niloticus)*, in South Africa was described by Horner[1,4]. This is the first report of an outbreak of pox virus infection in captive juvenile caimans *(Caiman crocodilus fuscus)* in South Africa.

The National Zoological Gardens, Pretoria, Republic of South Africa received a consignment of juvenile caimans (of indeterminate age) bred at Pet Farm in Florida, United States of America, on 16th February 1990. All were placed in the quarantine section of the reptile park, which is an insect-proof enclosure isolated from the remainder of the reptile park, with a separate water supply and drainage system. The water in the pond is derived from the municipal supply and is changed daily. The temperature in the quarantine ward is maintained at approximately 30°C by cable heaters. The group of 8 that survived the first 6 weeks, developed extensive, light grey, discrete (occasionally coalescent), circular and macular skin lesions 1-3 mm in diameter in May 1990 (Fig. 1). The lesions occurred mainly on the dorsal surface of the head and body as well as the limbs; no lesions were found on the ventral surface. At a post mortem examination performed on a specimen that died on 30th May 1990 as a result of an enteritis, it was noted that similar lesions were present on the palate, tongue, and gingiva. The remaining live animals were examined and found to have similar lesions on the skin and in the oral cavity, but were otherwise healthy, with a good appetite and habitus.

Subsequent to the discovery of the extent of the lesions, artificial sunlight was supplied by means of incandescent and ultraviolet light, and additional cover was supplied to reduce stress and promote basking. Over a 6-week period, a considerable reduction in the number and extent of the lesions was noted in the remaining caimans.

A complete necropsy was undertaken on the caiman that died acutely. Samples of several skin and mucosal lesions as well as from brain, lung, trachea, kidney, liver, heart and gastrointestinal tract tissue were taken from the necropsied caiman in 10% buffered formalin for histopathological investigation. Sections

Fig. 1 **Juvenile caiman with round, pale macular lesions on dorsal surface of head, body and on limbs**

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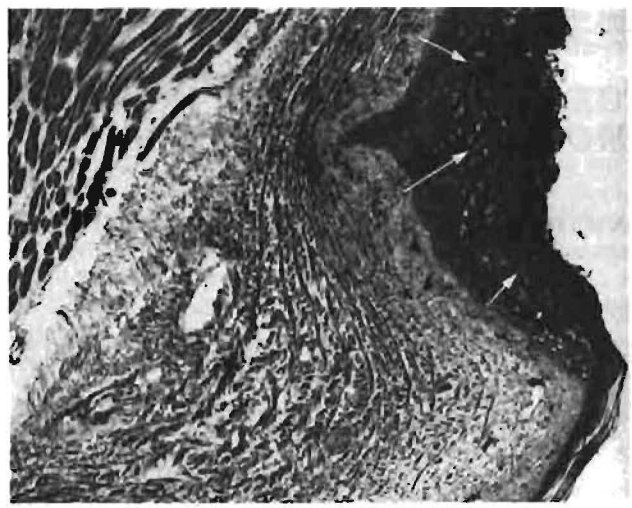

Fig. 2: **Section through skin showing a pox lesion. Intracytoplasmic inclusion bodies are arrowed. HE X 40**

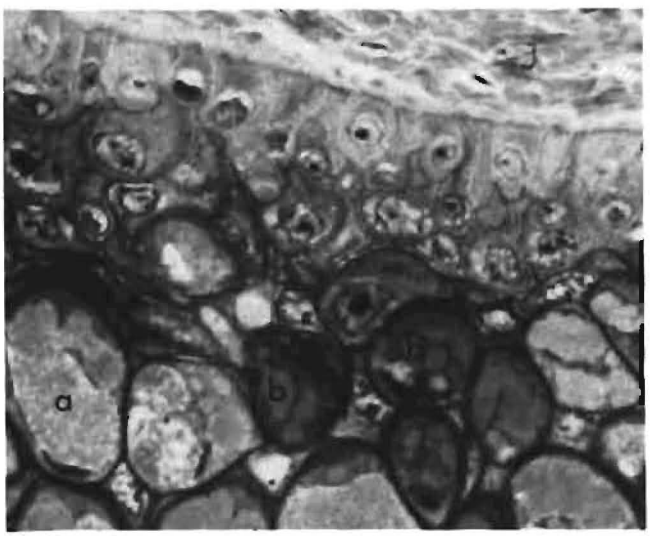

Fig. 3: **Section through lesion showing (a) Bollinger bodies and (b) Borrel bodies in cytoplasm of affected cells. HE X 200**

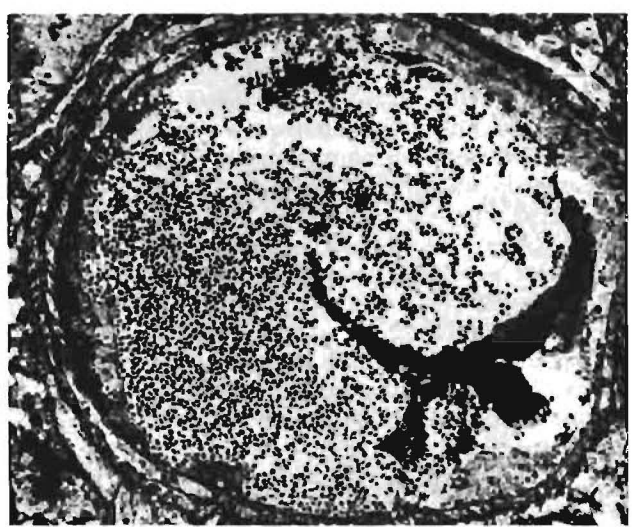

Fig. 4: **Cytoplasmic inclusion body containing viral particles in an epithelial cell. Electron micrograph X 3000**

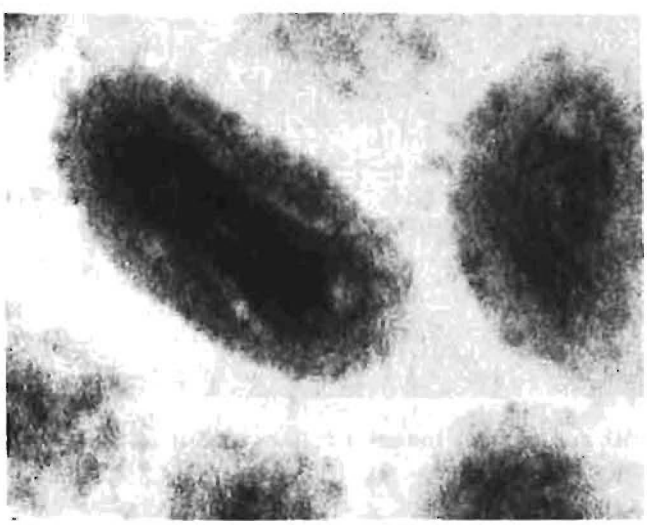

Fig. 5: **Nucleocapsid (central dumbbell-shaped body) typical of the pox viruses well delineated in the viral particles. Electron micrograph X 80 000**

were routinely prepared and stained with haematoxylin and eosin (HE), the Brown-Hopps modification of Gram's stain and periodic acid-Schiff methods. Selected portions of the skin lesion and adjacent normal tissue were processed for transmission electron microscopy.

Thin sections were stained with toluidine blue for light microscopy. Ultrathin sections were mounted on grids, stained with uranyl acetate and lead citrate and examined in a Jeol JEM1200 EX electron microscope.

Swabs for bacterial and fungal culture were taken from skin and oral lesions of a living specimen. Subsequently, skin scrapings and water samples for viral identification were taken and submitted to appropriate specialists (Veterinary Research Institute, Onderstepoort), but to date attempts to isolate the virus have not been successful.

The most significant macroscopic lesion at necropsy, was an acute severe catarrhal to pseudomembranous (necrotic) enteritis. The stomach contained 3 juvenile mice. The pox lesions were confined to the skin and oral mucosa. No other pathological lesions were present.

Examination of sections of the intestinal tract confirmed the severity of the enteritis, which was attributed to the excessive numbers of Gram negative bacilli associated with the lesion. The epidermis was predominantly involved with the pox lesions (Fig. 2 & 3) which consisted of hyperkeratosis, marked acanthosis, and partial involvement of the stratum basale. The basement membrane was thickened in some of the lesions. Changes included ballooning degeneration and necrosis of the epithelial cells. Affected cells were swollen, with nuclear changes including margination of chromatin, loss of nucleoli, and pycnosis; the pycnotic nuclei often being distorted and compressed. Eosinophilic cytoplasmic inclusions were present in many cells (Fig. 2 & 3). All lesions showed cells with large cytoplasmic inclusions resembling Bollinger bodies (Fig. 3 & 4), and some showed cells with smaller, more deeply eosinophilic inclusions resembling Borrel bodies (Fig. 3). Inflammatory changes in the dermis were minimal, and included oedema and foci of mononuclear cell infiltration.

Inclusion bodies were demonstrated in the epithelial cells by transmission electron microscopy (Fig. 4). These appeared at lower magnifications as granular areas (Fig. 4). At higher magnification they were seen as light areas containing large numbers of viral particles, as well as more irregular, granular masses. The viral particles (virions) were round to oval and contained a dumbbell-shaped body, the nucleocapsid (Fig. 5).

Pseudomonas aeruginosa was isolated from the skin swabs. The macroscopic and microscopic evidence implicated bacterial enteritis as the cause of death of the necropsied caiman. The macroscopic, microscopic and ultramicroscopic features taken in conjunction with the distribution of the skin lesions bear a strong resemblance to previous descriptions of pox virus infection in caimans[5 6]. The virus exhibits a distinct tropism for the epidermis and epithelium of the oral cavity. The dumbbell-shaped nucleocapsid as revealed by electron microscopy provides positive identification of the virus as belonging to the pox virus group[3 6 7].

The origin of the infection is unknown. However, 3 epidemiological aspects are significant in this outbreak. Firstly, the fact that the caimans originated from Florida in the USA, to which area the infection has hitherto been unique[5 6]; secondly, that the disease occurred in all of the animals shortly after arrival; and thirdly, that the caimans were kept under strict quarantine. These factors suggest that the infection was present in a latent but subclinical form and underwent clinical recrudescence under the stress of a changed environment. It is unlikely that infection occurred subsequent to their arrival. Complicating stress factors probably involved lack of sunlight and disturbance due to activities of staff in the isolation unit. Part of the managemental control of skin conditions in farmed Nile crocodiles entails increasing the time the animals spend out of the water, and that they preferably be exposed to direct sunlight. In addition to a presumed development of immunity, the provision of artificial sunlight, additional cover, and large basking surfaces out of the water, resulted in an improvement in the skin condition of the caimans.

Comparison of pox virus infection in caimans and crocodiles reveals a number of distinct differences. While the microscopic and ultrastructural appearance of the lesions is very similar to that of pox virus infection in the Nile crocodile[4], the macroscopic appearance and distribution of the lesions is very different. Crocodiles usually develop brown, wart-like lesions mainly on the skin of the head, sides of the mouth and ventral neck, the sides of the body, the belly, and on all four limbs[4]. Lesions may show patterns that suggest association with trauma due to bite wounds. The occurrence of lesions on the skin of the belly is potentially important from an economic viewpoint, this being the most valuable part of the skin. Crocodiles develop pox lesions when kept in overcrowded and unhygienic conditions, while the caimans developed lesions in a very clean and spacious tank. The pattern of the lesions did not indicate any association with trauma in the caimans. Pox lesions have not been noted in any of the Nile crocodiles kept in the collection of the National Zoological Gardens, which are completely separated from the caimans in the quarantine unit.

It therefore seems likely that the caimans became infected with the pox virus before leaving Florida, that a long period of latency is possibly, and that the caiman pox virus is different to the pox virus that affects Nile crocodiles in South Africa, even if it should prove to be transmissible to them.

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LITHIUM TOXICITY IN TWO DOGS

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ABSTRACT

Two cases of lithium toxicity are reported on in dogs having had lithium hypochlorite chlorinated water as their sole source of drinking water. Clinical signs in one dog included polyuria, polydipsia, loss of body mass, dehydration, diarrhoea and general weakness and in the other case, polyuria, polydipsia, loss of body mass and seizures. Withdrawal of the water resulted in complete recovery.

Key words: Toxicity, lithium, dog, polyuria, polydipsia, seizures, muscle tremors

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INTRODUCTION

Lithium carbonate is used in man to treat a multitude of psychiatric disorders including manic-depressive conditions. Lithium treatment has resulted in the reversal of the long-term prognosis of manic-depressives, effecting total rehabilitation in most cases[12]. The use of lithium to stimulate granulocytopoiesis in humans, has recently been reviewed[2]. In dogs, lithium has been used to stimulate erythropoiesis in aplastic anaemia due to oestrogen toxicity[7 9] and other disorders of erythropoiesis[10]. Cyclic haematopoiesis of grey collies has been controlled with lithium therapy[6 9].

Apart from the medicinal uses of lithium, chlorine and lithium in the form of lithium hypochlorite, form a highly soluble chlorine granule which is used in swimming pool chlorination. Lithium chloride was also used as a table salt substitute in the 1940's, resulting in numerous cases of human toxicity[4 12].

This case report documents lithium toxicity seen in 2 dogs whose sole source of drinking water was swimming pool water, chlorinated with a lithium hypochlorite compound.

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CASE REPORT

Two dogs belonging to the same owner were presented for clinical investigation. Both dogs were fed a normal balanced diet, but the sole source of drinking water for several months, had been the swimming pool.

Case 1:

A 30-month-old pure-bred German Shepherd bitch was presented with a history of severe polyuria, polydipsia, a loss of body mass, intermittent diarrhoea, muscle tremors in the hindquarters and general weakness.

Faecal flotation was positive for *Ancylostoma* sp eggs, and initial laboratory blood tests showed no obvious abnormalities. Urinalysis consistently showed a urine specific gravity (SG) of 1,001. The dog was discharged after deworming with pyrantel pamoate (5mg kg^{-1} Nemex liquid, Pfizer) with a view to performing water deprivation and vasopressin concentration tests at a later stage.

Three weeks later, the animal presented with severe diarrhoea and dehydration. Urine SG was 1,006 in the presence of clinical dehydration. The haematology and blood chemistry tests were repeated with the following abnormalities (normal values for the laboratory used in parentheses): albumin 44g ℓ^{-1} (29-33g ℓ^{-1}), serum potassium 6,5 mmol ℓ^{-1} (3,7 - 5,8 mmol ℓ^{-1}), packed cell volume ,59 (,37-,55), and absolute lymphocyte count 300 x 10^9 ℓ^{-1} (1000 — 4800 x 10^9 ℓ^{-1}). Blood glucose, serum cholesterol, serum alanine transferase, serum alkaline phosphatase, serum creatinine, serum urea, serum calcium, serum inorganic phosphorus, serum sodium and serum chloride concentrations were all within normal limits.

Treatment included intravenous fluid therapy with a polyionic electrolyte solution (Ringers lactate, Labethica) (20 ml kg^{-1}), trimethoprim-sulphadiazine (Tribrissen 24%, Coopers Animal Health) (30 mg kg^{-1}) and prednisolone (Prednisolone, Centaur) (1 mg kg^{-1}). After 3 d of this therapy, the dog started eating but was still clinically dehydrated with a urine SG of 1,015. As serum potassium was still elevated (6,1 mmol ℓ^{-1}), fluorocortisone (Florinef, Bristol-Meyers Squibb) (0,2 mg d^{-1}) was added to the regimen.

At this stage it transpired that the swimming pool water, which was the only souce of drinking water, had been chlorinated with soluble lithium hypochlorite granules (Solchlor, AECI Explosives and Chemicals Limited). This had been the sole method of chlorination for 3 months. Serum lithium levels were determined, using an atomic absorption spectrophotometer (Model 5000 Perkin-Elmer Corp., Norwalk, Conn. USA), and were found to be very high (1,5 mmol ℓ^{-1}). The serum of 2 healthy control dogs showed lithium concentrations of 0,03 mmol ℓ^{-1} and 0,04 mmol ℓ^{-1}. The swimming pool water lithium level was 29 ppm compared to zero in 2 control swimming pools chlorinated with sodium hypochlorite granules. As the dog had not been exposed to lithium medication, it was assumed that the swimming pool water was the source of the abnormal lithium levels detected in the serum.

After 3 d of treatment with fluorocortisone, the serum potassium returned to normal (4,8 mmol ℓ^{-1}) and the dog was discharged. The owner was instructed to prevent the dog from drinking the pool water. The mineralocorticoid therapy was discontinued after 10 d.

Serum lithium concentration 2 months later, had decreased to 0,13 mmol ℓ^{-1} with serum sodium and potassium concentrations within the normal range. The dog was healthy and showed no further signs of weakness or polyuria. The urine SG returned to within normal limits 3 mon-

ths after the source of lithium had been withdrawn. Subsequent faecal examinations were negative for helminth eggs.

Case 2:
A 2-year-old sterilised Labrador crossbreed bitch was presented with a history of polyuria, polydipsia and loss in body mass of several months duration. She had also experienced 2 seizures during this period. Prior haematological and blood chemistry tests performed by the referring veterinarian, had detected no abnormalities. Urine SG was consistently lower than 1,010. A renal biopsy had been performed and no histopathological abnormalities had been found.

On initial examination, the patient showed no obvious clinical abnormalities. The urine SG was consistently 1,005. Serum sodium and potassium levels were within the normal range. A faecal flotation was negative for helminth ova. Because the dog belonged to the same owner as did Case 1, and as it also had lithium hypochlorite treated water as its sole source of drinking water, serum lithium concentration was determined and found to be 1,1 mmol ℓ^{-1}. A diagnosis of chronic lithium intoxication was made and therapy consisted of providing the dog with alternative drinking water.

After 2 months, the serum lithium concentration was 0,41 mmol ℓ^{-1}, urine SG was 1,019 and the dog showed none of the previous clinical signs. Seizures have not been reported in the 3 years since the lithium treated water was withdrawn as the source of drinking water.

DISCUSSION

The methods by which lithium achieves its therapeutic effects are complex. In the body, lithium exists as a small ion with a positive charge[4]. Lithium competes with sodium, potassium, magnesium and phosphorus, and displaces these minerals from bone and other intracellular sites[12]. The half-life of lithium in man and dogs is approximately 29 h[12] and 22 h[8] respectively. Muscle and bone act as reservoirs for lithium, due to sequestration in these sites[3].

Lithium is excreted unchanged in the urine, and 80% of excreted lithium is reabsorbed in the proximal and distal renal tubules[2 4 12].

Neutrophilia is induced by lithium, following the stimulation of a granulopoietin[1 6], leading to increased neutrophil output. Although lithium has been used in aplastic anaemia therapy[9], in vitro experiments with human bone marrow show that lithium decreases the generation of erythrocytes[1]. Lithium might also inhibit a population of suppressor T-lymphocytes which usually limits haematopoiesis[1].

Lithium has a narrow therapeutic index, and toxicity can arise even at levels within the normal therapeutic range[12]. Lithium poisoning can arise after a single massive overdose, or after cumulative overdose following low dose ingestion. Decreased lithium excretion due to renal disease in a patient on therapeutic doses may also cause toxicity as may decreased sodium or water intake[4 12].

Most cases of lithium intoxication in man occur with long-duration lithium therapy, and small increases in dosage may induce toxicity[12]. The typical signs of lithium intoxication in man include central nervous system manifestations, gastrointestinal signs, cardiovascular signs, neutrophilia, lymphopaenia, skin lesions and renal signs[4 12].

The central nervous signs described in human patients include initial fine hand tremors, followed by spastic or choreiform muscle tremors, parkinsonism, anxiety, seizures, delirium and coma[12]. In the more common chronic cases of human intoxication, nervous signs develop gradually, starting with fine hand tremors and progressing to severe, protracted impairment of consciousness[4]. Lithium affects nerve excitation, synaptic transmission and neuronal metabolism in many ways. The lithium ions substitute for sodium ions, leading to altered electrical conductivity and increasing the excitability of the nerve due to the raised number of positive ions in the cell[4]. The inhibition of adenylate cyclase, which decreases cylic AMP production, is an important pharmacologic and toxicologic mechanism[4 12]. Cyclic AMP acts as a "second messenger" for a multitude of hormones, including the catecholamines, adrenocorticotrophic hormone, vasopressin, parathyroid hormone, calcitonin, glucagon, gastrin and others[5]. Serotonin release in the hippocampus is stimulated by chronic lithium therapy[12], and the release and re-uptake of noradrenalin at nerve endings is inhibited[12]. The beneficial effects of lithium in the treatment of mental disease are probably due to these varied neuroendocrine effects. During lithium intoxication, the lithium ion may also lower the seizure threshold[4]. Case 1 presented with muscle tremors and Case 2 showed seizures.

Severe gastroenteritis has been seen in acute human lithium overdoses[12]. Signs which have been recorded include nausea, vomition and diarrhoea. The exact pathophysiological mechanisms by which lithium causes these signs are not well defined, and several mechanisms have been implicated. Lithium enters the mucosal cell with sodium, but the sodium ions are then actively pumped out whilst lithium remains in the cell[4]. Instead of sodium moving along its concentration gradient from the intestinal lumen into the cell, it is then retained in the gut. This leads to decreased absorption of glucose because glucose usually moves along the same concentration gradient as sodium[5], and an osmotic diarrhoea results. Lithium may also cause gastrointestinal signs due to direct irritation of the gastrointestinal mucosa[12], or by interference with gastrin[4]. The diarrhoea in Case 1 could also have been caused by concurrent hookworm infestation.

The electrocardiographic signs which have been recorded in humans, include T-wave inversion and S-T segment depression. These changes are due to lithium ion interaction with intramyocardial sodium and potassium, and are reversible[4]. Electrocardiographic examination was not performed on the 2 cases seen. Thoracic auscultation revealed no obvious cardiac problems.

Neither of the 2 patients showed the neutrophilia which has been reported in human cases of lithium toxicity. The severe lymphopaenia (lymphocyte count of 300 x 10^{-9} ℓ^{-1}) seen in Case 1, is consistent with human cases of lithium poisoning[12].

Skin lesions which have been recorded in human cases include acne, exacerbation of psoriasis, rashes and alopecia[12]. The 2 canine cases described, showed no skin lesions.

Lithium has multiple effects on the kidneys. The lithium ion competes with sodium and potassium at the renal tubular level, and may also directly decrease the normal hypothalamic thirst response to dehydration[12]. The main renal effect is nephrogenic diabetes insipidus, possibly caused by the inhibition of adenylate cyclase[4 12]. In dogs, the mechanism may involve morphologic changes in the distal tubules with less effect on the renal adenylate cyclase[3]. Under normal circumstances, water intake is greater than the water needs of the body. During lithium intoxication, water intake is decreased and dehydration develops, leading to decreased renal excretion of lithium. The resultant increased serum lithium concentration causes further inhibition of water absorption in the kidneys, exaggerating the dehydration.

Both dogs presented with severe polydipsia, polyuria and decreased urine specific gravity. Case 1 also showed severe dehydration in the presence of hyposthenuria. Polyuria and polydipsia are regarded as early signs of lithium poisoning in man[12]. Water deprivation and vasopressin tests were not performed to confirm the presence of the suspected nephrogenic diabetes insipidus. The increased serum potassium seen in Case 1, is not a sign of lithium toxicity in man,

but lithium therapy has been reported as a possible cause of hyperkalaemia in dogs[11].

The serum lithium concentrations seen in these dogs were at the upper human therapeutic range (0,6 mmol ℓ^{-1} to 1,6 mmol ℓ^{-1}) of serum lithium at the laboratory used (I J Van Wyk 1990 Du Boisson and Partners, Sandton, personal communication). Levels of 1,5 mmol ℓ^{-1} have been associated with tremors, weakness, ataxia, agitation, fascicular muscle twitching, vomition and diarrhoea in man[4 12].

Recommended therapy for lithium intoxication in human patients, includes hospitalisation to control seizures, withdrawal of the drug, saline infusion to restore fluid and electrolyte balance, forced diuresis and haemodialysis[4 12]. It is worth noting that permanent renal and neurological defects have been noted in about 10% of human lithium poisoning cases[12].

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Book review/Boekresensie

CANINE PRACTICE THE "IN PRACTICE" HANDBOOKS

E BODEN (EDITOR)

1st Edition. Bailliere Tindall, W.B. Saunders, 24-28 Oval Road, London, NW1 7DX. 1991. pp VIII and 298, 28 tables, 11 diagrams and 168 plates.

This book, as described in the foreword, is a compilation of a variety of aspects of canine medicine and surgery, with comprehensive differential diagnosis lists being afforded to the relevant topics.

Most of the topics are addressed by different authors and range from procedural descriptions (nasolacrimal duct canulation, myelography and urinary catheterisation in the bitch), to indepth evaluations of specific medical and surgical conditions and procedures. Of the latter, this book offers a comprehensive update in the diagnosis and treatment of a variety of syndromes including neonatal disease and its management, and an approach to infertility in the dog and the bitch.

The book likewise serves as a good reference guide, with numerous radiographs detailing contrast studies in the urogenital tract and the spine.

One impediment in the section on autoimmune conditions describes immune-mediated haemolytic anaemia as being the most common cause of haemolytic crisis in the dog. Readers from countries where *Babesia canis* is endemic in the dog population will dispute this claim, however this observation will remain valid for those countries unaffected by the aforementioned parasite.

While the book is recommended for practitioners and students alike, a cautionary note relating to the sections on cervical and thoracolumbar disc surgery is advised. Notwithstanding the apparent ease of the surgical interventions as described, these procedures should remain the domain of a competent and experienced surgeon in possession of the requisite facilities and instrumentation.

This book remains a valuable update on a wide variety of topics and should be recommended to both under- and post-graduate students and practitioners in the field alike.

R.D. Kirkpatrick

LABORATORY ANIMAL BEDDING: A REVIEW OF SPECIFICATIONS AND REQUIREMENTS

F J POTGIETER* and P I WILKE**

ABSTRACT

The literature is reviewed regarding existing specifications and requirements for laboratory animal bedding. The lack of comprehensive specifications in the guidelines of laboratory animal governing bodies, and the introduction of external variables by unsuitable bedding into experimental design, are discussed on the basis of examples from the literature.

Key words: Laboratory animals, contact bedding, specifications, requirements, variables.

Potgieter F.J.; Wilke P.I. **Laboratory animal bedding: A review of specifications and requirements.** *Journal of the South African Veterinary Association* (1991) 62 No. 3, 143-146 (En.) Animal Unit G 20, Faculty of Medicine, University of the Orange Free State, P.O. Box 339, 9300 Bloemfontein, Republic of South Africa.

INTRODUCTION

Laboratory animals are important research tools and every effort should therefore be made to ensure that they are accommodated in an optimal environment. Bedding is an important environmental component which is often ignored, but really needs thorough consideration before experiments are carried out.

Proper care of experimental animals, to uphold normal growth, reproduction and health status, is of the utmost importance during day-to-day husbandry, as well as during experimental procedures. Good laboratory practice will enable both the researcher and animal technician to provide these requirements and at the same time reduce the influence of external factors on experimental results. This produces results which are more consistent and which may mean a reduction in the number of animals required, resulting in more cost-effective research.

The environment of laboratory animals can be divided into the animal room (macro-environment) and the micro-environment in the animal cage. Between these 2 environments, there are major differences which are determined by factors such as the animal cage which acts as a partial barrier between the environments, room heating and ventilation, relative humidity, light intensity, population density, odours and dust. The use of cage filtertops, different types of cage lids and bedding materials can have a marked influence on the micro-environment.

The use of suitable cage bedding and nesting material is an essential husbandry practice that may result in a reduction of stress in animals[13]. It may also result in an improvement in the micro-environment by enhancing hygiene by means of reducing ammonia levels and absorption of moisture[7 20]. Changes in the environment can lead to abnormal biologic responses and so render unreliable results[15 30].

An example of such a change in environment, with major repercussions on experimental results, was reported by Sabine et al.[21]. They recorded a decline, from a virtual 100% to almost 0%, in the incidence of mammary and liver tumours in C3H-A^{vy} and C3H-A^{vy}fB mice, imported to Australia from the National Institute of Health in the United States. The low tumour incidence occurred when the mice were kept on sawdust bedding, derived predominantly from Douglas fir (*Pseudotsuga* spp.) and fed a commercial Australian diet. The high tumour incidence was seen when the animals were reared on the American diet and red cedar (*Juniperus virginiana* Linnaeus) bedding used at the National Institute of Health. Schoental[23] ascribes this phenomenon to a natural estrogen, possibly zearalenone, in the American diet and the lignan podophyllotoxin found in wood shavings from the red cedar.

The adverse effects of variables on experimental results were highlighted when Heston[11], the supplier of the C3H-A^{vy} and C3H-A^{vy}fB mice, replied to the findings of Sabine et al.[21]. He maintained that the decline observed in the occurrence of hepatomas and mammary tumours was related to the condition of the animals. On receiving animals back from Australia, he found them heavily infested with small mites. According to him, this as well as the difference in weight between the American (heavier) and Australian-reared counterparts, could be the only factor responsible for this phenomenon since any factor that decreases growth, would also decrease the occurrence of tumours.

The above-mentioned incident illustrates the importance of also defining and controlling the environmental factors, which are as important as the health and genetic status of the experimental animals. By applying the effect of variables on experimental data, variations in results can be minimised, not only within a specific laboratory, but also between different laboratories.

REQUIREMENTS FOR SUITABLE BEDDING MATERIALS

The type of bedding used is determined by the purpose for which the animals are utilised. For laboratory animals conditions must be optimised in order to eliminate variables, other than those imposed by the experiment[14].

Many different types of contact bedding and nesting materials, have been utilised in the past. This includes processed wood products such as shavings, wool, chips, shreds, filaments and sawdust, paper products, peat moss, cotton, ground corncobs, peanut hulls, hay, and inorganic substances such as attapulgite

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(hydrated magnesium aluminium silicate) and certain clays, and even organic compounds such as polyethylene granules[14]. Some of these have become unpopular for a variety of reasons. Raw wood products, derived from cedar and pine trees for example, contain substances that may interfere with certain enzyme systems which may render this kind of bedding unsuitable for pharmacological studies[5 6 19 29 31 33]. Other examples are hay which is edible, peat moss and used newsprint which tend to stain the animals' coat and attapulgite which is too hygroscopic for some animals[14]. Kraft[14] proposed a number of desirable and self-explanatory criteria for laboratory animal contact bedding (Table 1).

Table 1: **Desirable criteria for laboratory animal contact bedding according to Kraft[14]**

Ammonia binding	Disposable by incineration
Dust free	Fire resistant
Easily stored	Manifests batch to batch uniformity
Inedible	Non-deleterious to cage washers
Moisture absorbent	Non-desiccating to the animal
Nestable	Optimises normal behaviour
Non-malodorous	Readily available
Non-palatable	Relatively inexpensive
Non-staining	Remains chemically stable during use
Non-toxic	Unable to support microbial growth
Non-traumatic	Uncontaminated
Sterilisable	Unlikely to be chewed or mouthed
Deleterious products not formed as a result of sterilisation	
Non-injurious and non-hazardous to personnel	

SPECIFICATIONS FOR LABORATORY ANIMAL BEDDING

It seems that personal preference and what is available in the market place often determines the choice of contact bedding, while the objective should rather be to select a product that will create an optimal environment for the animals and not interfere with the experiment in any way. This trend can also be observed in the rather nonchalant way that specifications for laboratory animal bedding is treated by different regulatory bodies, worldwide.

Although the International Committee on Laboratory Animals[12], the Canadian Council on Animal Care[2], the European convention for the Protection of Vertebrate Animals used for Experimental and Other Scientific Purposes[4] and the Good Laboratory Practices Act[28] of the United States Food and Drug Administration do, directly or indirectly, acknowledge the fact that bedding material is a source of variation in animal experimentation, no definite standpoint on specifications for bedding is taken. The Institute of Laboratory Animal Resources, National Institutes of Health[17 18], do however address the problem to some extent by determining standards for bedding. They describe how beech, birch and maple or any mixture thereof should be processed as well as how the quality assurance is to be done. In contrast, codes governing laboratory animals in South Africa, do not mention bedding materials specifically, except that paraphrases like, "hygienic surroundings" and "animals must be kept in optimal conditions at all times"[26], could be regarded as an indication that bedding material should be used to achieve this goal.

Comparing Kraft's criteria[14] for animal bedding with those set by some other governing bodies, we find that only 5 feature regularly. They are:

a) "Dust free" - dust as found in fine sawdust is contra-indicated in breeding boxes[2], bedding shall be inspected for sandings and dust[17 18] and should be non-dusty[4].

b) "Uncontaminated/non-toxic" - wood products may (i) carry pollutants (insecticides, fungicides, etc.) (ii) introduce disease (particularly mites and tape worms) into the colony, (iii) significantly affect experimentation by influencing response to pharmacologic agents[2], (iv) Bedding should be non-toxic and free from infectious agents or vermin or any other form of contamination, (v) care should be taken to avoid bedding material derived from wood which has been chemically treated[4] and (vi) storage areas should be protected against infestation or contamination[8 28].

c) "Ammonia binding" - must help to control ammonia generation[2].

d) "Non-traumatic" - (i) the type of litter material chosen may exert a considerable influence on the physiological responses of the test animals, (ii) wood shavings have been reported to cause injury to foot pads with eventual granuloma formation in hamsters. Synthetic bedding materials including shredded paper are suggested for these species[2], and (iii) bedding should not interfere with the purpose or conduct of the study[28].

e) "Moisture absorbent" - bedding should be absorbent[4] and it should be changed as often as necessary to keep the animals dry[8].

Regulatory bodies not only omit many of the important criteria for bedding as stipulated by Kraft[14], but those that they do address, are often vague and/or ill-defined.

DISCUSSION

The Canadian Council on Animal Care[2] is rather vague on what kind of wood shavings would induce liver microsomal enzymes, whilst it is a well-documented fact that soft wood (wood derived from the Gymnospermae, especially pine, cypress and cedar) could be a source of organic compounds such as the tricyclic sesquiterpenes, cedrol and cedrene that exercise an adverse effect on the animals' response to certain pharmacologic substances[5 6 19 29 31 33], and the carcinogens coniferaldehyde and sinapaldehyde; constituents of wood lignins[22 24], and podophyllotoxin[23]. From the literature reviewed, hardwood could also present problems, due to the fact that it contains tannins, alkaloids, and lignin[34] (the same constituents encountered in softwoods). Silverman & Adams[25] further confirm the fact that bedding manufactured from hardwood is not the ultimate in laboratory animal bedding as N-nitrosamines, which are carcinogenic to laboratory animals, were detected in 50% of the heat-treated (815°C) hardwood chip bedding samples they examined. Acheson et al.[1], postulated that the aetiological agent, causing nasal cancer in wood workers, could be a constituent or constituents of wood dust that are inhaled and are present in such commonly-used hardwoods as oak and beech. The National Institutes of Health make no specific statement in their specification[17] on the reasons for the use of only hardwood bedding materials but it could with reasonable safety, be deduced that the effect of the organic compounds in softwood, as illustrated by the events with red cedar in Australia[21 23],

moved them to totally ban the use of bedding materials derived from softwood trees. Wood is according to Wirth[34] still the most suitable raw material for animal bedding. Firs and spruce are, according to this author, the best source of raw material for the manufacturing of contact bedding. Species belonging to these two genera *(Abies* and *Pseudotsuga)* however also contain terpenoids such as pinene[3 16 32], limonene[3], carene[26], camphene[3], phellandrene[16] and santene[3]. An oleoresin, i.e., a mixture of mostly resins and essential oils, of the turpentine type is obtained from the common Douglas fir, *Pseudotsuga menziesii* (Mirbel) Franco[16].

Heston's[11] argument on the reasons for the decline in the occurrence of spontaneous tumours might be true, but the effect of the bedding or a constituent in the bedding on the animal is still ignored. Some of the previously mentioned authors[5 19 30 31] have identified the presence of hepatic microsomal enzyme-inducing substances such as cedrene and cedrol, in red cedar shavings. Couldn't this, or another substance such as podophyllotoxin[23], also exercise an effect on the natural occurrence of tumours in mice?

Heston[11] acknowledged the fact that the addition of at least some cedar shavings to the bedding of experimental animals, as a normal husbandry procedure, prevents infestation of the animals by ectoparasites. He, however, does not elaborate on the pesticidal properties of this bedding. The manufacture of "fragrant mothproof" chests from the wood of the white cedar *(Calocedrus decurrens* [Torrey] Florin) and the "insect repellant" properties of the wood from the Lawson cypress *(Chamaecyparis lawsoniana* [A Murray] Parlatore)[3] confirm the presence of such an inherent insecticidal substance in some wood species. A reasonable assumption would be that this substance, possibly a terpenoid, and in the case of Heston's findings, cedrene and cedrol, the 2 main constituents of oil of cedar, could be responsible for this insecticidal action. Irrespective of the beneficial insecticidal properties of red cedar bedding, the active substance would also have exercised an adverse effect on the enzyme system of these animals.

According to Hartwell et al[9], extraction of plant material from red cedar yields 0,10% podophyllotoxin, one of the isolation products of podophyllin. Topical application of podophyllin can, according to them, cure condyloma acuminatum, whilst its isolation products were found to damage experimental tumours. The question thus arises whether housing animals on this type of bedding is acceptable, whilst the occurrence of tumours, either spontaneous or induced, or the progressive changes occurring in vivo during the development of cancer, are studied.

To justify this rather non-specific attitude towards specifications for bedding the assumption could be made that comprehensive knowledge on physical and chemical properties of the different bedding materials in use, does exist. This is totally untrue. Although a fair number of scientific papers mention the type of bedding materials used, most of them, especially with regards to wood, sadly lack precise information about the species of tree(s) utilised as bedding source. The absence of this information plus:

(a) the continuation of the use of sawdust, an undefined byproduct originating from wood, used in the building and furniture industries and thus treated with poisonous wood preservatives such as the tributilic tin compounds, pentachloric phenol and chromium and copper salts,

(b) the utilisation of wood, rich in tannins, alkaloids, hydrocarbons, etc., for the manufacture of bedding materials, thus introducing variables into the experimental model that could exercise adverse effects on experimental results, and

(c) the continued use of vermiculite, especially in South Africa, notwithstanding the evidence that the long-term maintenance of mice on vermiculite causes a reduction in both the number of litters born and their growth rate, and that histological changes occur in the lungs of these animals, similar to industrial pneumoconiosis[10],

are rather indicative of the contrary, or perhaps even of an ignorance regarding the possible ill effects variables could impose on experimental results.

The time has probably arrived to urgently address this seemingly insignificant, but still essential aspect of animal experimentation, to at least impose specific minimum specifications for laboratory animal bedding materials, not only for the benefit of the animals but also to obtain reliable experimental results. Until the materialisation of such specifications, users of this commodity should remember that the majority of modern-day bedding materials are of natural origin and regardless of the advantages or disadvantages of any particular product, contamination of and variability between different batches and types are and remain important factors. These factors should not be ignored during experimental design and the interpretation of results.

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Book review/Boekresensie

A COLOUR ATLAS OF SMALL ANIMAL DERMATOLOGY

G T WILKINSON

Wolfe Medical Publications, Wolfe Publishing Ltd, Brook House, London 1991, pp 272, 513 colour photographs. Price: R146-00 (ISBN 0-7234-1705-9).

"A colour atlas of small animal dermatology" has been compiled with the premise that the eye is the most important diagnostic aid in the diagnosis of skin disorders. It is a colour photographic record of common ectoparasites, and skin lesions and diseases of dogs and cats. A concise caption explains the purpose of each photograph.

The book starts with a valuable contribution on primary and secondary skin lesions as well as a very short section on the configuration of lesions. Skin diseases of the dog and cat are then presented in sections according to the different aetiologies.

Although the majority of photographs are of an acceptable standard, some photographs, in my opinion, need to be replaced in future editions: The photographs of lice infestation of a kitten; tick infestation in a dog; an adult *Sarcoptes scabei* mite and fly myiasis are examples. The inclusion of more photographs in which complete pictures of the patients are given to illustrate the size and distribution of lesions, could only add to the value of the book.

The book may add to the confusion which exists regarding the terminology used in skin disorders: "Allergic perioral dermatitis" is used to describe an allergic reaction to a plastic feeding bowl; and "juvenile pyoderma" to describe a "clinical entity in puppies... aetiology... unknown but initial lesions are allergic in type...". An attempt at standardisation in further editions may be appropriate.

The authors have certainly embarked on a commendable mission. Skin diseases are common in all veterinary practices. The updating and expansion of this text on a regular basis would certainly make it a valuable diagnostic aid in any small animal practice, especially when used in combination with a standard text on veterinary dermatology (as recommended by the author).

J. van Heerden

FELINE HUSBANDRY: DISEASES AND MANAGEMENT IN THE MULTIPLE CAT ENVIRONMENT

NIELS C PEDERSEN

American Veterinary Publications Inc., 5782 Thornwood Drive, Goleta, California 93117 1991 458, numerous figures and tables. Price: $39.00 (ISBN 939674-29-7).

This book is a multi-author text on the economic and careful management of cats. It is stated in the preface to the text that the comparatively thin veneer of domestication and the distinct constitutional nature of domestic cats make them one of the most difficult animal species to keep and breed under modern conditions of confinement and intensification. The book offers 2 approaches to ensure optimum health and reproduction in domestic cats: further domestication by careful selective breeding and the optimisation of conditions in multiple-cat environments. Chapters on the history of cats and cat breeds, genetics and breeding programmes, reproduction and reproductive disorders, common infectious diseases, behaviour, nutrition, toxicology and cattery management are thus presented.

The first chapter includes information on the history and evolution of *Felis catus* as well as a description and illustration of each of the different breeds of cats. The next chapter deals with a description of the basics of genetics, colour variation in the cat, variation in coat hair, physical variation, genetic disorders, developmental anomalies and breeding programmes. This is appropriately followed by a chapter which contains information on reproductive physiology, mating behaviour, vaginal cytology, pregnancy diagnosis, parturition, manipulation of reproduction and reproductive disorders. A major part of the book (126 pages) deals with common infectious diseases of multiple-cat environments. In the introduction to this chapter, basic terminology and the factors which influence the precipitation of clinical signs of disease are very aptly discussed. In the excellent chapter on behaviour, the tremendous importance and popularity of cats as primary pets, are highlighted. The contribution on nutritional disorders could have been improved upon by the inclusion of examples of diets, commercial and home-made, for specific nutritional needs. The diagnosis, treatment and various classes of toxins (rodenticides, insecticides, herbicides, avicides, metals, plants, snake bites, household products and common drugs) are discussed in the chapter on toxicology. The book is concluded with a chapter on housing and management of cats.

The book contains numerous black and white figures, tables and sketches. Each section is concluded with a list of references and the reader is at times encouraged to consult these for more detailed information on a specific topic.

Despite the fact that the book has been orientated largely towards American readers, it is a must for anybody in South Africa with an interest in the domestic cat. It aims to promote good husbandry practices, which as stated by the authors, is a moral obligation for everyone involved in the human-animal interaction.

J. van Heerden

JOURNAL OF THE SOUTH AFRICAN VETERINARY ASSOCIATION

This is a refereed journal. All submissions will be refereed by the Editorial Committee and two independent referees.

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