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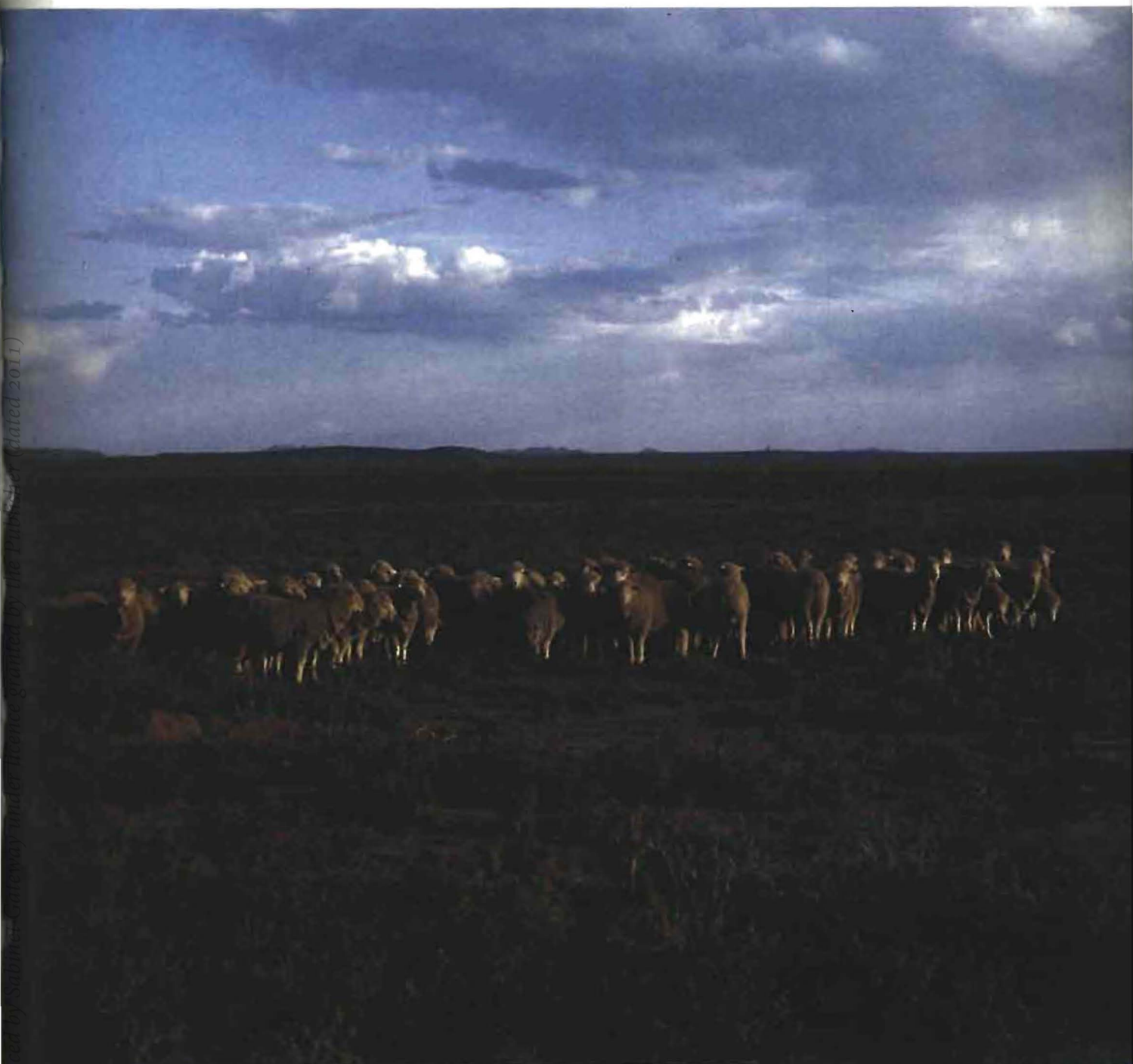

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Contents ● Inhoud

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THE EPIDEMIOLOGY AND CONTROL OF NEMATODE PARASITES AND *OESTRUS OVIS* IN THE WINTER RAINFALL AREA

Van Wyk[15] stated, "Most frequently, drenching recommendations were offered by the various investigators (such as Reinecke & Thomas[12]...) after their surveys to determine the seasonal incidence of nematodes, but these recommendations were not evaluated at all". Subsequently he made the following remarks: "Reinecke has suggested 4 different drenching programmes for the winter-rainfall region (Table 2). One possible reason for his drawing up so many different programmes, is the dearth of information regarding the epidemiology of gastro-intestinal nematodes in this region"[16].

Apart from being contradictory[15 16], the last statement refers to my textbook[7], published in 1983 and no reference is made to our findings published during the past 6 years[3-5, 8-11 13 14] in which total and differential gastro-intestinal worm counts as well as counting and identifying *Oestrus ovis* were done on more than 300 sheep, representing 31% of sheep slaughtered in our trials in the Boland and Overberg from April 1982 to date. These included pregnant and lactating Merino ewes, suckling and weaned lambs, as well as 4-, 6- and 8- week tracers:

The following genera are present: *Teladorsagia (= Ostertagia), Trichostrongylus* and *Nematodirus;* a few *Oesophagostomum* and *Trichuris* and rarely, *Chabertia, Dictyocaulus* and *Muellerius. Haemonchus* is a major genus on spray-irrigated grass legume pastures. It is also present on the west coast (sea mists?) or where there are vleis.

With the exception of hill and mountain fynbos, or arid areas such as Namaqualand, Bushmanland, Little Karoo, etc., sheep graze from autumn to spring, either on permanent dry-land lucerne, lupins or other annual legumes, alternating in the dry summer and early autumn on wheat, oats, barley or lupin stubble. Spray- or flood-irrigated grass/legume or grass (Kikuyu, rye grass, fescue, etc.) pastures are rare (1%) but very intensively grazed (12-30 ewes per hectare) by dual purpose or mutton breeds.

Mean monthly temperatures of 20°C in summer (December - March) are the most important ecological factor ensuring the safety of pastures[10 11 14]. Neither eggs nor preinfective larvae of *Teladorsagia* and *Trichostrongylus* are able to survive and develop to the infective stage.

Some larvae survive in faecal pellets[1] (< 1%) or on pastures over the hot, dry summer in a resting phase and are acquired by grazing sheep, including worm-free tracers when they graze on herbage after autumn or winter rains[10 11 14].

Optimal conditions for infestation of grazing sheep occur from June to October when mean temperatures are less than 20°C and a total of 40mm rain fall on 8 or more d in any 4-week period[14]. During autumn and winter; lambs acquire *Nematodirus,* supplanted by *Teladorsagia* and *Trichostrongyolus* before or after weaning[10 11]. Periparturient relaxation of resistance (PPRR) leads to a massive build-up of infestation in lactating ewes from 6 to 10 weeks after lambing[9 10].

Retarded third (L_3) and fourth (L_4) stage larvae of *Teladorsagia* comprise more than 60% of the total worm population in lambs and older sheep from July to January and these hypobiotic larvae are also dominant in lambs treated either with albendazole (Valbazen, Smith Kline Beecham) or ivermectin (Ivomec, Logos) in winter and spring[3]. Lambs develop resistance to *Nematodirus* by 12 weeks of age, soon after reaching peak infestations, worm burdens falling 43-97% in 18-week and 11-month old sheep respectively. Within 6 weeks to 6 months of sheep grazing on safe pastures, spontaneous cure of *Teladorsagia* occurs, worm burdens falling 77-99% respectively, but not of *Trichostrongylus*[9 11].

O. ovis is endemic on all farms. Flock sheep are infested for 10 to 12 months and tracers for 5 to 9 months of the year. Larvae deposited in autumn are retarded and pupae formed from late April to early August fail to produce flies. Peak infestation are reached in summer and autumn. Strategic treatments are included with anthelmintic treatments in May, August and November (see below).

Control by "dip and dose" must be avoided as this only benefits the pharmaceutical industry, the farmers having spent R73,2 million on anthelmintics in 1989[6]. We have shown that sheep grazing on safe pastures spontaneously expell most of the efficient worm control[3 9-11]. Moreover, mass gains in suckling lambs grazing infested lucerne pastures and treated on 9 August with albendazole and on 28 September with ivermectin in winter and spring were not significant, when compared with controls. On the contrary, mass gains in the flock treated on 26 November with ivermectin and moved to safe wheat stubble on the same day, gained 4,7 kg more than the controls by May of the following year, which was significant ($P < 0,05$). Obviously an efficient anthelmintic alone will only really benefit the sheep if reinfestation is limited to a minimum. Louw's[3] preventive worm control programme, including the treatment for *O. ovis,* limits strategic anthelmintic treatment to 3 treatments per year:

November: ivermectin effective against all stages of *Teladorsagia, Trichostrongylus* and *Nematodirus,* as well as larvae of *O. ovis.*

May/June: levamisole or morantel + closantel or rafoxanide.

August/September: levamisole or morantel + closantel or rafoxanide.

Tests by means of various techniques have shown that *Teladorsagia* are resistant to bensimidazoles (BZ) on 9 different farms. In addition, *Haemonchus* is probably resistant to ivermectin at Vredendal on the west coast and Swellendam. Sales of BZ are 38,5% and combinations of BZ + closantel another 19,4% in the Bredasdorp district alone[5]. In Western Australia BZ resistance has been found on 95% of farms but only 20% of farmers have had resistance tests done[2]. Obviously we need more tests for anthelmintic resistance.

At the Australian Veterinary Association Conference on Perth (March 1989) Anderson stated that all anthelmintics will, sooner or later, be found to be ineffective against resistant strains of various nematodes and we are merely putting off the evil day when no drugs will be effective.

I see only 2 possible solutions: either successful vaccines, or genetic selection of sheep which are resistant to worms.

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To the editor/Aan die redakteur

THE EPIDEMIOLOGY AND CONTROL OF NEMATODE PARASITES AND *OESTRUS OVIS* IN THE WINTER RAINFALL AREA

Reinecke does not say in what way my papers[6 7] are contradictory. However, I contend that, despite the great deal of work that has been done recently by him and co-workers in the south-western Cape, my statement on the lack of proof on the efficacy of any one of the control programmes that are being recommended at present in South Africa, is as valid today as it was in 1985[6]. The programme suggested by Louw[1] for that region, differs from the one that was tested by him. Thus, as before, a programme is being offered on the strength of epidemiological data, and not on controlled trials to compare the efficacy of the recommendations in treated and control groups of sheep grazing throughout on similar, but separate pastures. However, when making this statement, I realise that[6] it will be very difficult to test such a programme under realistic field conditions in South Africa.

Because most of the grazing in the RSA consists of natural pasture, or unirrigated improved pasture, realistic field testing of control programmes should mostly entail the use of multiple field camps that can be paired off very well as regards vegetation and soil type, topography, etc., and these may be extremely difficult and expensive to obtain. If possible, provision should be made for pasture spelling in the test. Obviously, this would complicate matters even further, and the failure to test the worm control programmes being recommended here today, probably stems from the many problems concerned in obtaining facilities and funds for such work. On the other hand, as stated it is better to base control programmes on extrapolations from the knowledge of the epidemiology of each worm species, than on no information at all of either their epidemiology or seasonal incidence. In addition to the above considerations, as discussed later, I contend that Louw's trial, to which Reinecke refers and which contains the suggested drenching programme[1] for the south-western Cape, was not adequately controlled, as the groups of sheep did not graze similar pastures throughout the trial.

My paper of 1990[7] was submitted for publication before the papers having a bearing on drenching programmes for the region under discussion[1 2 3 4 5] were published. At the time of my last editing of the paper, I should have included Louw's suggested programme, but I had seen much of the data from the Cape previously, and at the time it slipped my mind that Louw's paper, which contains the drenching programme to which Reinecke refers, had already appeared in print. On the other hand, it does not affect my statements that

a) Reinecke's 4 different programmes that were listed by me were probably the result of a dearth of information; and,
b) None of the recommended programmes had been tested satisfactorily in the field.

Reinecke refers to the fact that Louw[1] is supposed to have shown a statistically significant difference of 4,7 kg in mass between groups of lambs. However, in my opinion it is not valid to apply a statistical test to the mass gains of the 2 groups in question, as they are not comparable, not having grazed similar pastures throughout: On p 187 and 188 of Louw's paper the following is stated:

a) "27 September 1987 — Moved control animals to rested lucerne pasture..." Presumably the treated animals remained on the previous pasture, and, apparently, the controls remained on the new pastures for 2 months until all of the lambs were moved to stubble pasture.

   It can be argued that the rested pasture may have had a lower level of infestation than that on which the lambs had grazed previously, but this fact was apparently not proved, and, hence, cannot be accepted as being true, therefore remains an unknown factor that may have had a bearing on the results of the trial.
b) "25 February 1988 — Moved control lambs to lucerne pasture. ... 24 March 1988 — returned control lambs to wheat stubble." Here is another period of almost a month in which the controls grazed on totally different pasture from the treated lambs. Once again, there is no indication of the comparative nutritive value or the levels of parasite infestation of the 2

different pastures, and any statistical comparison of the 2 groups is, in my opinion, not valid.

c) "24 March 1988 ... All lambs received lucerne, hay and grain supplements daily." In my opinion it is important to know what amounts of supplementary fodder were offered, whether the different groups of lambs had similar intakes of the supplements, and whether they were fed under similar conditions (e.g. in troughs, or fodder spread on the ground, etc.). If they were, for instance fed most of their nutrients in this way, then the effect of the stubble lands may have been almost immaterial to the outcome of the trial.

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THE BENT-LEG SYNDROME IN SHEEP II. ASSOCIATION WITH GENETIC MARKERS

F E VAN NIEKERK*, C H VAN NIEKERK**, G REID*** and J COETZEE****

ABSTRACT

Comprehensive blood typing (17 factors) and electrophoretic protein markers (haemoglobins, transferrins and albumins) were determined on S.A. Mutton Merino sheep (n=275). The frequencies of these genetic markers were compared between ram lambs with normal and ram lambs with bent-legs and in a second trial between non-affected ewes and their lambs and ewes and lambs which were considered to be carriers of genetic factors resulting in the bent-leg syndrome. The presence of blood factors 2, 3 and 13 and the absence of factors 8, 10 and 17 is possibly linked to the bent-leg syndrome. Although the frequencies of TF^A and TF^D alleles were higher in the suspected carrier animals than non-affected animals, no definite linkage to the bent-leg syndrome was found. Haemoglobin and albumin type showed no correlation with the bent-leg syndrome.

Keywords: Blood factors, electrophoretic markers, bent-leg syndrome, sheep

Van Niekerk F.E.; Van Niekerk C.H.; Reid G.; Coetzee J **The bent-leg syndrome in sheep II. Association with genetic markers.** *Journal of the South African Veterinary Association* (1990) 61 No. 4, 151-154 (En.) Elsenburg Agricultural Centre, Private Bag, 7606 Elsenburg, Republic of South Africa.

INTRODUCTION

Recent studies have indicated that ram lambs of certain ewes are more receptive to the development of the bent-leg syndrome[20]. The main cause of this syndrome was thought to have been a nutritional (mineral) imbalance or deficiency[19]. Van Niekerk et al[20], however, found that up to 15% of the ewes whose ram lambs developed this syndrome again gave birth to lambs with this syndrome in following years. In some cases where the ewes had twin ram lambs, both developed this syndrome which indicates that this condition most probably has a genetic origin.

In swine, a similar condition to bent-legs, which is generally referred to as leg weakness, causes severe economic losses. According to studies, the heritabilities of leg weakness and osteochondrosis are low to moderate (0,1-0,3)[6]. As in sheep[20], no association was found between growth rate or the performance index of boars with leg weakness[21].

Blood groups have been used by Nguyen & Bunch[10] for specie identification as well as for frequency analysis for breed characteristics[11] and correlations with other metabolites and physiological functions[18]. A similar study was undertaken by Collis & Milson[3] where the transferrin system in Herdwick sheep was investigated with special reference to the susceptibility of the flock to experimentally produced scrapie.

Certain productive and reproductive parameters could be positively linked to genetic markers. Rasmussen & Tucker[12] indicated that Finnish Landrace ewes of transferrin type BD had smaller litters than ewes of other types. The variation in serum alkaline phosphatase activity found in sheep, is controlled to a large extent by the genotype for the R-O-i blood groups[13]. This variation in alkaline phosphatase activity might affect the incidence of the bent-leg syndrome[19].

By using genetic markers, the present study aimed at identifying at an early age, sheep which could develop the bent-leg syndrome. Therefore, comprehensive blood typing which included the determination of 17 blood group factors and 3 electrophoretic markers, was carried out on 275 S.A. Mutton Merino sheep. The breeding history as well as data concerning the incidence of the bent-leg syndrome in the lambs of this flock over the past 8 years, were available.

MATERIALS AND METHODS

Trial 1:

During the 1987 lambing season, blood samples were collected from S.A. Mutton Merino ram lambs (n=20) with normal legs and with bent-legs (n=9) at the age of approximately 8 months. The lambs with the normal legs were chosen at random from the flock.

Trial 2:

During the 1988 lambing season when the lambs were approximately 160 d of age, blood samples were collected from both the weaned lambs and their dams. Blood samples were also taken from the 3 breeding rams. Only S.A. Mutton Merino sheep were included in this study. The management and feeding of this herd has been described previously[19]. For the purpose of this experiment, the ewes whose progeny never contracted this syndrome, are referred to as "normal" animals. The ewes of which some of the progeny developed the bent-leg syndrome, and which might therefore be carriers of certain genetic factors which eventually could result in this syndrome, are referred to as "carrier" animals.

Blood sampling and preparation

Two blood samples, one in EDTA (5 ml) and the other without anti-coagulants (7 ml) were taken from the jugular vein of each sheep in vacuum tubes (Vac-U-Test, Radem Laboratories). The sample collected in EDTA was placed in a second tube with dextrose as an energy source and sodium cyanide as anti-bactericide. The other sample was left for 5h after collection to clot. It was then centrifuged and the serum placed in a tube containing boric acid.

Blood typing procedures

Erythrocyte antigen determinations were

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Table 1: **The frequency of blood factors of ram lambs (Trial 1)**

Blood factors	Normal legs n = 20	Bent-legs n = 9
1	-	-
2	0,60	0,89
3	0,50[a]	0,89[b]
4	-	-
5	-	-
6	0,45	0,44
7	0,40	0,55
8	0,35	0,55
9	-	0,11
10	0,60	0,77
11	0,30	0,22
12	0,75	0,66
13	0,55	0,77
14	0,55	0,55
15	not tested	not tested
16	not tested	not tested
17	not tested	not tested

[a,b]Values in the same row with different headings, differ significantly ($P \leq 0,05$)

build-up reaches a titre of 1/1000 the recipient is bled, the serum centrifuged and stored at -20°C. This serum often contains more than one antibody type and is thus purified, using absorptions to remove the unwanted antibodies, isolating a single antibody. The resulting serum, containing a single antibody, is then referred to as a reagent. For these tests, 17 different reagents were used, each identifying a specific factor. These factors are divided into systems as described by Tucker & Ellory[17]. The blood was washed 3 times with isotonic saline to remove all the plasma. The remaining red blood cells were diluted to give a 1% solution and then added to the reagents. Absorbed rabbit serum, referred to as complement, was used as a catalyst in these tests. After the reagents (2 drops), blood (1 drop) and complement (1 drop) were mixed, the test plates were placed on a shaker for 2,5 h at 30°C to keep the red blood cells in suspension. The cells were then allowed to settle for a further 1,5 h at the same temperature whereafter they were read macroscopically using a mirror. If the sheep had a specific factor (antigen) on its erythrocyte membrane, it would react positively for this factor.

Because only 21 blood factors are presently recognised in sheep, use was also made of electrophoretic protein markers. Three different markers were determined, namely, haemoglobins, transferrins and albumins as was done by Efremov & Braend[5]. The original techniques of Smithies[14,15] for horizontal starch gel electrophoresis were used.

The electrophoretic technique for the haemoglobin determinations was done as described by Buschmann & Schmid[2]. No staining was required as the haemoglobin bands are easily visible.

Serum albumins were separated using the methods described by Ashton[1] and Kristjansson[9] after small modifications in the pH of both the gel buffer and electrolyte. Staining was done by the method of Johns[8].

Transferrins were separated using the method of Kristjansson[9]. Modifications of this technique were necessary to acquire good separation in the number of closely-situated bands. In the gel buffer, cacodylic acid was substituted with citric acid and the optimum pH was found to be 8,60, whilst the optimum electrolyte pH was found to be 8,08. Staining was done according to the method of Johns[8]. Frequencies of blood-factors in normal and bent-leg sheep (Trial 1) and normal and carrier sheep (Trial 2) were compared by Chi-square methods, using the P4F programme of the BMDP statistical packet[4].

Table 2: **The haemoglobin phenotypic distribution and gene frequency of ram lambs (Trial 1)**

Group	Number of animals	Phenotypes AA	AB	BB	Gene frequency Hb^A	Hb^B
Normal	20	1	4	15	0,15	0,85
Beng-legs	9	-	2	7	0,12	0,88
Previous study[16] S.A. Mutton Merino	288				0,18	0,82

done using haemolysis testing as described by Tucker[17]. The necessary blood factor reagents were self-produced (ADSRI, Irene), as they are not commercially available. This production is based on iso-and hetero-immunisation, resulting in antibody production. When the antibody

Table 3: **A comparison of the gene frequency of transferrin types of ram lambs (Trial 1)**

Transferrin type	Normal legs n = 20	Bent-legs n = 9	Previous study[16] S.A. Mutton Merino n = 288
TF^A	0,45	0,44	0,47
TF^B	-	-	0,02
TF^C	0,12	0,06	0,15
TF^D	0,28	0,28	0,27
TF^E	0,15	0,22	0,09

RESULTS

The frequencies of the blood factors of the 29 ram lambs of Trial 1 conducted in 1987, are recorded in Table 1. Reasonable differences were found in the frequencies of 4 blood factors namely 2, 3, 8, and 13 of which only factor 3 differed significantly ($P \leq 0,05$).

The phenotypic distribution and gene frequency of the haemoglobin types (Table 2) were similar for ram lambs with normal limbs and those with bent-legs. Transferrin gene frequencies did not differ between lambs with normal limbs and those with bent-legs (Table 3).

The frequencies of the blood factors of the adult ewes and ewe and ram lambs (Trial 2) of both the normal and carrier animals, are recorded in Table 4. Although the frequencies of factors 8, 10 and 17 tended to differ between normal and carrier ewes and ewe lambs, these differences were much more evident in the rams. In the ram lambs, the frequency of factors 8 ($P \leq 0,06$), 10 ($P \leq 0,05$) and 17 ($P \leq 0,05$) differed by more than 0,3 between normal and carrier animals which was higher than that of both the adult ewes or ewe lambs. Unfortunately, blood groups of only 2 of the 3 rams used for breeding were determined. In the case of ram WTM, only factors 12 and 13 were present, while in ram BRS, factors 3, 4, 10, 12, 13, 14 and 15 were recorded.

Haemoglobin phenotype as well as the gene frequencies are recorded in Table 5. No significant differences between

Table 4: **A comparison of the frequency of blood factors between adult ewes, ewe and ram lambs of normal and carrier sheep**

Blood factor	Adult Ewes		Ewe lambs		Ram lambs	
	normal n = 84	carrier n = 12	normal n = 64	carrier n = 8	normal n = 62	carrier n = 11
1	0,559	0,666	0,468	0,500	0,596	0,636
2	0,619	0,750	0,187	0,250	0,290	0,272
3	0,571	0,500	0,781	0,500	0,806	0,727
4	0,440	0,333	0,766	0,500	0,790	0,636
5	0,023	0,083	0,031	0,125	0,016	-
6	0,523	0,583	0,437	0,375	0,580	0,545
7	0,523	0,583	0,375	0,250	0,580	0,545
8	0,607	0,416	0,406	0,375	0,483[a]	0,181[b]
9	0,095	0,166	0,093	0,250	0,016	-
10	0,750	0,750	0,703	0,500	0,677[a]	0,363[b]
11	0,619	0,500	0,297	0,125	0,483	0,363
12	0,643	0,917	0,781	1,000	0,838	0,909
13	0,226	0,333	0,250	0,250	0,306	0,272
14	0,750[a]	0,583[b]	0,703	0,375	0,693	0,545
15	0,273	0,500	0,593	0,375	0,483	0,363
16	0,011	-	0,015	-	-	-
17	0,547	0,333	0,515	0,375	0,419[a]	0,091[b]

Factor 8 — [a,b]Values in this group differ significantly ($P \leq 0,06$)
Factors 10, 14, 17 - [a,b]Values in these groups differ significantly ($P \leq 0,05$)

Table 5: **Distribution of haemoglobin types of adult ewes, ewe and ram lambs of normal and carrier sheep**

Group	Number of animals	Phenotypes			Gene frequency	
		AA	AB	BB	Hb^A	Hb^B
Ewes (normal)	84	-	12	72	0,071	0,928
Ewes (carrier)	12	-	3	9	0,125	0,875
Ewe lambs (normal)	64	4	19	41	0,211	0,789
Ewe lambs (carrier)	8	-	5	3	0,312	0,688
Ram lambs (normal)	62	3	25	34	0,250	0,750
Ram lambs (carrier)	11	-	5	6	0,227	0,773

Table 6: **Comparison of the transferrin gene frequencies in adult ewes, ewe and ram lambs of normal and carrier sheep**

Transferrin type	Adult ewes		Ewe lambs		Ram lambs	
	normal n = 84	affected n = 12	normal n = 64	affected n = 8	normal n = 62	affected n = 11
TF^A	0,476	0,542	0,515	0,625	0,508	0,545
TF^B	0,029	-	0,008	-	0,008	-
TF^C	0,137	0,125	0,070	0,063	0,065	0,045
TF^D	0,204	0,292	0,289	0,312	0,338	0,365
TF^E	0,154	0,041	0,118	-	0,081	0,045

groups of 'normal' and 'carrier' animals were evident. Transferrin gene frequencies are recorded in Table 6 and transferrin phenotypes in Table 7.

Twenty nine per cent of the carrier ewes were of the homozygous type DD compared to 6% of the normal ewes. All the carrier sheep either had a TF^A or TF^D allele.

Albumin type was found to be monomorphic for both trials and was therefore disregarded in the allele frequency analysis.

DISCUSSION

Van Niekerk et al[19 20] hypothesised that high plasma phoshorus concentrations which resulted in an inverse plasma Ca:P ratio, as found in the carrier ewes, is a contributing factor in the development of the bent-leg syndrome and is possibly caused by genetic factors. The linkage of these genetic factors to certain blood group(s) or electrophoretic markers, would make the identification of 'carrier' sheep an easy task.

This flock can be regarded as a free-mating population as ewes are mated at random to rams and therefore differences in the frequencies of blood factors would not be expected. The results of the 2 trials were not in agreement. In Trial 1, the frequencies of blood factors 2, 3, 8 and 13 were more than 20% higher in rams with bent-legs compared to normal rams. As for Trial 2, blood factors 8, 10 and 17 seem to be linked to the bent-leg syndrome as the frequencies of these blood factors were more than 30% lower than that of the normal rams.

A further complicating factor is that it is not known whether it is the presence or absence of a certain blood factor or a combination thereof, if at all, which causes this syndrome. The possibility that this 'bent-leg factor' can be sex-linked, must not be overlooked.

By taking the results of both trials into consideration the presence of blood factors 2, 3 and 13 and the absence of 8, 10 and 17, or combinations thereof, seem to be possibly linked to the bent-leg syndrome.

Haemoglobin type can be disregarded as a contributing factor in the development of the bent-leg syndrome.

Comparing the transferrin gene frequencies of adult ewes, ewe and ram lambs of normal and carrier animals (Table 6) revealed that TF^A and TF^B were the major TF alleles found in this flock. In both trials, only 5 of the 11 possible alleles were found, these being A, B, C, D and E. All 3 the rams used for breeding were of transferrin type AD and it was therefore expected that the frequency of the TF^A and TF^D alleles could be higher in the offspring than in the ewes.

Table 7: **Transferrin phenotypes of normal and carrier sheep**

GROUP		TRANSFERRIN PHENOTYPES												
		AA	AB	AC	AD	AE	BD	BE	CD	CE	DD	DE	EE	TOTAL NUMBER
Normal ewes		15	4	12	19	15	-	1	5	5	3	4	1	84
Normal ewe lambs		17	1	7	14	8	-	-	3	-	6	6	-	62
Normal ram lambs		17	-	1	22	6	2	-	5	-	6	3	-	62
Carrier ewes		4	-	2	2	1	-	-	1	-	2	-	-	12
Carrier ewe lambs		2	-	1	4	-	-	-	-	-	5	-	-	12
Carrier ram lambs		3	-	1	4	1	-	-	1	-	1	-	-	11
Normal	Ewes	32 (22)	5 (3)	19 (13)	33 (23)	23 (16)	--	1 (0,6)	8 (5)	5 (3)	9 (6)	10 (7)	1 (0,6)	146
	Rams	17 (27)	-	1 (0,1)	22 (35)	6 (9)	2 (3)	-	5 (8)	-	6 (9)	3 (5)	-	62
Carrier	Ewes	6 (25)	-	(12)	6 (25)	1 (4)	-	-	1 (4)	-	7 (29)	-	-	24
	Rams	3 (27)	-	(9)	4 (36)	1 (9)	-	-	1 (9)	-	1 (9)	-	-	11

Numbers in parenthesis indicate the % of the total number

The ewes (adult and lambs) were grouped, and comparing the numbers it became evident that phenotypes AA, AD and DD were of special interest. Type AA varied very little between the 4 groups, while in the case of type AD, ram lambs had an 11% higher frequency of this phenotype compared to ewe lambs of both the normal and carrier ewes. The fact that all 3 rams used in this flock were of type AD, might have contributed to this.

Despite results of Trial 1, there were indeed indications that the frequency of TF^A and TF^D alleles were higher in carrier sheep than in normal animals. The frequency of transferrin phenotype DD was extremely high in carrier ewes (29%) when compared to that of normal ewes (6%). Hidiroglou et al[7] suggested that high dietary iron intakes could act as a conditioning factor in osteodystrophic conditions in sheep. Although plasma iron concentrations were not determined in this trial it can, however, be accepted that transferrin types have an influence on this parameter. Both haemoglobin and transferrin frequencies of the normal rams correlate well with a previous study of S.A. Mutton Merino sheep[16].

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SELECTED LABORATORY PARAMETERS OF THOROUGHBREDS

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ABSTRACT

Selected haematological, blood chemical and serological variables were investigated in healthy Thoroughbreds (n=45) in training. Haemoglobin concentration, haematocrit, red, white and differential cell counts as well as serum concentrations of total and ionized calcium, sodium, potassium, chloride, urea, creatinine, total protein, albumin, inorganic phosphorus, total bilirubin, iron, glucose, magnesium, alkaline phosphatase, gamma-glutamyltransferase, lactate dehydrogenase, aspartate transaminase, alanine transaminase and creatine kinase were found to be within ranges previously reported for horses. No statistically significant difference was found between the haematocrit (Ht) of horses (n=44; $\bar{x}$=0,44; SD=0,02) of different performance or between those of different age groups. A significant difference was found between the Ht of males ($\bar{x}$=0,43; SD=0,02) and females ($\bar{x}$=0,45; SD=0,02) and between quiet ($\bar{x}$=0,44; SD=0,02) and excitable ($\bar{x}$= 0,46; SD= 0,02) horses. No significant difference in red cell potassium concentration was found between horses of different performance. Cortisol, insulin, parathormone (C-terminal), aldosterone and folate concentrations respectively varied between 89-204 ($\bar{x}$=144,4; SD=25,47) nmol ℓ^{-1}, 4,2-23 ($\bar{x}$= 10; SD=4,30) m U ℓ^{-1}, 65,2-91,4 ($\bar{x}$=79,46; SD=9,34) pmol ℓ^{-1}, less than 138 to 379 pmol ℓ^{-1} and 9,4-21,5 ($\bar{x}$=14,5; SD=2,87) nmol ℓ^{-1}. Vit B_{12} concentrations exceeded 1 400 pmol ℓ^{-1}. Blood lead concentrations in all animals were below 15 ug ℓ^{-1}. Fifteen (33,3%) of the horses were carriers of babesiosis. Laboratory findings concerning these horses did not differ from those of the other horses.

Key words: Thoroughbreds, haematology, blood chemistry, hormones, lead, *Babesia equi*, *Babesia caballi*, serology

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INTRODUCTION

Laboratory aids are extensively employed in the diagnosis of disease, in preventive medicine and as management tools. Reference values, although ideally established for a specific laboratory, of clinically sound and normal performing animals, serve as a guide to the clinician in evaluating parameters from specific animals. Apart from the investigations by Neser in 1923[10] and scant reference to selected clinical pathological variables[3,4], we could find no published record of normal haematological and blood chemical parameters of Thoroughbreds in the Republic of South Africa. Preliminary serological investigations in mainly cold-blooded horses and free-living equidae have indicated that a relatively large percentage of these animals are carriers of either *Babesia equi* or *B. caballi* or both (De Waal & Van Heerden, 1986-1989 unpublished data). The incidence of such a carrier-status in local Thoroughbreds, which may have an influence on certain laboratory parameters, is unknown.

The aim of this investigation was to establish selected baseline haematological and blood chemical parameters as well as to establish the prevalence of a carrier-status of *B. equi* and *B. caballi* in a group of local Thoroughbreds.

MATERIALS AND METHODS

Thoroughbreds (n=45) in very good physical condition, in full training, stabled and trained at an altitude of 2 000 m above sea level, were bled by venipuncture of the jugular vein between 06:00 and 08:30. All horses, which included stallions and geldings (n=27) and mares (n=18), 2-6 years of age (2-3 years: n=23; 4-6 years: n=22) were starved for a period of at least 8 h prior to bleeding. The animals were bled in their respective stables after simple manual restraint with a halter and within 30 s of having entered the stable. Collection of blood specimens was completed within 3 min. Horses were classified by the trainer according to their temperament (calm, excitable or hyperexcitable) and their racing performance (poor, average, good and excellent).

Blood specimens were collected in evacuated tubes (Vac-u-test, Radem Laboratory Equipment, Wijnberg). Separate specimens for red cell potassium and lead determination were collected in heparinised specimen tubes (L.H. 3 200 Vacutainers™, Becton-Dickinson, Rutherford, New Jersey 07070) and stored at 4°C for 12 h until analysed in a batch. For ionized calcium, a 5 ml plastic syringe, previously heparinised with heparin supplied by the manufacturer of the ionized calcium analyser (Radiometer A/S, Copenhagen), was used to collect a separate venous specimen. The syringe was stoppered to keep the specimen anaerobic until ionized calcium was measured, approximately 30 min later. Blood for glucose samples was collected in evacuated oxalate/sodium fluoride tubes (Vac-U-Test).

Blood was collected in EDTA-tubes for the determination of haemoglobin concentration (Coulter Haemoglobinometer, Coulter Electronics, Hialeah, USA). The Ht was performed with a Heraeus-Christ Haemofuge Cat No 775 (Heraeus-Christ, West Germany).

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Table 1: **Haematocrit, red and white cell count, and haemoglobin concentration in Thoroughbreds**

	n	$\bar{x}$	SD	range
Haemoglobin g ℓ^{-1}	44	175	12,04	147-205
Red cell count x 10^{12} ℓ^{-1}	44	8,20	0,75	6,46-9,66
Haematocrit (all horses)	44	0,44	0,02	0,38-0,50
females	17	0,45	0,02	0,41-0,50
males	27	0,43	0,02	0,38-0,50
2-3 year olds	22	0,44	0,02	0,38-0,50
4-6 year olds	22	0,44	0,03	0,40-0,50
quiet horses	29	0,44	0,02	0,38-0,50
excitable horses	15	0,46	0,02	0,46-0,50
White cell count x 10^{9} ℓ^{-1}	44	8,63	1,57	5,7-12,1
Neutrophils (mature) %	44	61,59	7,45	49-79
Neutrophils (immature) %	44	1,63	2,23	0-12
Lymphocytes %	44	30,27	7,44	15-44
Monocytes %	44	3,84	2,53	0-10
Eosinophils %	44	2,20	1,77	0-8
Basophils %	44	0,43	0,66	0-2

Serum was analysed for sodium (Na), potassium (K), chloride (Cl), inorganic phosphorus (P), albumin (Alb), total protein (TP), urea, creatinine and enzyme activities of alkaline phosphatase (ALP), alanine transaminase (ALT), aspartate transaminase (AST), gamma-glutamyltransferase (GGT), creatine kinase (CK) and lactate dehydrogenase (LDH) while plasma was analysed for glucose concentration using previously published methods[13].

Table 2: **Electrolyte, urea, creatinine, total protein, albumin, bilirubin, iron, glucose, enzyme, cortisol, insulin, parathormone and folate concentrations in Thoroughbreds**

	n	$\bar{x}$	SD	range
Total calcium mmol ℓ^{-1}	45	2,99	0,078	2,84-3,19
Ionized calcium mmol ℓ^{-1}	45	1,62	0,05	1,55-1,75
Sodium mmol ℓ^{-1}	45	138,6	1,75	134-143
Potassium mmol ℓ^{-1}	45	3,54	0,286	3-4,4
Chloride mmol ℓ^{-1}	45	102,8	2,417	98-108
Urea mmol ℓ^{-1}	45	5,69	0,912	3,9-8,2
Creatinine μmol ℓ^{-1}	45	151,08	20,459	123-222
Total protein g ℓ^{-1}	45	60,31	2,88	55-67
Albumin g ℓ^{-1}	45	36,91	1,68	33-42
Inorganic phosphorus mmol ℓ^{-1}	45	1,20	0,151	0,93-1,56
Total bilirubin μmol ℓ^{-1}	45	46,17	13,34	18-81
Iron μmol ℓ^{-1}	45	28,37	5,21	18,2-42,5
Red cell potassium mmol ℓ^{-1}	44*	104,79	7,25	86,4-115,0
Glucose mmol ℓ^{-1}	45	5,16	0,32	4,5-6,4
Magnesium mmol ℓ^{-1}	45	0,71	0,04	0,6-0,87
Alkaline phosphatase U ℓ^{-1}	45	297,8	85,59	141-516
Gamma-glutamyltransferase U ℓ^{-1}	45	23,4	11,7	2-75
Lactate dehydrogenase U ℓ^{-1}	44**	164,3	41,05	104-287
Aspartate transaminase U ℓ^{-1}	44**	232,7	82,54	139-652
Alanine transaminase U ℓ^{-1}	44**	10,3	4,7	5-34
Creatine kinase U ℓ^{-1}	44**	62,6	15,94	37-98
Cortisol nmol ℓ^{-1}	45	144,4	25,47	89-204
Insulin mU ℓ^{-1}	45	10,0	4,30	4,2-23
Parathormone pmol ℓ^{-1}	13***	79,46	9,34	65,2-91,4
Folate nmol ℓ^{-1}	27***	14,5	2,87	9,4-21,5

*one specimen was unsuitable for analysis

**outlying values of LDH (1572), AST (3 300), ALT (491) and CK (510) in one horse were not included

***restricted numbers of specimens were analysed for economic reasons

Ionized calcium concentrations were measured with an ICAl ionized calcium analyser (Radiometer A/S, Copenhagen) utilising an ion-selective electrode. Total serum calcium and magnesium concentrations were analysed with an atomic absorption spectrophotometer (Model 5 000 Perkin-Elmer Corp.)

Red blood cell potassium concentrations were determined with an Astra 8 (Beckman Instruments Inc; Brea California) according to the method described by Muylle et al[8].

Hormone and vitamin assays were conducted by means of diagnostic reagent kits using human antibodies: insulin (Phadeseph, Pharmacia Diagnostics, Upsala, Sweden); cortisol (GammaCoat (^{125}I) Cortisol, Baxter Travenol Diagnostics Inc., Cambridge, MA, USA), aldosterone (Compagnie Oris Industrie SA, St-Quentin-Yvelines Cedex, France) and parathyroid hormone (C-terminal PTH, Instar Corp., Stillwater, Minnesota, USA). Vitamin B_{12} and folate were determined with a combined radio immuno-assay (RIA) method (Quantaphase, Bio-Rad Laboratories, Hercules, California USA).

Whole blood lead concentrations were determined with the micromethod described by Fernandez & Hilligoss[5]. This method utilises an atomic absorption spectrophotometer (Model 5 000 Perkin-Elmer Corp., Norwalk, Conn. USA 06856) equipped with a graphite furnace (Model HGA 500, Perkin-Elmer) and automatic sampler (Model AS-40, Perkin-Elmer). The spectrophotometer was calibrated, using lead standards prepared in ammonium dihydrogen phosphate in an acid medium.

Thin blood smears were prepared from blood collected in evacuated EDTA-tubes and fixed and stained by the Diff-Quick method (Cam's-Quick Stain, C.A. Milsch, Krugersdorp) for examination for the presence of parasites.

The indirect fluorescent antibody test was performed on serum samples as described by Maddan & Holbrook[7]. Anti-horse IgG (AFY-conjugate, Bio-maker, Kiryat Weuzmann 76326, Rehovot, Israel) was used. Each sample was tested against *B. caballi* and *B. equi.*

The Mann-Whitney test (a non-parametric test) was used to compare the mean values of the Ht between calm and excitable to hyperexcitable horses, between horses with a poor to average and

Table 3: **The results of examination of blood smears and indirect fluorescent antibody test (IFAT) titres to *Babesia equi* and *B. caballi* in Thoroughbreds (only positive findings are presented)**

No. of horse	*B. caballi* IFAT titre			*B. equi* IFAT titre			Smear
	1/20	1/40	1/80	1/20	1/40	1/80	
1	+	-	-	+	+	+	Negative
6	-	-	-	-	-	-	Positive
9	+	-	-	+	+	+	Positive
13	+	+	+	-	-	-	Negative
18	-	-	-	+	+	+	Negative
23	-	-	-	+	+	+	Negative
26	-	-	-	+	+	+	Negative
28	-	-	-	+	+	+	Positive
30	-	-	-	+	+	+	Negative
31	-	-	-	+	+	+	Negative
35	-	-	-	+	+	+	Negative
37	-	-	-	-	-	-	Positive
39	-	-	-	+	+	+	Negative
41	-	-	-	+	+	+	Negative
43	-	-	-	+	+	+	Negative

* conjugate dilution was 1/80; a titre of 1/80 is regarded as positive
\+ All smears described as positive, demonstrated *B. equi* parasites only

good to excellent performance, between horses 2-3 and horses 4-6 years of age and between male and female horses. The test was also used to compare the mean values of the red cell potassium concentration between horses with a poor to average, and horses with a good to excellent performance, between horses with a calm and horses with an excitable to hyperexcitable temperament, and to compare the mean values of the serum cortisol concentration between horses with a calm and horses with an excitable to hyperexcitable temperament. The Krushall-Wallis test was used to compare the average PCV of horses bled at different half-hour intervals. Differences were regarded as significant when P-values were less than 0,05.

Investigated laboratory parameters of horses with babesiasis were compared with those without serological evidence of babesiasis and without a positive blood smear.

RESULTS

Sixteen horses were rated as either excitable or hyperexcitable and 29 as calm. Some of the horses classified as excitable or hyperexcitable, tensed up during the collection of blood specimens. The rest of the horses appeared very calm. The racing performance of the horses was described as poor to average (n=22) and good to excellent (n=23).

Haematological parameters are presented in Table 1. No statistically significant difference in haematocrit was found between the averages of horses collected at different 0,5 h intervals, between the different age groups and between horses of different performance. A significant difference was found between the different sexes and between horses of different temperament (Table 1).

Haematological parameters in horses with serological evidence of babesiasis or with positive blood smears, did not differ from horses without babesiasis.

The results of blood chemical, hormonal and vitamin assays are presented in Table 2. There were no differences in the values of these parameters between horses with and horses without babesiasis. No statistically significant difference in red cell potassium concentration was found between horses with poor to average performance and horses with good to excellent performance, and between calm and excitable to hyperexcitable horses.

No statistically significant difference was found between serum cortisol concentrations in calm and excitable to hyperexcitable horses.

Aldosterone concentrations in 40 horses were less than 138 pmol ℓ^{-1} and in the remaining 5 animals, ranged between 178 and 379 pmol ℓ^{-1}. Vitamin B_{12} concentrations exceeded 1 400 pmol ℓ^{-1} in all animals.

Blood lead concentrations were below 15 $\mu g\ \ell^{-1}$ in all animals.

Fifteen (33,3%) of the horses were found to be carriers of either *B. caballi, B. equi* or both. The results of the examination of blood smears as well as serological examinations are presented in Table 3.

DISCUSSION

The presented results are generally in agreement with published laboratory parameters for healthy Thoroughbreds at rest and could be accepted as reference baseline values[1].

Our investigation into resting Ht, supports the finding that the determination of resting Ht only, without measuring the response to exercise, does not suffice to differentiate between poor and excellent performance[1]. As expected, excitability resulted in an increase in Ht and underscores the need to collect specimens for haematological examination in a calm manner. The higher Ht in mares is also in line with our present knowledge of factors influencing the resting Ht in horses[1 11].

Contrary to the findings by Muylle et al.[8 9], investigations into red cell potassium concentration in this relatively small group of horses, failed to demonstrate differences between horses of different temperament and performance.

No attempt was made to relate serum biochemistry finding to performance. Despite the possible use of parameters like serum phosphorus and certain enzymes, the marked fluctuations in normal ranges in these parameters, usually makes this a frustrating exercise[1].

Ionized serum calcium concentrations were approximately 54% of the value for total serum calcium. This value is slightly higher than the approximately 50% given for dogs, but is in agreement with the percentages reported for cattle[2].

The acceptable serum concentrations of folate and the very high serum concentrations of vitamin B_{12}, underscore the futility of the practice of administering these vitamins to horses to improve their performance. Subsequent analysis of serum specimens from 20 of the same group of horses during the winter months, yielded similar serum concentrations of folate and Vitamin B_{12} (Van Heerden & Kirkpatrick 1990, unpublished data).

Despite the fact that these horses were kept in a peri-urban area, blood lead concentrations were surprisingly low, especially when compared to blood lead levels of 34 ug ℓ^{-1} in humans in a remote, unpolluted area in the Republic of South Africa[6].

Relatively little has been published on parathyroid hormone concentrations in horses. The commercial kit used in this investigation has been validated for equine carboxy-terminal parathyroid hormone[12] and our results are in agreement with their reported range of 0,32 to 0,92 ng ml^{-1} (32-92 pmol ℓ^{-1}) in 10 healthy female horses. Parathyroid hormone con-

centrations should, however, always be interpreted in relation to serum calcium and phosphorus concentrations and could be of use in the diagnosis of primary and secondary hyperparathyroidism. The determination of serum concentrations of parathyroid hormone may well be of more diagnostic use than the determination of serum concentrations of calcium and phosphorus in identifying dietary imbalances and metabolic bone disease.

The relatively low incidence of a carrier-status of *B. equi* and *B caballi* probably reflects on the excellent ectoparasite control programme practised at the yard, as well as the need to maintain the programme. However, despite strict tick control measures, horses are still exposed to infected ticks and clinical cases are encountered from time to time (Nichas 1989, unpublished data). Horses which had positive blood smears at the time of our investigation were subsequently found to have seroconverted (Van Heerden & De Waal 1989, unpublished data).

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News/Nuus

SIXTH INTERNATIONAL CONFERENCE ON EQUINE INFECTIOUS DISEASES. CAMBRIDGE, ENGLAND. 7-11 JULY 1991

The provisional programme for this conference will consist of plenary review lectures given in the mornings of the conference by international experts on equine topics including immunology, parasitology, bacteriology, epidemiology of equine infectious diseases and virology. Afternoon sessions will consist of oral presentations selected on the basis of abstracts received, workshops and poster sessions.

There will be a full programme for accompanying persons and the social programme will include an afternoon at Newmarket Races and a visit to Tattersalls' sales paddocks.

Abstracts of submitted papers must be received by 31 October 1990 and registration for the conference, which is limited to 300, must be made by 30 April 1991.

Further details can be obtained from EIDC Organising Committee, Department of Clinical Veterinary Medicine, University of Cambridge, Madingley Road, Cambridge CB3 OES, United Kingdom.

OVERBERG RESEARCH PROJECTS VII. ANTHELMINTIC SALES IN THE SWELLENDAM AREA OF THE SOUTHERN CAPE

J P LOUW*

ABSTRACT

In the Swellendam area, benzimidazoles held 34,5% of the market for sheep and goat nematocides in 1988, ivermectin 30,1%, levamisole 13,7%, combination products 13,5%, salicylanilides 6,8% and morantel 1,4%, while products containing benzimidazole accounted for 48,1% of sheep and goat nematocide sales.

The sheep and goat nematocide and cestocide markets in the Swellendam area totalled 1 386 280 and 94 080 therapeutic doses respectively, costing R448 887,10 and R79 242,02, while the cattle anthelmintic market totalled 12 209 therapeutic doses, costing R30 520,22. A mean of 3,45 therapeutic doses of nematocide were purchased for sheep and goats during 1988 at a per capita cost of R1,12 at December 1988 prices. Furthermore, the mean per capita expenditure on anthelmintics against both the nematodes and cestodes of these animals, amounted to R1,31. Nematocides used for sheep and goats in this market segment, cost 36 cents per dose. The mean escalation of anthelmintic prices between January and December 1988 was 10%, ranging from -6 to 30%.

Key words: Anthelmintic market, south western Cape, sheep, goats, cattle, dosing costs.

Louw J.P. **Overberg Research Projects VII. Anthelmintic sales in the Swellendam area of the southern Cape.** *Journal of the South African Veterinary Association* (1990) 61 No. 4, 159-162 (En.) Department of Parasitology, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, 0110 Onderstepoort, Republic of South Africa.

INTRODUCTION

Anthelmintic sales statistics can be employed to gauge market trends and provide clues regarding the intensity and scheduling of anthelmintic application in relation to local helminth epidemiology. According to Reinecke[8] farmers use anthelmintics too frequently, a practice that can be expected to lead to anthelmintic resistance in nematodes, since the frequency of drenching with a given compound correlates with the occurrence of resistance[3 7 10]. An anthelmintic sales survey was conducted in the southern Cape region to determine the volume and value of the various anthelmintics purchased in the area and, furthermore, to compare the scheduling of purchases with suggested control programmes[5] based on local helminth epidemiology[9].

The present study was carried out in the Swellendam area of the southern Cape region.

MATERIALS AND METHODS

The regional segment of the anthelmintic market included in the present study, comprised the whole of the area affiliated with the Central-South Agriculture Co-operative. This institution sells to stock farmers owning approximately 389 000 sheep, 13 000 goats and 27 000 cattle[1]. As a result of extensive incentive schemes such as credit sales, deferred payment and discounts offered by the co-operative to its members, almost all anthelmintic sales, even those made by representatives of the manufacturers, are channelled through the institution. Thus, for the purpose of this paper, anthelmintic sales by the co-operative were regarded as the total of all anthelmintic sales in this area. The stock figures were extracted from the official 1988/89 stock census. The retail sales figures were arranged by trade name and compounded by generic group viz. ivermectins (Ivm), benzimidazoles (Bnz), levamisoles (Lvz), morantel (Mrl) salicylanilides (Sa), combinations of Bnz and Sa (Cmb) and the cestocides. Sales volumes of anthelmintics for sheep and goats, as well as for cattle were expressed in 50 kg doses. Local retail prices of anthelmintics were recorded in January 1988 and again in December 1988 and the cost of individual doses, as well as the price escalation during the 12-month period, calculated.

Mean monthly temperature and total monthly rainfall recorded at the Swellendam prison, were incorporated in the study.

RESULTS

The percentage division of the sheep and goat nematocide market by generic group of products is illustrated in Fig. 1, while the monthly sales of the 3 leading generic groups of anthelmintics are presented in Fig. 2.

The number of doses of anthelmintics per 50 kg body mass sold during 1988, totalled 1 590 240 of which 1 480 360 doses were for sheep and goats and 109 880 for cattle. Converted to 450 kg body mass doses, the anthelmintics sold for cattle during 1988 would be sufficient to treat approximately 12 209 adult cattle. Although some benzimidazole products are registered and probably used as cestocides, proportions could not be determined by this survey and all benzimidazoles were regarded as nematocides. The total monthly sales of nematocides and cestocides for sheep and goats, together with mean monthly temperature and total montly rainfall are presented in Fig. 3. Total anthelmintic sales in this segment of the market amounted to R558 649,34 in December 1988 and R509 169,11 in January 1988, and R538 129,12 and R481 988,33 respectively for sheep/goat anthelmintics. Price changes during the 12 months' duration of the survey ranged from -6 to

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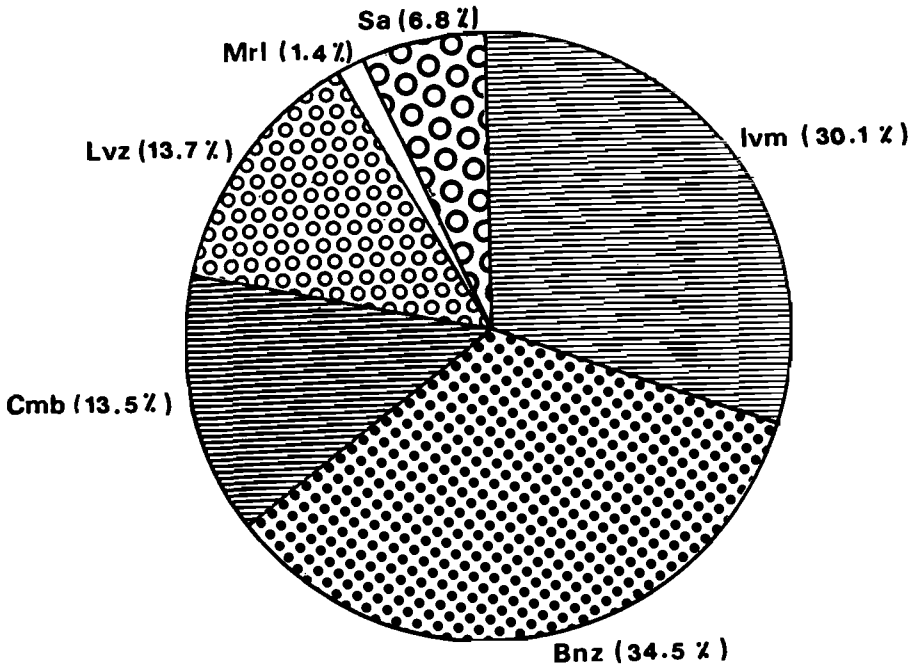

Fig. 1: **Percentage division by generic group of products of the nematocide market for sheep and goats in the Swellendam area in 1988. Bnz = benzimidazoles; Ivm = ivermectins; Lvz = levamizole; Mrl = morantel; Sa = salicylanilides; Cmb = combinations of benzimidazoles and salicylanilides**

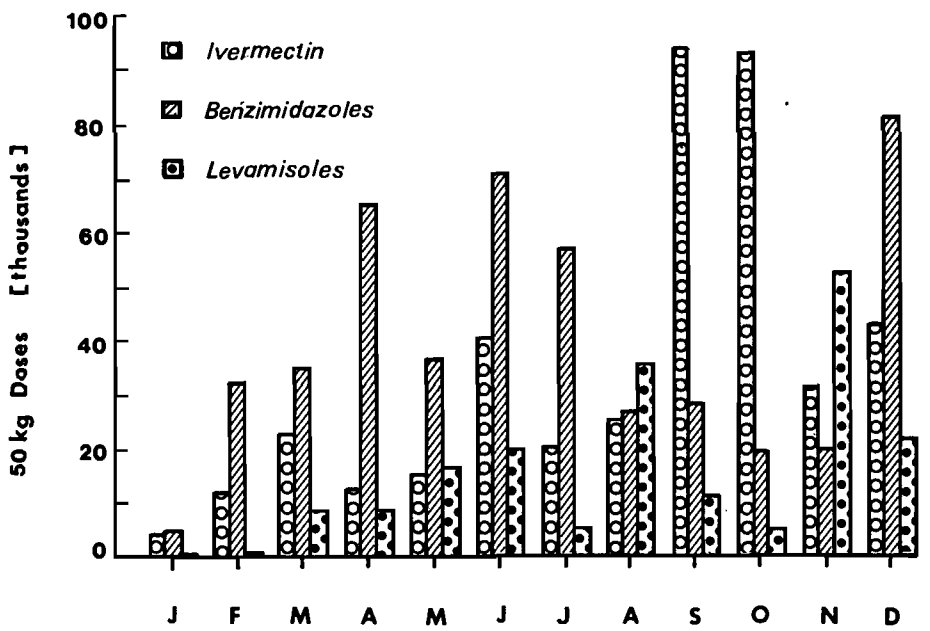

Fig. 2: **Monthly sales of the 3 leading generic groups of anthelmintics for sheep and goats in the Swellendam area during 1988**

30%, with a mean increase of 10%. The monthly sales of sheep/goat nematocides accumulated in multiples of 402 225 (the number of doses required for a single treatment of all sheep and goats in the area) from January to December 1988 are presented in Fig. 4. A total of 3,45 market segment therapeutic doses (multiples of 402 225 doses) of nematocides were sold for sheep and goats in the area.

DISCUSSION

Market share

The benzimidazoles were the market leaders with a 34,5% share in the sheep and goat market, followed by the ivermectins (30,1%) and the levamisoles (13,7%). Benzimidazole-derived products sold in this segment of the market for sheep and goats, accounted for 48,1% of sales, compared with 57,9% in the Bredasdorp area[6]. The sales success of the pure benzimidazole products may be attributed to their attractive prices, claimed efficacy against *Ostertagia,* the major nematode in this region, and the efficacy of some against the cestodes of lambs.

Ivermectin was probably the drug of choice for the simultaneous control of nematodes and *Oestrus ovis* and sold 122,4% more doses than the rival combination products of benzimidazoles and closantel, rafoxanide and trichlorfon.

Sales volume analyses by individual compound, revealed that ivermectin (416 500 doses) was highest in demand for sheep and goats, followed by albendazole (308 750 doses) and anthelmintics containing mebendazole (197 800 doses).

Market trends

Sheep nematocide sales peaked in June and August to December (Fig. 3), coinciding with the time of the year when sheep are moved to winter pastures, lambs are weaned and sheep are moved to summer pastures. The consistently high sales figures recorded during the summer were probably due to substantial summer rains, typical of this area which forms the transition to the non-seasonal rainfall area (Fig. 3). Cestocide sales coincided with the lambing season, reaching a peak in June when lambs were approximately 2 months of age and in need of treatment (Fig. 3). The cumulative monthly sales diagram (Fig. 4) indicates that market segment therapeutic doses of nematocides for sheep and goats were sold by June, September and December. Approximately 12 209 adult cattle, with a mass of 450 kg each, could be treated with the anthelmintics sold for cattle during 1988.

Injectable formulations of ivermectin for sheep are used primarily for the control of sheep scab, and were purchased mainly during late winter and spring.

Market price

In December 1988, a dose of sheep and goat anthelmintic used in the Swellendam region, cost the farmer a mean of 36 cents. Niclosamide and ivermectin sold for more than the mean price, and cost 84 and 49 cents respectively, while levamisole was the cheapest anthelmintic at 17 cents per dose. The cost of treating a 50 kg sheep or goat 3,45 times per year, as indicated by Fig. 4, could be as high as R1,69 if Ivomec was used or as low as 59 cents if Levamisole was used. The mean cost was R1,12. Cattle with a live mass of 450 kg, cost R2,52 to treat.

The mean price increase of all sheep and goat anthelmintics was 10%, but the price of ivermectin, morantel, the benzimidazoles and the salicylanilides increased by 15, 15, 12 and 12%, respectively. During 1986, the mean cost of all

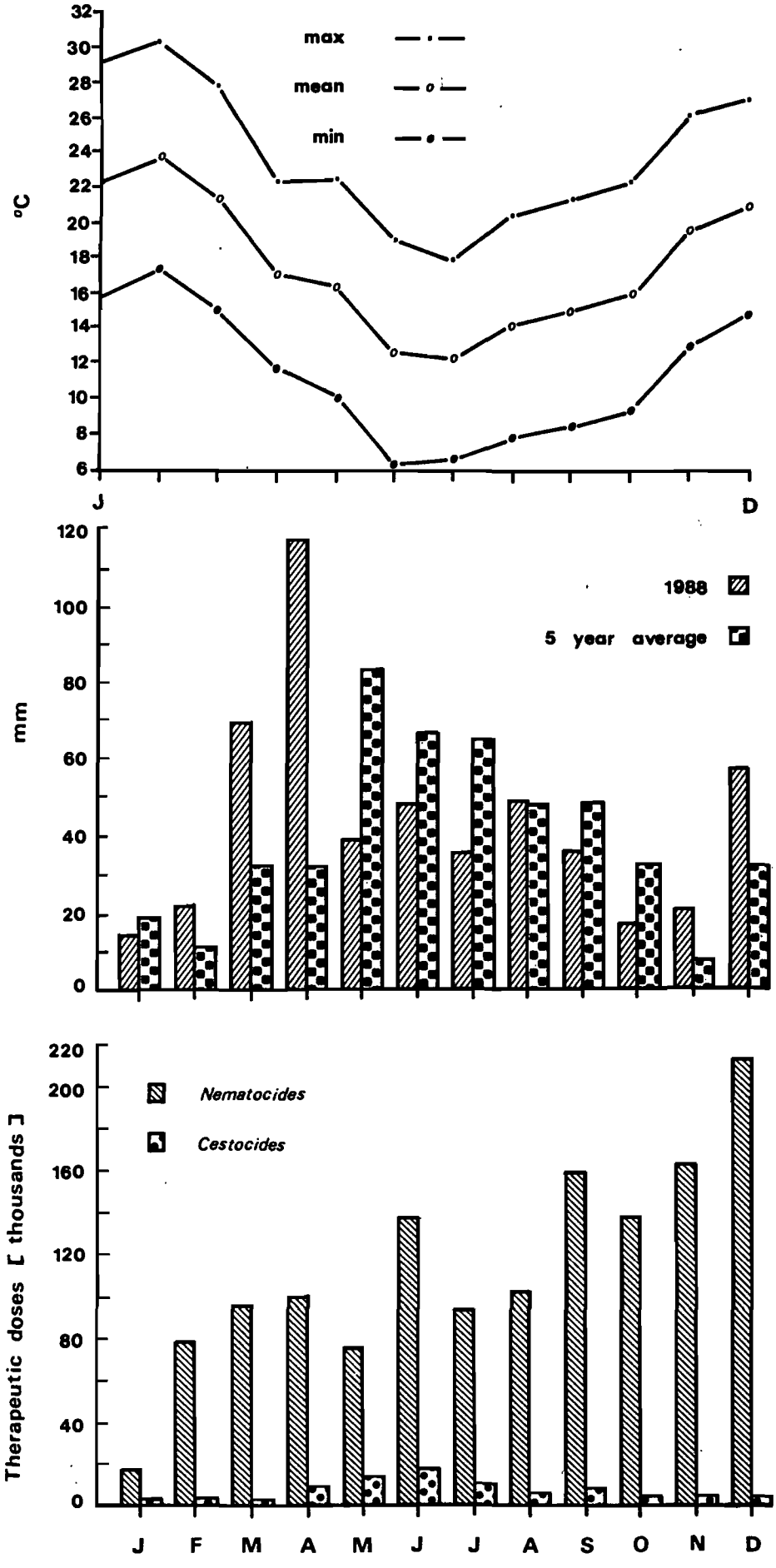

Fig. 3: **Monthly sales of nematocides and cestocides for sheep and goats, together with temperature (°C) and rainfall (mm) recorded in the Swellendam area during 1988**

anthelmintics used in the country was 16 cents[4]. The difference between the mean 1986 national cost of a therapeutic dose of sheep and goat anthelmintic, and the mean for 1988 for the Swellendam region, was 20 cents (125%).

The rate at which parasites develop resistance to an anthelmintic, is a function of the intensity of exposure to that compound[3,7]. The recorded mean of 3,45 doses sold per year for sheep and goats in the Swellendam area is, however, relatively low compared with drenching frequencies generally regarded as conducive to the development of anthelmintic resistance in nematodes[10]. It is more likely that the protracted and extensive use of the benzimidazoles as anthelmintics (48,1% of anthelmintics used, contained benzimidazoles) will precipitate such tolerance. However, the market share of benzimidazoles in Swellendam was (9,8%) lower than in the Bredasdorp region[6].

Even at prices which prevailed at the termination of the survey, the mean per capita cost of sheep and goat anthelmintics in the Swellendam region was R1,31, a negligible production cost factor amounting to little more than 1% of the current mean gross margin of R123,70 for sheep in this region[2].

Based on the epidemiology of the parasites of sheep in the Rûens, a region which includes the Swellendam area, anthelmintic treatments in May/June, July/August, September and November are recommended[5]. In the present study, market segment therapeutic doses were purchased by June, September and December of 1988.

ACKNOWLEDGEMENTS

I wish to thank the management and staff of the Central-South Co-operative (in particular Mr Gawie Steyn) for their support, also Mrs J. Mathewson for drawing the graphs and Prof R.K. Reinecke for his support and encouragement.

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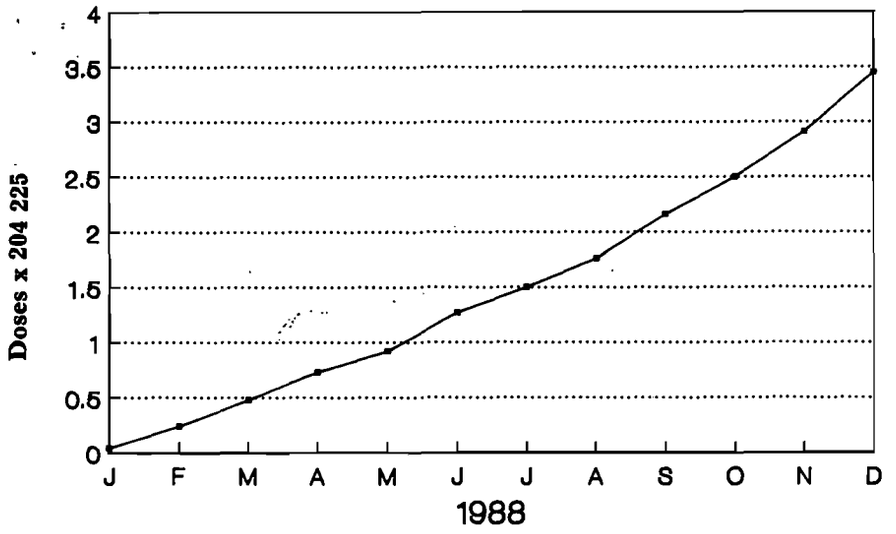

Fig. 4: **Cumulative monthly sales of nematocides for sheep and goats in multiples of 402 225 doses (the market segment therapeutic dose)**

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OVERBERG RESEARCH PROJECTS. VIII. THE PRODUCTIVITY OF MERINO EWES SUBJECTED TO DIFFERENT INTERNAL PARASITE CONTROL PROGRAMMES IN THE WINTER RAINFALL REGION OF SOUTH AFRICA

J P LOUW* and R K REINECKE**

ABSTRACT

Suckling Merino lambs on lucerne pasture, demonstrated no appreciable mass gain when compared with untreated controls, despite regular treatment with anthelmintics. This was ascribed to severe parasitic challenge. After weaning and transfer to wheat stubble fields with no parasitic challenge, however, the live mass of the untreated lambs, still harbouring a residual burden of nematodes, was depressed. Control sheep produced 1,2 kg less wool than regularly-treated sheep, but produced finer wool which had a higher market value. Regularly-treated ewes (F_1) produced 12,1% more lambs, but their mean live mass was 2,6 kg lower than that of ewes treated less frequently. The overall financial benefit was in favour of the group which received fewer anthelmintic treatments and was due mainly to the higher market value of the finer wool produced by these apparently stressed animals.

Key words: Sheep, body mass, wool production, reproductive performance, economic evaluation.

Louw J.P.; Reinecke R.K. **Overberg Research Projects VIII. The productivity of Merino ewes subjected to different internal parasite control programmes in the winter rainfall region of South Africa.** *Journal of the South Arican Veterinary Association* (1990) 61 No. 4, 163-167 (En.) Department of Parasitology, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, 0110 Onderstepoort, Republic of South Africa

INTRODUCTION

Internal parasites may be responsible for losses in sheep[3 6 7] and economic criteria can be used to evaluate these losses in producing animals[1 2 6 7 10]. Wool production, live mass gains, conception and lambing rates are measureable parameters which can be applied as instruments of economic assessment. Partial budgeting[2 9] considers only those costs and returns directly related to, and affected by the variables tested, and can be applied to simplify the process of evaluation.

A field experiment was conducted on the farm Boontjieskraal, in the southern Cape, for the purpose of determining the epidemiology of nematodes[12], a practical helminth control programme[8], as well as the economic effects of internal parasites on sheep in the region. The present report is confined to the effects of internal parasites on the live mass, wool production and conception and weaning rates measured in a trial which was essentially designed to study helminth epidemiology and control.

MATERIALS AND METHODS

The farm Boontjieskraal (34°12'S, 19°21'E), situated 10 km west of Caledon in the southern Cape, was selected for the trial. A flock of 500 pregnant Merino ewes on the farm was divided equally into 2 treatment groups. The ewe lambs subsequently born (Progeny I) in each group were used to compare the productivity of sheep regularly treated with anthelmintics (Group A) with that of sheep receiving limited anthelmintic treatment (Group B). The animals in the 2 treatment groups were identified with numbered ear tags of 2 different colours.

Animals of the 2 different treatment groups grazed in separate paddocks throughout the trial, from June 1987 (when Progeny I was born) until Progeny II was weaned in October 1989.

Anthelmintic treatments applied to the 2 groups of animals are summarised in Table 1. Animals in Group B were treated from 6 September 1988, when it became apparent from the monitor group (Fig. 3) that these ewes might not reach the target mass of 40 to 45 kg before the breeding season which was due to commence on 2 January 1989.

Management of the 2 groups of animals is summarised in Table 2. The 2 groups of animals were kept on separate pastures in order to confine the effects induced by treatment (or the withholding thereof) to the group. Pastures which were comparable in size were used and it was ensured that ample grazing was available at all times.

Initially, 6 male lambs from Progeny I and after weaning, 6 wethers from the same progeny of each group, were killed between 16 June 1987 and 5 December 1988 as well as on 27 April 1989 and on 5 June 1989. All the animals were processed and the internal parasites recovered[8 12].

Twenty ewe lambs were selected randomly from each flock and identified with an additional ear tag. This group of animals served as a monitor group, being checked every 6 weeks from 23 July 1987, when the Progeny I lambs were 4-6 weeks old, until 29 May 1989. In addition, the live mass of all the adult ewes was recorded immediately after shearing on 6 September 1988 (when they were 15 months old) and on 20 October 1989.

The wool production of each ewe was obtained on 6 September 1988 and again on 5 April 1989, by measuring the mass of the individual fleece, to which was added the mean of the pieces swept from the floor and pooled for each treatment group. The value of each fleece was estimated from wool samples analysed by the South African Wool Testing Centre for fibre diameter and clean yield and the auction price for wool of that type. The value of the wool produced by each ewe was estimated by multiplying the unit

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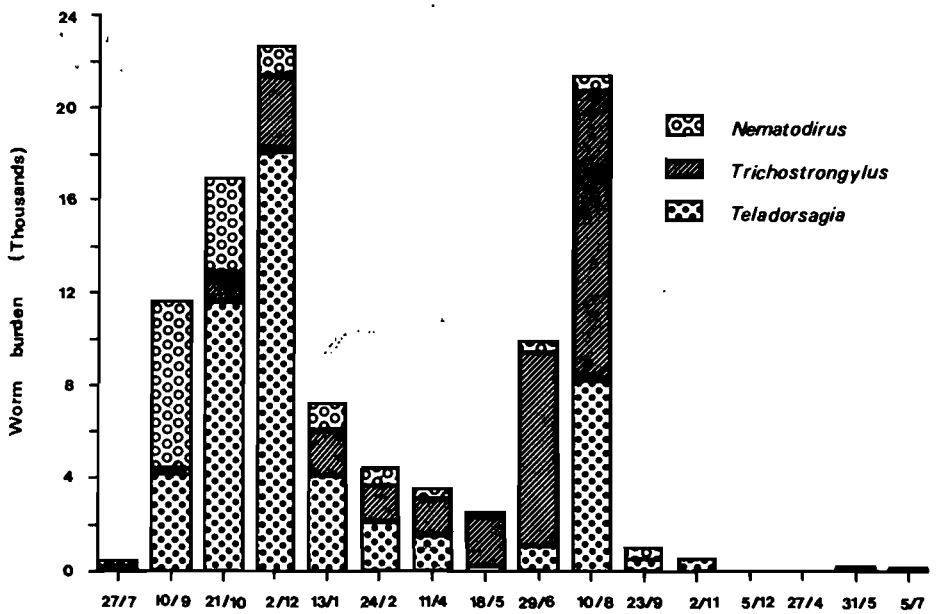

Fig. 1: **Geometric means of the *Teladorsagia, Trichostrongylus* and *Nematodirus* recovered from 6 male animals from Group B slaughtered every 6 weeks from the age of 8 weeks**

value of each fleece by the mass of wool produced by that ewe.

During April 1989, all ewes were scanned ultrasonically and the conception rate determined. On 20 October 1989 the lambs weaned from each group were counted and their weaning mass recorded.

In order to evaluate the economic value of the internal parasite control programmes followed for the 2 experimental groups of animals, a comparison of estimated returns and cost (excluding labour cost) was made.

RESULTS

Burdens of the 3 most common genera of nematodes *(Teladorsagia, Trichostrongylus* and *Nematodirus)* recovered from the animals necropsied from the 2 experimental groups, are presented in Fig. 1 and 2. With the exception of the necropsies done on 10 September 1987, very few nematodes were recovered from the animals of Group A (Fig. 2); but large numbers of all genera initially, but especially *Trichostrongylus* thereafter, were recovered from the animals of Group B until 10 August 1988 (Fig. 1). The lambs killed on 16 June 1987 were approximately 2 weeks of age, harboured no parasites and were not included in the trial results.

The mean live mass recorded every 6 weeks from the 2 monitor groups is illustrated in Fig. 3. On 23 July 1987, when the lambs were 4-6 weeks old, the mean live mass of Groups A and B was 16,4 kg and 16,7 kg respectively, while the corresponding live mass figures recorded on 29 May 1989, were 55,6 kg and 55,5 kg respectively. On 6 September 1988, however, when the decision was taken to apply anthelmintic treatments to all animals of Group B, the mass (43,7 kg) of these animals was significantly ($P < 0,05$) lower than that of the Group A animals (48,2 kg). On 20 October 1989, when the trial was terminated, the mean live mass of the animals in Group B was 59,1 kg and that of the animals in Group A 56,5 kg. This difference of 2,6 kg was not significant ($P > 0,05$).

Income from wool production, the reproductive performance of the ewes and the economic evaluation of the treatments, are summarised in Table 3, 4 and 5. The mean production of wool was significantly ($P < 0,05$) higher (1,20 kg) in Group A, but wool produced by animals of Group B realised significantly ($P < 0,05$) higher prices. The mean financial gain of R18,23 by Group B was not statistically significant.

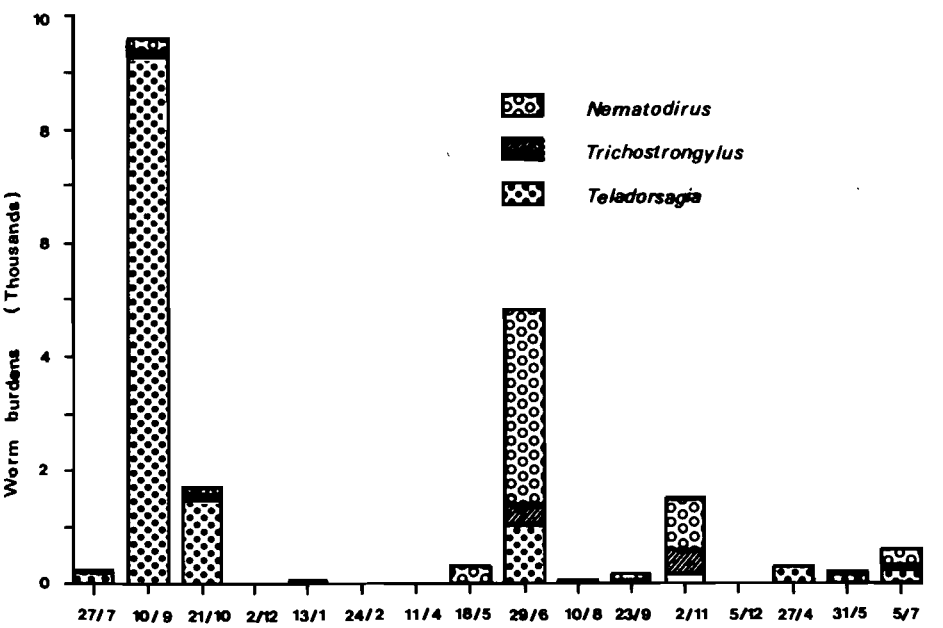

Fig. 2: **Geometric means of the *Teladorsagia, Trichostrongylus* and *Nematodirus* recovered from 6 male animals from Group A slaughtered every 6 weeks from the age of 8 weeks**

DISCUSSION

Although the Group A lambs necropsied until 21 October 1987, harboured low numbers of parasites (Fig. 2) when compared with the Group B lambs (Fig. 1), they did not outgrow the lambs in Group B (Fig. 3). While some workers[1,5] reported the dubious value of anthelmintic treatment while young sheep were exposed to severe helminth challenge, others[11] reported the remarkable ability of untreated lambs carrying large numbers of nematodes, to match the mass gains of lambs treated regularly with anthelmintics, while they were on nutritious pasture. When larval challenge subsides, however, as happens on drying-off pastures or under conditions of rising ambient temperature, the lambs start to drop out. The results of the present study confirm these observations. The mean live mass of the Group B animals after weaning, was consistently lower than that of the Group A animals, until regular anthelmintic treatments from 6 September 1988 reversed the trend (Fig. 3). It would appear, therefore, that the apparent drop in the nutritional status of the lambs when they were weaned and transferred to stubble lands, was an important factor in the manifestation of the negative effects of parasitism.

Anderson et al[2] observed that plasma

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Table 1: **The anthelmintic treatments applied to 2 groups of experimental sheep**

Date	Group A	Group B
21 July 1987	niclosamide (50 mg kg^{-1})	-
9 August 1987	albendazole (3,8 mg kg^{-1})	-
28 September 1987	ivermectin (0,2 mg kg^{-1})	-
26 November 1987	ivermectin (0,2 mg kg^{-1})	-
30 June 1988	ivermectin (0,2 mg kg^{-1})	-
6 September 1988	-	morantel (12,5 mg kg^{-1})
24 November 1988	ivermectin (0,2 mg kg^{-1})	ivermectin (0,2 mg kg^{-1})
25 April 1989	ivermectin + levamisole ($\pm$6 mg kg^{-1})	levamisole (6 mg kg^{-1}) (Ovivax) (Ovivax)
4 August 1989	morantel (12,5 mg kg^{-1})	morantel (12,5 mg kg^{-1})

Table 2: **Management of the experimental sheep**

Date	Procedure
6 May 1987	-
6 May 1988	- The experimental design, including the 500 Merino ewes (F) and their progeny have been described[8 12].
30 June 1988	- Group A treated with ivermectin, kept in pen for 5 h and both groups moved to lucerne pastures spelled for approximately 9 months.
6 September 1988	- Sheared all animals, recorded fleece mass and live mass. Group B treated and moved to a medics/lucerne pasture spelled for approximately 11 months.
24 November 1988	- Both groups of animals moved to separate stubble lands.
2 January 1989 - 28 February 1989	- Mated ewes.
10 February - 25 April 1989	- All animals received daily supplements of oats at a rate of approximately 500 gm/s per animal per day.
5 April 1989	- Sheared all sheep and recorded fleece mass.
25 April 1989	- Scanned all ewes ultrasonically for pregnancy. Both groups vaccinated with Ovivax and moved to separate, newly established lucerne pastures.
29 May 1989	- Treated all animals with selenium and vitamins A, D and E
8 June 1989	- Pregnant ewes moved to maternity pens and, after lambing, returned to lucerne pastures which had been established the previous year.
6 July 1989	- Moved Group A to another lucerne pasture.
10 September 1989	- Moved Group A to another lucerne pasture
25 September 1989	- Sheared all sheep
20 October 1989	- Recorded the live mass of all ewes and lambs and the number of lambs weaned. Terminated the trial

Table 3: **The total gross return from wool produced by each sheep**

Group	Mass, physical properties and value of wool produced						
	6 Sept 88	5 Apr 89	Total	Fibre diameter	Yield	Price	Income
	kg	kg	kg	u	%	R c	R c
Group A	5,03	4,88	9,91	20,9	69,9%	18,38	181,67
Group B	4,04	4,67	8,71	19,5	71,1%	23,54	202,21

pepsinogen levels of treated lambs did not differ from those of untreated controls under conditions of severe challenge, despite anthelmintic treatment every 2 weeks of the former. He ascribed this to lesions caused by *Teladorsagia* (=*Ostertagia*). It can be postulated, therefore, that unless the intake of *Teladorsagia* larvae and resultant mechanical damage are greatly reduced, lambs which are particularly sensitive to the proper functioning of their protein metabolism, will not benefit from anthelmintic treatments in any other way than by being protected from the risk of acute parasitism.

The depressed wool yield of the Group B animals (1,20 kg) is in accordance with the observations of Steel et al[13] who noted a marked depression of wool production in lambs concurrently infected for extended periods of time (Fig. 1) with *Teladorsagia* and *Trichostrongylus*. In the present trial, however, the parasitic stress exerted on the Group B animals resulted in the production of finer wool of higher market value, boosting the mean income from wool in Group B by R20,54, when compared with that of Group A (Table 3).

In Group A, 12,1% more lambs were weaned when compared with Group B, but the mean body mass of the former was 2,6 kg lower (Table 4). The difference in the body mass of the ewes, although not statistically significant, could either be due to a difference in the quality of the pastures grazed by these 2 groups of animals, or to the fact that the ewes of Group A had more lambs to rear.

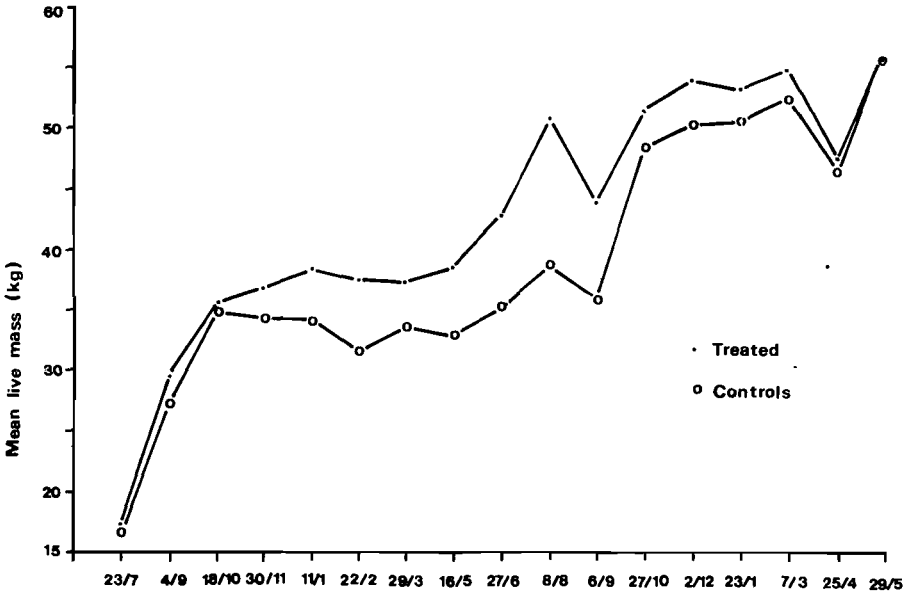

Fig. 3: **The mean live mass of the monitor animals from Group A and B recorded from 23 July 1987 to 29 May 1989**

Table 4: **Number of lambs weaned and the body mass (kg) of the ewes and the lambs at weaning**

Group	Ewes mated	Ewes conceived (%)	Lambs weaned	Weaning mass	Body mass Ewes at weaning
Group A	93	83 (89,3%)	78 (83,9%)	33,3	56,5
Group B	103	85 (82,5%)	74 (71,8%)	33,5	59,6

In the present study, an evaluation based merely on the physical performance of the animals, would probably rate Group A as the superior group (Table 3 & 4). However, if the market prices of the different commodities were considered (Table 5), the mean profit of Group B animals exceeded that of Group A by R18,23.

Numerous other workers following such a holistic approach, have found it difficult, or failed, to justify parasite control on economic grounds[5]. The outcome of such experiments depend largely on the prevailing market value of the parameters measured. In the present study, the steep gradient in the pricing of wool, favouring fine wool, was determined by market forces and caused a major upset in the economic analysis. Sensitivity analyses of the economics of treatment[1 10] will remain a retrospective exercise, of little value in decision making, due to the chronological order of accrued cost and realised value of the end product. Peripheral forces such as the constantly changing environment and market forces which influence decision-making, operations as well as profitability on a commercial farm, together with the variability of the parameters measured, often jeopardise the economic evaluation of parasite control programmes, but reflect the combined effect of all interacting forces in operation in a commercial farming enterprise.

ACKNOWLEDGEMENTS

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Table 5: **Summary of the financial returns from the 2 groups of animals**

	Group A	Group B
Wool income	R181,67	R202,21
Lambs produced: (Lambing %-age x mass x R3,00)	R 83,82	R 72,16
Value of ewes (body mass x R2,50)	R141,25	R149,00
	R406,74	R423,37
Less anthelmintics	R 2,60	R 1,00
	R404,14	R422,37
Difference		R18,23

Reinecke and Mrs Johanna Mathewson for their valuable contributions.

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SEROLOGICAL STUDIES OF BOVINE RESPIRATORY SYNCYTIAL VIRUS IN FEEDLOT CATTLE IN SOUTH AFRICA

M VAN VUUREN*

ABSTRACT

Serum antibody titres to bovine respiratory syncytial virus were determined in cattle (n=282). Samples were obtained at various feedlots and were collected in the 1989 and 1990 winter seasons, during the course of investigations into outbreaks of respiratory tract infections in feedlot cattle. Results of the survey revealed an antibody prevalence of 43% to bovine respiratory syncytial virus. Titres ranged from 1:10 to 1:1 280 as determined by the indirect fluorescent antibody test. The results also indicate that bovine respiratory syncytial virus is probably widespread in feedlot cattle in South Africa, and that this virus may be a contributing factor in the bovine respiratory complex.

Key words: Bovine respiratory syncytial virus, feedlot, cattle

Van Vuuren M. **Serological studies of bovine respiratory syncytial virus in feedlot cattle in South Africa.** *Journal of the South African Veterinary Association* (1990) 61 No. 4, 168-169 (En.) Department of Infectious Diseases and Public Health, Faculty of Veterinary Science, Medical University of Southern Africa, P.O. Box 236, 0204 Medunsa, Republic of South Africa.

INTRODUCTION

Infectious diseases of the respiratory tract remain the most important problem during the winter months in feedlots in South Africa. An aetiological diagnosis, based on clinical signs and post mortem lesions only, remain a difficult task. In addition, very little is known about the prevalence and role of certain viruses in feedlots in South Africa.

The presence of bovine respiratory syncytial virus (BRSV) has been confirmed in Europe, North America and Asia[10]. The importance of the virus as a causative agent in many cases of respiratory infection, has been confirmed by various authors[3 4 9]. However, the role of BRSV on the continent of Africa is largely unknown.

The isolation of BRSV is notoriously difficult, as a result of the lability of the virus and the very slow cytopathic effects produced in the initial passages[7]. To achieve any measure of success with viral isolation, it is imperative to utilise an effective transport medium and to inoculate cell cultures as soon as possible, following collection of the specimens[1]. In addition, the sample of choice is bronchoalveolar lavage fluid, the collection of which may be unpractical for some veterinarians[2]. Considering these constraints, it was decided to first confirm the presence of this virus in South Africa before embarking on viral isolation and other diagnostic techniques. The objective of this study was to confirm the presence of BRSV in South Africa and to determine the seroprevalence of BRSV in feedlot cattle.

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MATERIALS AND METHODS

Cell culture

Bovine embryonic lung cells were harvested from a bovine foetus obtained from a local abattoir. The cells were grown and maintained in Hank's-Eagle's medium with 10% foetal calf serum (Highveld Biologicals, Johannesburg) with antibiotics. In the indirect fluorescent antibody (IFA) test, the medium was used with 2% foetal calf serum, and cells of the sixth passage were used.

Virus

The Iowa (FS1-1) strain (American Type Culture Collection) was used as the source of antigen for the IFA test. The origin of this virus has been described[8].

Serum samples

Serum samples for the survey were obtained during weekly visits to various feedlots (n=6) located in Transvaal, north-west Cape and eastern Free State during the 1989 and 1990 winter seasons. Sera were collected either from animals that were noticed that morning in their camps with suspected respiratory tract involvement, or from animals in the feedlot hospital camps, which might have had antibiotic treatment for one, 2 or 3 d. Serum samples were heat-inactivated at 56°C for 30 min and stored at -20°C until serotesting was done. For screening purposes, the sera were diluted 1:10, and for antibody titre determinations serial twofold dilutions were made from 1:10 to 1:1 280.

Indirect fluorescent antibody test

The method of Rossi et al.[6] for the preparation and staining of BRSV-infected cells was used with minor modifications. Briefly, the technique involved the inoculation of monolayers of bovine embryonic lung cells with aliquots of BRSV suspended in serum-free tissue culture medium, containing diethylaminoethyl dextran at a concentration not exceeding 40 $\mu g\ ml^{-1}$ of medium to enhance attachment of virus to cells[5]. After adsorption for 2 h, the monolayers were washed with phosphate buffered saline and maintenance medium, containing 2% foetal calf serum, was added. Cells were harvested by trypsinisation 24 h later, counted and spotted on 8-well slides. The slides were fixed in cold acetone and stored at -70°C.

For the indirect test, slides were incubated with the test sera for 30 min at 37°C in a moist chamber, before undergoing 3 separate 5 min-washes using a magnetic stirrer. Fluorescein isothiocyanate conjugated rabbit anti-bovine immunoglobulin G (Zymed Laboratories, San Francisco) diluted 1:80 with 0,05% Evans Blue stain, were added to the dried slides and incubated for another 30 min, followed by serial washing of the slides. Every slide contained one spot with positive and negative reference sera (Wellcome Research Laboratories, Beckenham, England) respectively.

RESULTS

Antibodies to BRSV were found in

121(43%) of the serum specimens collected. Antibody titres ranged from 1:10 to 1:1 280. Titres were only determined in serum dilutions up to 1:1280. Every single group of animals bled on any specific occasion, yielded some positive specimens. Specimens determined to be positive during screening tests, were retested to determine antibody titres, and the majority yielded titres of 1:320 or more, with a mean of 1:640.

DISCUSSION

Baker reported that it is unlikely that animals older than 6 months would have colostral antibodies[1]. In addition, positive titres as a result of vaccinations can be ruled out, in view of the fact that vaccines against BRSV are unavailable in this country. It has also been found that no cross-reactions occur between BRSV and other viruses implicated in differential diagnoses such as bovine viral diarrhoea virus, parainfluenza type 3, bovine herpesvirus type 1 and adenovirus[10].

It would seem likely, from the high antibody titres, that most animals seroconverted after arrival in the feedlot. According to Wellemans[10], antibody titres develop rapidly after infection and reach peak levels within 7 d. The antibody level remains elevated for a month and then steadily declines. He found average IFA titres of 1:810 during the first few days, and average titres of 1:135 after 2 months. Although high antibody titres with IFA tests demonstrate recent infection with BRSV, it cannot be concluded from the present study that BRSV played a primary role in those animals which presented with clinical signs of respiratory tract infection.

In order to be able to show conclusively an association between infection with BRSV and clinical pneumonia, it would be necessary to embark on viral antigen detection procedures and to collect paired or follow-up serum samples. However, there are practical constraints in the usual commercial feedlot operation that make the collection of paired serum samples very difficult.

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SEPTICAEMIC *ERYSIPELOTHRIX RHUSIOPATHIAE* INFECTION IN THE LITTLE SWIFT *(APUS AFFINIS)*

M VAN VUUREN* and J M M BROWN**

ABSTRACT

Erysipelothrix rhusiopathiae was found to be the causal organism of high mortality in a colony of Little Swifts (*Apus affinis* (Gray), occupying the vertical walls of high-rise buildings. The mortality continued for a period of about 4 weeks. Negative post-mortem findings necessitated a diagnosis based on bacterial examination during which the causal organism was isolated in pure culture from the liver, spleen and heart blood of affected birds.

Key words: *Erysipelothrix rhusiopathiae,* Little swift, *Apus affinis,* Apodidae, septicaemia

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Erysipelothrix rhusiopathiae is the causative agent of erysipelas in pigs, sheep and turkeys, and of erysipeloid in humans. In avian species it causes an acute or subacute septicaemic disease characterised by high mortality[2].

The organism is a Gram-positive, thin, straight or partially bent rod. Although it can be isolated from certain soil samples, it cannot multiply or survive indefinitely in soil, and is maintained by carrier animals[4]. It is reported that 30-50% of pigs harbour the bacterium in their tonsils and other lymphoid tissues. Turkeys are known to carry the organisms in their caecal tonsils, liver, and other organs[5].

It is assumed that carrier animals, especially turkeys and pigs are the sources of infection during outbreaks[4]. *E. rhusiopathiae* has also been isolated from a variety of ectoparasites which may act as a source of infection[1].

A large colony of Little Swifts, *Apus affinis* (Gray), family Apodidae use the vertical walls of the high-rise buildings of the Medical University of Southern Africa as their nesting and roosting site. During the late autumn of 1987, a considerable number of these birds died of an unknown cause. The weather during this time was fair to mild. Many ailing and moribund birds as well as large numbers of dead birds were found on the ground beneath their nesting and roosting sites. The incidence of mortality continued for about 4 weeks, during which time a number of these birds were presented for examination.

Post-mortem examinations were performed on 10 birds. These birds were also examined for the presence of ectoparasites. Specimens of the liver, spleen and heart blood were collected for bacteriological examination. They were cultured anaerobically at 37°C on Columbia blood agar plates containing 7% defibrinated horse blood and in an atmosphere of air containing 10% carbon dioxide.

Ectoparasites from all the birds were homogenised and placed in enrichment broth before being cultured on agar plates.

Post-mortem examinations were unremarkable and the most significant findings were slight enlargement and congestion of some internal organs, most notably the spleen.

Erysipelothrix rhusiopathiae was isolated from the liver, spleen and heart blood of 6 of the 10 birds. Colonies appeared on the second day and were minute, dew drop-like, pinpoint in size, and smooth. Partial haemolysis, which was easier to see after removal of some of the colonies from the growth medium, was present in the culture medium.

The primary isolate was identified on the basis that it was negative for the production of catalase, indole, nitrates and hydrolysis of aesculin, and positive for the production of H_2S in triple sugar iron agar. Culturing produced a "test tube brush" growth after 3 d in gelatine at room temperature. The cultures did not alter litmus milk and the bacteria were non-motile[1].

All of the birds were infected with the louse-fly *Crataerina acutipennis.* Attempted isolation of *E. rhusiopathiae* from these ectoparasites was unsuccessful.

The isolation of *E. rhusiopathiae* in pure culture from 6 out of 10 birds, suggests that the cause of the mortality was due to septicaemic *E. rhusiopathiae* infection. Mortality following *E. rhusiopathiae* septicaemia, has been described in 15 wild bird species[2]. As was the case with most of the other recorded outbreaks in avian species, the source of the infection could not be established in this outbreak[2]. The Veterinary Faculty of the Medical University of Southern Africa includes a Large Animal Production Unit where poultry, pigs, horses, cattle, sheep and goats are kept. *E. rhusiopathiae* can be passed in the faeces by carrier animals[4]. The sewage from all sections of this establishment is processed and then pumped into 2 large storage dams for use in irrigation of the gardens and cultivated pastures. Due to the large quantity of sewage processed, problems sometimes occur and the storage dams develop an odour indicative of heavy microfloral contamination. These dams are used by the swifts for drinking purposes. Swifts drink in flight during glides over water, and the birds in question could have been infected by contaminated water.

A number of birds and fish are present at and in these dams and they might have presented a further source of infection. The most likely cause of infection with *E. rhusiopathiae* in wild birds (as in mammals) is ingestion of contaminated food or water. These hosts could have maintained the bacterial infection having been first infected by contaminated sewage. No at-

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tempt however was made to isolate *E. rhusiopathiae* from the dam water.

Swifts are highly specialised insectivorous birds and only feed in flight. The only major disadvantage of an aerial life, is their total dependence on flying insects or insects carried in air currents for their source of food. This causes prolonged periods of cold or inclement weather to pose a very real threat to their survival. It has been observed that a few days of continuous rainy or cold weather has led to many Little Swifts being unable to fly, most likely as a result of hypoglycaemia following on the unavailability of food (R. Earle, personal communication). In this particular case, the weather was mild, making it unlikely that death could have been caused by the elements.

The nests are usually domed structures made from aerial detritus collected in flight, such as pieces of straw, grass, plantfloss, feathers, down, tiny twigs or human refuse like pieces of paper, plastic material, knitting wool or other fabrics, all of which are cemented together and on to the substrate with copious amounts of highly muco-proteinaceous, quick-setting saliva. It is possible that some of this material originated from the Animal Production Unit and could have been contaminated with *E. rhusiopathiae.*

The nests harbour not only parasites, but also many scavenging organisms and commensals. These affect not only the comfort of the nestlings and their parents, but also their vitality[3]. There was no evidence of overwhelming parasitic infestation on any of these birds, so that this is unlikely to have been a factor contributing to the death of these birds. Of all the insect pests which swifts have to endure, the most common are probably the louse-flies belonging to the family Hippoboscidae. The adults, which may be either oviparous or viviparous, generally live permanently on the host, feeding on its blood. In Africa the main culprits belong to the genera *Crataerina, Pseudolynchia,* and *Ornithomya.* Little Swifts particularly, are parasitised by *Crataerina acutipennis* in this country. This organism was found on most birds examined during the period under discussion. However, attempts to isolate *E. rhusiopathiae* from these parasites, were unsuccessful.

When investigating the cause of mortality within any bird colony, many contributors to mortality should be considered. In this case, *E. rhusiopathiae* was considered to be the most likely cause of death. Future studies directed at healthy swifts, may provide significant information regarding a possible carrier status.

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To the editor/Aan die redakteur

COMPLICATIONS OF OVARIAN AUTOTRANSPLANTATION IN BITCHES : AN UPDATE

Following my original report[1], 14 further patients have developed post ovarian auto-transplantation (ATOPA) complications. Five bitches showed recurrent prooestrus, 6 developed bleeding gastric ulcers due to hyperplasia of transplanted ovarian tissue, and 3 developed neoplasia within the transplant or at the site of the transplant. One bitch died from severe anaemia due to the bleeding ulcer.

In addition to the clinical signs described in the original article, one case presented with a large anterior abdominal mass due to "ovarian lutein neoplastic transformation" of the transplanted ovarian tissue in the spleen. Histopathological examination of the other 2 neoplasms revealed a granulosa cell tumour of the stomach wall, and "sarcomatous transformation of the ovarian autograft" in the spleen. The bitch which developed the granulosa cell tumour had a hyperplastic ovarian transplant removed 2 years previously.

A total of 55 out of 1 130 cases have now shown complications of the ATOPA operation. As originally noted, some of these cases can take a long time (up to 128 months in one case) to manifest clinically.

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SEROLOGICAL SURVEY FOR BOVINE LEPTOSPIROSIS IN THE VOLKSRUST DISTRICT

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ABSTRACT

Serum samples (n=860) from cattle in the Volksrust district were tested for *Leptospira* antibody titres. Seventeen (2%) of the animals were positive for leptospirosis, while 9(1%) animals showed suspect reactions. Titres against *L. hardjo, L. pomona,* and *L. tarassovi* were the most prevalent.

Key words: Bovine leptospirosis, serology

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Leptospira organisms could be the cause of disease in cattle, other domestic animals, game and man[5 11]. In cattle the disease causes economic losses due to abortions, stillbirths, infertility, decreased milk production and deaths[3 11].

Leptospirosis is a disease of world-wide importance and the organisms causing it have been found in most countries in the world[2 3]. Epidemiological data on leptospirosis in Africa is lacking[2], however the serological surveys that have been done in Africa and southern Africa, suggest that *Leptospira* organisms occur in Africa and that they contribute to economic loses[1 2 4 6 9 10]. *Leptospira interrogans* serovars, *pomona* and *hardjo* have been associated with abortions in southern Africa[6 9 10]. Abortions and infertility occur commonly in cattle in southern Africa and leptospirosis is usually considered to be an important differential diagnosis in the case of abortions (Q T Otto 1990, unpublished data).

This study was conducted to determine the prevalence of *Leptospira* antibodies in cattle in the Volksrust district. The prevalence of antibodies could serve as an indicator of the importance of leptospirosis in this area. Farmers in the Volksrust district do not use leptospirosis vaccines as a rule (Q T Otto 1990 personal communication).

Serum was collected from cattle during a campaign for the detection of antibodies against *Brucella abortus*. Cattle older than 18 months in the Volksrust district, were bled over a 3-month period and tested for brucellosis at the regional veterinary laboratory. Ten per cent of these samples were selected and tested for leptospirosis antibody titres. The sera were tested using the microscopic agglutination micro-volume technique[7 8]. The following antigens were used: *canicola, copenhageni* (icterohaemorrhagiae), *grippotyphosa, hardjo, mini* (szwajizak), *pomona, pyrogenes* and *tarassovi* (hyos). Antigens were

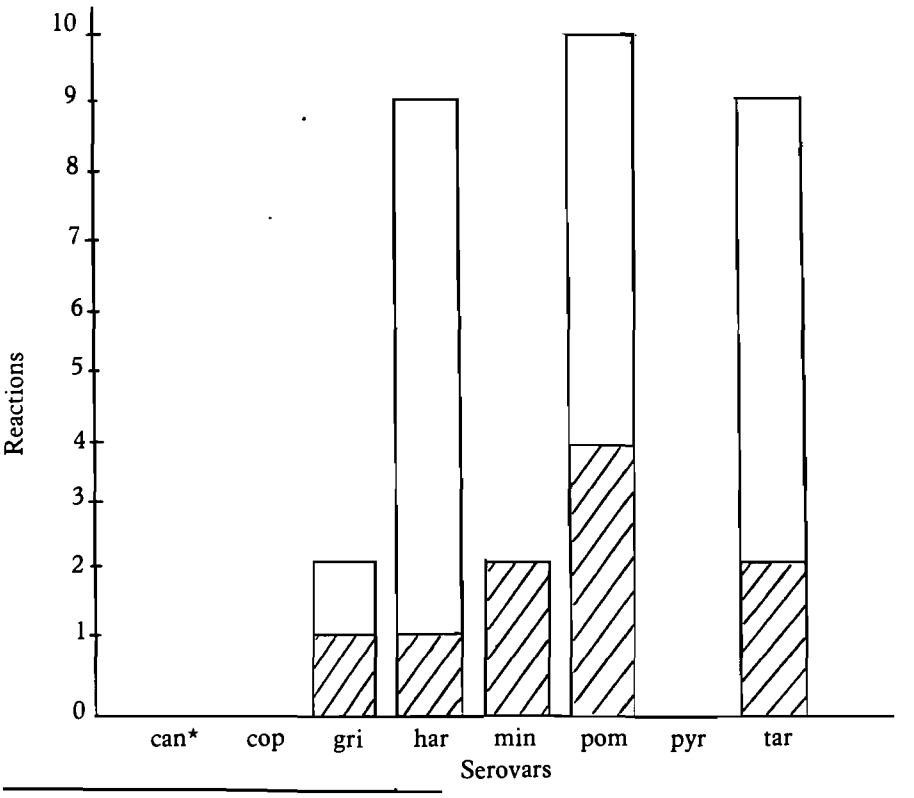

Fig. 1: **Number of reactions against serovars for all sera tested**

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grown on liquid EMJH (Difco Laboratories, Detroit michigan, USA) medium and used between 4 and 14 d when the growth of the leptospires exceeded $2x10^7$ organisms per ml[7 8]. The end-point titre was taken as the dilution where 50% of the organisms, as compared with the negative control, were either absent or visibly agglutinated and where there was a greater degree of agglutination in the immediately preceding lower dilution. A titre of 40 was regarded as negative, 80 as suspect and 160 or higher as positive[7].

Seventeen (2%) of the animals tested, were positive for leptospirosis, while 9(1%) animals showed suspect reactions. These animals originated from 44 herds and 13 (29,5%) herds had one or more animals which tested positive.

The total number of reactions (positive and suspect) against these serovars are reflected in Fig. 1.

The prevalence of animals with positive antibody titres in this survey is not very high (2%). The low prevalence of positive antibody titres, indicates that leptospirosis is not very widespread or important in the Volksrust district. We suspect that the relatively low soil pH[11] of the eastern Transvaal Highveld, as well as the extensive farming conditions may play a role.

The 13 (with one or more positive animals), out of 44 herds tested (29,5%), compares favourably with the results of Botes et al.[1], who found 31,5% of herds in the Transvaal to have positive animals.

The low titres recorded, point to carrier states or low grade exposure to the *Leptospira* serovars involved, which are all potential pathogens. *L. hardjo* and *L. pomona* could cause abortions[6 10], but information on the pathogenicity of *L. tarassovi* is lacking. Economic losses could be experienced on individual farms, when conditions are suitable for outbreaks to occur. Excretion of organisms by clinically sick animals or carriers, as well as the contamination of the environment, could cause abortions of epidemic proportions, if a large percentage of the susceptible animals in the herd were pregnant at the time.

In view of the fact that epidemiological data on leptospirosis is lacking, further serological surveys should be done in adjoining and other districts.

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SURVEY FOR ANTIBODIES TO SELECTED VIRUSES IN LABORATORY MICE IN SOUTH AFRICA

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ABSTRACT

Serum samples (n = 184) from 3 conventional laboratory mouse colonies were tested for antibodies with an indirect fluorescent antibody (IFA) test to Theiler's murine encephalomyelitis virus (TMEV), encephalomyocarditis virus (EMCV), and reovirus Type 3. The percentage of mice with antibodies to reovirus Type 3, were comparable in all 3 groups, namely 40, 46 and 60%. Antibodies to TMEV showed the greatest variation, namely 0, 10 and 31%, whereas antibodies to EMCV were confirmed in only one colony (7%). In addition, 98 serum samples from 2 other colonies yielded 69% animals positive for rotavirus with the IFA test.

Key words: Murine viruses, Theiler's murine encephalomyelitis virus, encephalomyocarditis virus, reovirus, rotavirus, mice

Van Vuuren M.; De Klerk W.A.; Van Niekerk T.A.; De Beer M.C. **Survey for antibodies to selected viruses in laboratory mice in South Africa.** *Journal of the South African Veterinary Association* (1990) 61 No. 4, 174-175 (En.) Department of Infectious Diseases and Public Health, Faculty of Veterinary Science, Medical University of Southern Africa, 0204 Medunsa, Republic of South Africa.

Laboratory rodents constitute the majority of the animals used for research in South Africa, as is the case elsewhere[2]. With the increasing establishment of barrier colonies of susceptible animals, the regular monitoring of these animals for infectious agents on a standardised basis becomes imperative. In addition, subclinical infections of laboratory mice may result in unreliable data with a concommitant negative impact on biomedical research.

There are more than 12 murine viruses that could infect mouse colonies without necessarily being diagnosed clinically, leading to interference in results of animal investigations[1].

Furthermore, data on the prevalence of diseases in laboratory animals has become increasingly important in South Africa, as animal experimentation is becoming more sophisticated, with a greater need for defined animals.

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The purpose of this report is to describe the prevalence of selected viruses in some contemporary laboratory mouse populations in South Africa.

Reovirus Type 3 and Theiler's murine encephalomyelitis virus (TMEV), together with reference antisera were obtained from J. Austin (MRC laboratory Animal Unit, P.O. Box 70, Tygerberg 7505). Encephalomyocarditis virus (EMC) and reference antiserum were obtained from R. Swanepoel (National Institute for Virology, Private Bag, Sandringham, Johannesburg). The bovine rotavirus strain used, was a field strain isolated by V.M. da Costa Mendes of the Department of Infectious Diseases and Public Health, Faculty of Veterinary Science, Medical University of Southern Africa.

Mouse antiserum to rotavirus was prepared by oral inoculation of suckling mice at 10 d of age, by means of a stomach tube with murine rotavirus-containing intestinal homogenates, obtained from T. Gerdes (Onderstepoort Veterinary Research Institute, 0110 Onderstepoort). Four weeks after inoculation, the mice were bled, and the sera pooled. For the remaining 3 viruses, hyperimmune ascitic fluid was produced in young adult mice, 4-5 weeks of age. Antibodies were titrated with an indirect fluorescent antibody assay and a working dilution determined for each antiserum.

Reovirus and EMCV were grown in the L-929 cell line, which is a derivative of Earle's strain mouse fibroblast (NCTC clone 929 of strain L). TMEV was grown in a baby hamster kidney, (BHK-21) cell line. Calf rotavirus was grown in MA-104 cells, a continuous line derived from rhesus monkey kidneys. Cells were grown in Eagles' minimum essential medium containing 10% foetal calf serum. The viruses were inoculated onto monolayers which were maintained with serum-free media. Although reovirus, TMEV and EMCV produced pronounced cytopathic effects (CPE) within 4 to 5d, cells were harvested after 24 h, before CPE became visible.

For cultivation of rotavirus, MA-104 cells were treated with crystalline trypsin at a final concentration of 10 μg ml^{-1} for 10 min, and washed twice with tissue culture medium. The virus was inoculated and left on the monolayer for 30 min, after which the cells were again washed twice. Serum-free maintenance media mixed in equal volumes with crystalline trypsin were added. CPE developed after 2 to 3 d.

Mice (n = 282) from 5 different conventional colonies were bled by cardiocentesis, under inhalation anaesthesia. The blood was allowed to clot at 4°C, followed by centrifugation and removal of the serum with a Pasteur pipet. Sera were heat-treated for 30 min at 56°C and stored at -20°C until used.

The results are recorded in Table 1 as the percentage positives in each colony. Ninety eight serum samples from 2 other colonies, yielded 69% animals positive for rotavirus.

These results represent only one component in an ongoing programme to try and place serologic screening on a sound footing at our laboratory animal centre, as there are many components to consider before a screening programme can be regarded as successful[5].

Reovirus is a ubiquitous virus in mouse colonies, and can infect mice of all ages. In infected colonies of mice, high morbidity and high mortality may be noticed in suckling mice characterised by emacia-

Table 1: **Percentage of positive serum samples in mice from 3 different colonies**

Colony	n	EMC	TME	REO
1	63	0%	0%	46%
2	61	0%	10%	40%
3	60	7%	31%	60%

tion, steatorrhoea with an oily hair effect, jaundice, incoordination with tremors, and paralysis prior to death.

TMEV is ubiquitous in mouse colonies, but can also infect rats. Apart from the obvious clinical disease caused by TMEV, leading to paralysis and death, less virulent TMEV may persist in the central nervous system for months, producing inflammatory changes but no clinical signs of disease. The interpretation of central nervous system histopathology can be compromised by such an infection[3]. In addition, mice persistently infected with a virus and used in diagnostic clinical microbiology, may yield completely unreliable results.

Certain strains of EMCV are used as models for the investigation of diabetes mellitus, since they can destroy pancreatic ß cells in mice. These strains may also produce myocarditis. With other strains, mice of all ages may develop an acute fatal encephalitis[6]. EMC viruses have not been known to cause disease spontaneously in laboratory mice. Rodents of any age may serve as reservoir hosts. Therefore, infection in experimental mouse colonies would most likely be an accidental infection or the result of contact with wild rodents.

The high prevalence rate of rotavirus among mice of 2 breeding populations did not surprise us, as diarrhoea was consistently present in mice within the first 10 d of life. Adult mice do not develop clinically apparent disease, but viral antigens can be detected in intestinal tissues and seroconversion occurs[4]. In addition, rotavirus antibodies were detected in sera from mice in colonies which have never exhibited clinical signs of infection or which have not done so for several years[7]. Nonmurine strains of rotavirus are frequently experimentally inoculated into suckling mice[4]. It is important that mice to be used in rotaviral research be screened for evidence of murine rotavirus infection.

Our initial results show that when utilising conventional mice for biomedical research in this country, there is a distinct possibility of working with animals with viral infections which may significantly influence results. The ideal would be to aim for the availability of SPF animals, and a continuous monitoring programme to maintain SPF conditions.

ACKNOWLEDGEMENTS

The authors would like to thank Dr. R. Swanepoel and Mrs. P. Leman for their valuable technical assistance with aspects of the immunofluorescent antibody assay.

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EFFECT OF A GROWTH PROMOTER ON PREWEANING GROWTH OF BEEF STEER CALVES

J G E VAN ZYL*

ABSTRACT

The influence of a growth promoter on preweaning growth of beef steer calves in the Kalahari thornveld and shrub bushveld was investigated. Calves (n=40) implanted with a testosterone propionate-oestradiolbenzoate compound at an average age of 120 d, attained a higher (P<0,005) weaning mass (256,7 vs 239,1 kg) and superior gains (93,9 vs 79,9 kg) over the trial period than the control group (n=40).

Key words: Growth promoter, preweaning growth, steer calves.

Van Zyl J.G.E. **Effect of a growth promoter on preweaning growth of beef steer calves.** *Journal of the South African Veterinary Association* (1990) 61 No. 4, 176-177 (En.) Department of Livestock Science, University of Pretoria, 0083 Hillcrest, Republic of South Africa.

A large proportion of commercial beef cattle farmers in South Africa produce weaners. While recognising that reproductive performance remains the single most important factor influencing the profitability of the cow/calf enterprise, cost-effective techniques to increase weaning mass should receive attention. The use of growth promoters in feedlots is well-documented[4], and some information on the influence of anabolic compounds on postweaning growth of steers, grazing natural pastures, is available[2]. The influence of growth promoters on preweaning growth of calves on natural and/or cultivated pastures has, however, received scant attention in South Africa.

The experiment was conducted on a commercial property in the north-western Orange Free State described as Kalahari thornveld and shrub bushveld[1]. Bonsmara-type male calves (n=80), born from mid-September to the end of November 1989, were allocated randomly within age of dam to a treatment (implanted) and a control (non-implanted) group (40 per group) for 90 d. Calves were identified at birth and castrated and dehorned within 14 d. The treatment group received a testosterone-oestradiol combination (testosterone-propionate 200 mg, oestradiolbenzoate 20 mg (F-TO^R, Upjohn (Pty) Ltd) in mid-February 1990 at approximately 120 d of age, and both groups were weighed on the same day. Cows grazed natural pasture, which was alternated at intervals of 10 d with cultivated babala *(Pennisetum americanum)* pastures in February and March. Both groups were weaned and weighed in mid-May 1990.

Differences were determined statistically in a one-way analysis of variance[5].

Calves which had received implants, grew significantly (P<0,005) faster than the control group over the trial period (Table 1). Average weaning mass attained by the treatment group, was higher (P<0,005) than that of the control group. The 17,5% superior growth rate is more than the 9,8% reported elsewhere[3] with calves implanted with zeranol. Pretreated average daily gain (ADG) of calves was about 1 042 grams per day (assuming an average birth mass of 37 kg) (D J Bosman, Beef Cattle Performance Testing Scheme, ADSRI, Private Bag X2, 1675 Irene, personal communication). The ADG for the treatment group (1 043 grams per day) over the trial period then indicates not so much an increase in growth rate as a prevention of decline in growth rate as observed in the control group (888 grams per day). This confirms the probable dual pharmacological action of this product as stated by the manufacturer, i.e. a direct prevention of protein catabolism by glucocorticoids, replaced by the androgenic substance in the muscle tissue cells, as well as an indirect enhancement of the protein deposition by the oestrogenic substance.

At variable weaner calf prices, the implanted calves were worth substantially more (above implantation cost) than non-implanted calves. Even considering the present feedlot scenario, where calves with a low initial mass are desired, the non-implanted calves in this example would need to command an unrealistic premium to be equivalent to the implanted calves. Alternatively, the implanted calves would have to be worth much less to be equal to the controls. Both possibilities seem unlikely under present circumstances.

This trial raises the following questions which deserve further research: it needs to be established whether implanted calves retain their mass advantage

Table 1: **Body mass changes of steer calves with (treatment) and without (control) implants**

	n	Start (kg ± SD**)	Wean (kg ± SD**)	Gain (kg)	ADG*** (g/d)
Treatment	40	162,8 ± 21,90	256,7 ± 26,9	93,9	1043
Control	40	158,2 ± 19,80	239,1 ± 25,4	79,9	888
Significance		NS* (F=0,98)	P<0,005 (F=9,19)	P<0,005 (F=34,83)	

* NS = Non-significant
** SD = Standard deviation of mean
***ADG = Average daily gain over trial period (90 d)

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throughout the feedlot period, or if non-implanted calves exhibit sufficient compensatory growth after receiving an implant at the feedlot, to be comparable at the end of the feedlot phase; the possibility of producing finished, or near-finished carcasses at slightly older weaning ages (±9-10 months) with implanted calves being creepfed to increase preweaning growth rate, or, alternatively, to shorten the postweaning finishing period sufficiently to enable more farmers to finish their calves, instead of selling to feedlots, needs investigation; the effect of re-implantation with the same and different combinations of growth promoters under both pasture and feedlot conditions, warrants further research; the effect of implants with and without creep feeding on the accuracy of performance testing, and thus selection in the cow herd, should be determined.

The correct use of growth promoters can contribute to an increased income from the sale of weaner calves, and unless feedlots are prepared to pay a substantial premium for calves of low weaning mass, weaner producers can benefit from the use of growth promoters.

It needs to be emphasised, however, that this technique will only be worthwhile when cows produce sufficient milk, and poor results should be expected if cows, for whatever reason, are incapable of weaning relatively heavy calves without the aid of growth promoters[3].

ACKNOWLEDGEMENT

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Book Review/Boekresensie

DIE KLINISCHE UNTERSUCHUNG DES RINDES

G ROSENBERGER

3e Hersiene en uitgebreide uitgawe (onder redaksie van G. Dirksen, H.-D. Gründer en M. Stöber). Verlag Paul Parey, Lindenstrasse 44-47, D-1000 Berlin 61. 1990, pp 718, 676 illustrasies insluitend 21 kleurplate, 76 tabelle. Prys nie vermeld nie (ISBN 3-489-56516- 9).

Rosenberger se handboek oor die kliniese ondersoek van die bees word algemeen as gesaghebbend aanvaar. Die tweede uitgawe (1977) is in Engels, Frans, Italiaans, Spaans, Japanees en Portugees vertaal. Na die afsterwe van prof Rosenberger in 1983, is die handboek deeglik hersien en uitgebrei onder 'n nuwe redaksie. Hierdie uitgawe is keurig versorg, met drukwerk van hoë gehalte, pragtige illustrasies en 'n dienlike buiteblad.

In die eerste hoofstuk word hantering, berusting en bedwang van die bees uiteengesit, asook toedieningswyses van lokale verdowing en algemene narkose. Die tweede hoofstuk handel oor uitkenmerke van individuele diere, anamnese, grondbeginsels van ondersoektegnieke en algemene ondersoeke. Daarna word 'n hoofstuk aan elk van die orgaanstelsels gewy. Oral word prosedures en toestande breedvoerig aan die hand van diagramme, sketse en foto's verduidelik. Daar word ook ruim van tabelle gebruik gemaak om bv. differensiële diagnoses aan te dui.

Die langste hoofstukke is dié een wat oor die spysverteringstelsel en die geslagstelsel handel. Lg. sluit selfs 'n breedvoerige uiteensetting van semenevaluering en van obstetriese ondersoek in.

Aan die einde van elke hoofstuk word die vervaardigers of verskaffers van genoemde apparaat, instrumente of middels gelys. Alhoewel hulle feitlik uitsluitlik in Duitsland gesetel is, behoort dit tog plaaslike veeartse of klinieke in staat te stel om spesifieke, gespesialiseerde items te bekom.

Hierdie handboek word met vrymoedigheid aanbeveel by alle grootdierpraktisyns wat 'n leeskennis van Duits het. Die prys is nie vermeld nie, maar met die huidige swak wisselkoers sal 'n boek van hierdie gehalte etlike honderde Rande kos. Om dié rede is die resensie-eksemplaar aan die biblioteek van die Fakulteit Veeartsenykunde, Universiteit van Pretoria, geskenk ten einde dit tot die beskikking te stel van alle belanghebbendes.

B L PENZHORN

LEAD POISONING IN A DOG

J H WILLIAMS* and M C WILLIAMS**

ABSTRACT

A case of lead poisoning caused by ingestion of a lead roof-washer is described in a one-year-old, spayed Fox Terrier bitch, presented with nervous signs, and basophilic stippling of red blood cells. Blood concentrations of lead were in the low toxic range. Radiography of the abdomen revealed radio-dense objects in the stomach, which on gastrotomy included a lead roof-washer. Prior to removal of the foreign bodies, the dog showed remarkable improvement on non-specific symptomatic treatment alone, and recovered well after surgery, only to die unexpectedly several hours later. Concentrations of lead in the liver and kidneys were extremely high, and histology revealed typical intracellular inclusions and organ degeneration. In the light of these findings, it is suggested that all cases of suspected or confirmed lead poisoning be given specific chelation therapy.

Key words: Poisoning, lead, dog.

Williams J.H.; Williams M.C. **Lead poisoning in a dog.** *Journal of the South African Veterinary Association* (1990) 61 No. 4, 178-181 (En.) Department of Companion Animal Medicine and Surgery, Faculty of Veterinary Science, Medical University of Southern Africa, 0204 Medunsa, Republic of South Africa.

INTRODUCTION

Lead poisoning in dogs has been comprehensively researched, reported and reviewed in the literature[1-10], but despite being regarded as a "common" poisoning elsewhere, lead poisoning in dogs has not been reported in the Republic of South Africa. Twenty-eight cases of lead poisoning in dogs and cats were reported at Broken Hill Mine Township in Zambia[6]. It was found that the lead content of soil in the area where cases occurred, was high. In houses where some of the cases occurred, smelter ash (high in lead) from the mine was invariably distributed on their driveways. Cases where older dogs were involved, animals presented with clinical signs of restlessness and abdominal pain and occasionally with vomiting and/or diarrhoea which was sometimes bloodstained. Younger animals often showed intermittent bouts of nervous signs characterised by anxiety, hyperexcitability, fear, hysteria, continuous barking and convulsions.

The incidence of lead poisoning in dogs is reportedly seasonal[9] with most cases occurring in summer and autumn. This may be due to a decrease in faecal and urinary excretion of lead which occurs at high environmental temperatures[9], and/or increased amounts of vitamin D produced, following prolonged exposure to sunlight, thus promoting intestinal absorption of ingested lead[8]. There is probably no breed predilection, although in one survey Poodles were highly represented[9]. Most affected dogs are younger than one year old, this age-group being most prone to chewing and eating various oddments which could include lead-containing materials such as certain paints (no longer the threat it was in the first half of this century), linoleum and building materials[8 9]. Other specific sources mentioned include weights, toys, fishing sinkers, golf balls, putty, lead-glazed containers used as receptacles for water or food[7], batteries and solder[8]. A source of possibly-increasing importance is atmospheric lead from vehicles combusting leaded gasoline (which prevails in very high concentrations at dog-nose level on busy streets)[4 9]. It could be expected that cats, due to their grooming behaviour, might also ingest lead-containing fallout on their coats, in addition to inhaling it in congested urban areas.

The presenting clinical signs recorded in lead-poisoned dogs[3 7 8] include gastrointestinal dysfunction (anorexia, abdominal pain, vomition, diarrhoea or constipation); nervous disorders (clonic-tonic convulsions, hysteria, tremors, nervousness, behavioural changes, champing fits, paraplegia, muscle spasms, hyperaesthesia, blindness, deafness, retraction of the eyeballs with resultant protrusion of the nictitating membranes, Horner's syndrome, photophobia, miosis, incoordination and rarely, oesophageal paralysis); and pallor of the mucous membranes. Convulsions, if present, may show an extreme variation in frequency and duration. Sometimes there is weakness, weight loss and sialosis. Very occasionally there is a Burtonian line on the gums and in a few young dogs with active growth plates, there may be radiographic or post mortal evidence of "lead lines" in the metaphyses of long bones[7]. Radiographically these lead lines appear as densely sclerotic bands, 2-4 mm wide and, if present, are most easily visualised in the distal radius and ulna. In some animals mild proteinuria[7 9], casts and occasionally glycosuria[7 9] may be seen, but often urinalysis and blood urea concentrations are normal[3]. There may be excess concentrations of urobilinogen[7 9] and delta amino levulinic acid[2 5 7] in the urine.

Haematological changes include basophilic stippling of erythrocytes, which may be "punctuate" or "reticular", and the presence of immature (especially nucleated) erythrocytes, in the absence of severe anaemia. These changes are considered almost pathognomonic for lead poisoning in dogs[4 5 7 8 9] but are not always present[3]. Pale mucous membranes may be noticeable despite a relatively normal packed cell volume. This is due to reduced haemoglobin concentration of erythrocytes. Lead disrupts heme synthesis at several points and interferes with normal maturation of red cells[9], leading to decreased oxygen-carrying capacity and increased fragility. Globin synthesis is

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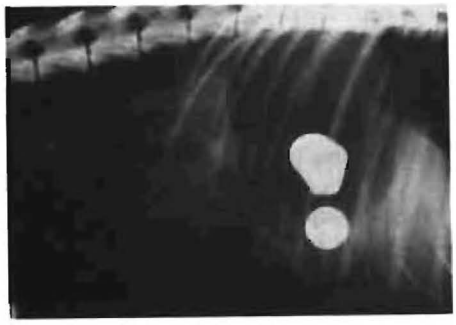

Fig. 1: **Radiodense roundish objects in the stomach of a dog suffering from lead poisoning**

Fig. 2: **Objects removed from the stomach during gastrotomy: a soft buckled roof-washer, a one-cent coin and a five-cent coin**

also partially inhibited, contributing to a decreased red cell life-span[5]. The enzymes most sensitive to inhibition by lead, are delta amino levulinic acid dehydratase (ALAD) and ferrochetalase[5]. Amino levulinic acid concentrations have been measured in the urine (U-ALA) as an indicator of lead poisoning in dogs, but results have been variable[2 5]. Due to these biochemical disruptions, a mild to moderate normocytic and hypochromic anaemia may be present[5 9]. Anisocytosis, poikilocytosis and polychromatophilia often occur. Some dogs show mild to moderate leucocytosis, usually attributable to absolute neutrophilia and a left shift. Lymphopaenia, eosinopaenia and monopaenia are other findings in these animals although a few animals have absolute leukopaenia, but monocytosis and eosinophilia[9]. Erythrocyte sedimentation rate is normal and Coombs' tests are negative[9].

Reports on bone marrow changes vary. Increases in segmented neutrophils and myeloid series cells, and increased myeloid-erythroid ratios (M:E), which decreased once lead administration had ceased, were reported in experimental dogs[4]. Bone marrow sections from 15 naturally-occurring cases[9] showed hyperplastic bone marrow, especially of erythroid elements.

An increased protein concentration[3 7 9] in cerebrospinal fluid in lead-poisoned dogs is occasionally seen.

Electro-encephalographic (EEG) abnormalities are usually intermittent, generalised, high amplitude, slow wave activity[3 9], which although not diagnostic for lead poisoning, may differentiate encephalopathy from encephalitis[3]. The slow waves vary from 1 to 4 cycles per second, with amplitudes of 100 uV to greater than 200 uV in moderately to severely affected dogs[3]. The EEGs were recorded with the animals lightly anaesthetised in one study[9], and non-sedated in another series[3].

Zook et al[9 10] state that the diagnosis of lead poisoning in dogs is fairly accurate when based on the history, clinical signs, radiography, haematological manifestations and clinical response to chelation therapy. Confirmation of the diagnosis is most meaningfully performed by measuring blood and urine concentrations compared before, and 24 h after, the initiation of calcium disodium ethylene diamine tetra-acetate (CaEDTA) therapy. Diagnostically-significant values quoted[10] are 35 microgram or more per 100 ml of blood and 75 ug or more per ℓ of urine before treatment and 821 ug or more per ℓ of urine 24 h after the start of chelation therapy. Normal blood lead concentrations in dogs are quoted as ranging from 0,02 to 0,05 mg 100 ml^{-1}, but a few dogs with concentrations of 0,04 to 0,05 mg 100 ml^{-1} did actually have lead poisoning[8]. Usually, concentrations of 0,06 mg 100 ml^{-1} or more, are considered to be diagnostic for poisoning[8]. There does not, however, seem to be a correlation between the concentration of lead in the blood and the severity or character of clinical signs, but dogs with very high blood levels tend to recover slowly and are more likely to have recurrences after termination of treatment.

Postmortally, liver specimens are the most reliable source for lead determination. Samples should be collected some distance from the gall-bladder or major bile ducts, since lead is concentrated in bile and may be imbibed into the parenchyma after death. Diagnostic levels of lead in liver (wet mass) are quoted as 3,6 μg or more per gram[10]. It has been shown that neither freezing of tissues for a few months, nor storage in 10% neutral buffered formalin for up to 5 years, affected the lead content of the stored liver tissue[10]. Kidney tissue is sometimes also analysed, but considerably more lead is found in the renal cortex, than in the medulla and hence uniform sampling is difficult[10].

In contrast to humans, hair-analysis in dogs gives unreliable results due to variable growth rates and seasonal shedding[7 10]. A value of 88 μg or more per gram of hair has nevertheless been quoted as diagnostic for dog hair[10].

Dogs which die from lead poisoning may show few gross changes at necropsy apart from meningeal congestion, occasional white "lead-lines" in the metaphyses of immature dogs, abnormally reddened bone marrow and foreign material in the gastrointestinal tract[7]. Microscopic changes are more specific and consistent, especially in the brain, metaphyses, kidneys, liver and bone marrow[7]. Lead encephalopathy is characterised by dilatation of blood vessels, swelling and necrosis of endothelial cells, hyalinisation and necrosis of some arterioles and occasionally thrombosis of capillaries. There may be oedema, fibrin and haemorrhage around these damaged vessels. Vacuolation, necrosis of neurons and gliosis may be present in the cerebral cortex. If nervous signs have persisted for more than a week, endothelial cells and new capillaries may proliferate in the cortical gray matter. The "lead-lines" in immature metaphyses consist of heavily-mineralised cartilaginous trabeculae covered with a thin layer of bone extending from the epiphyses towards the diaphyses. In most dogs characteristic, eosinophilic, acid-fast, nuclear inclusions are found in renal and hepatic epithelial cells. The bone marrow, especially the erythroid element, is hyperplastic[7 9]. Less common lesions include random necrosis of striated muscle fibres and peripheral neuropathy. The latter lesion is thought to be the cause of megaoesophagus following lead-induced vagal neuropathy. Suppression of ovarian follicles and spermatogenesis, and haemosiderosis of the liver and spleen have also been observed[7].

The approach to treatment of lead-poisoned dogs varies. Some researchers recommend[5] that patients exhibiting moderate clinical signs with no immediate threat to life, are best treated conservatively (by removal of unabsorbed lead from the digestive tract by means of laxatives, enemas, emetics or surgery), and only those with severe blood or nervous derangements should receive specific chelation therapy. Others[8], however, advise specific chelation therapy as soon as a diagnosis is made, in addition to prompt removal of the source of lead from the gut. In a recent experimental study[1] it was shown that in the absence of chelation therapy, following oral dosing of lead, several months were required for the blood lead concentrations to return to pre-dosing levels. It was also demonstrated that at this point there were

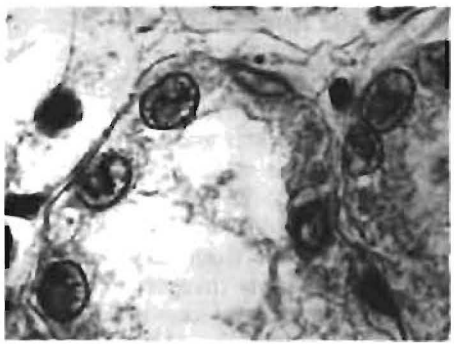

Fig. 3: **Renal proximal convoluted tubules. Large, irregular nuclear inclusions in most epithelial cells. HE X 1000**

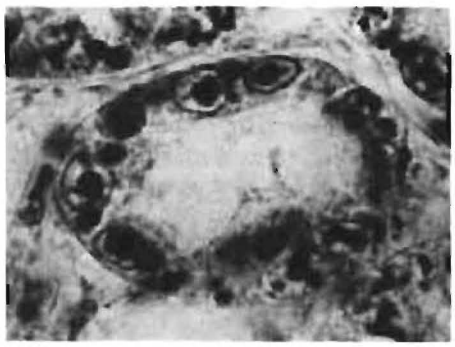

Fig. 4: **Renal proximal convoluted tubule with nuclear and cytoplasmic inclusions. Note chromatin margination. ZN x 1000**

still large amounts of lead in the bones, which would probably require several more years to eliminate.

Dimercaprol[2 5], calcium disodium ethylene diamine tetra-acetate (CaEDTA) [2,10] and penicillamine[2 5] can be used as chelating agents. CaEDTA has proved to be the most effective and has fewer adverse side-effects when compared to the others. CaEDTA forms complexes with circulating lead, leading to rapid excretion in the urine[2] and significant clinical improvement usually occurs within 24 to 48 h[2 7 8]. Shortly after commencement of treatment with CaEDTA, both nucleated and stippled red blood cells decrease rapidly in the peripheral blood and are usually absent after 5 d[9]. Additional 3-5d courses of CaEDTA at weekly intervals have been recommended if blood lead concentrations are still high or if clinical signs recur[2 8 10].

Recommended dosages of CaEDTA, routes and regimens vary eg. 22 mg kg^{-1} body weight 4 times daily for 4 to 5 d administered intravenously, subcutaneously or intraperitoneally after dilution in 5% dextrose[5]; 100 mg kg^{-1} CaEDTA divided into 3 portions administered subcutaneously over 12 h (at 4-hourly intervals) after similar dilution in 5% dextrose solution, for 5 d; per os administration of CaEDTA tablets at 100 mg kg^{-1} divided 3 times daily for 5 d; 110 mg kg^{-1} divided into 4 equal portions subcutaneously after dilution with 5% dextrose to a concentration of 10 mg CaEDTA ml^{-1} for 5 d[4 7]; and one source[10] recommends 110 mg kg^{-1} d^{-1} but not exceeding 2 gm d^{-1} total dose, similarly diluted and administered subcutaneously 4 times daily.

Sedatives[5 7 8] (eg.barbiturates), anticonvulsants[7], dexamethasone[7 8] and mannitol[8] may be given as supportive treatment, in conjunction with chelation therapy. Intravenous infusion of amino acid and glucose solutions may assist in re-establishing an adequate nutritional state in individuals with prolonged anorexia or severe nervous signs[5]. Whole blood transfusions may also be considered[5].

If properly and timeously treated, most dogs suffering from lead poisoning recover rapidly and completely[5]. The overall mortality in the USA is probably more than 15% including cases which receive no medical attention and those where the condition is misdiagnosed or the animals are euthanased or incorrectly treated[7].

CASE REPORT

A one-year-old, spayed, vaccinated, Fox Terrier bitch with a body mass of 7kg was presented in a recumbent state. The dog was showing paddling limb-movements and salivation. According to the owner the animal had shown similar clinical signs approximately 2 weeks previously. The dog's appetite and water intake had apparently been normal throughout.

A physical examination revealed generalised tonic-clonic convulsions, galloping movements which worsened when the animal was handled, sialosis and mydriasis. The nictitating membranes were retracted halfway across the eyes. There was a marked head-tilt to the right with immediate severe ataxia and falling to that side when the dog was placed in sternal recumbency. The pulse rate was 190 beats min^{-1}, the respiration rate 60 min^{-1} and rectal temperature 39°C. The mucous membranes were moderately pale and the dog was slightly dehydrated.

Examination of a peripheral blood-smear, revealed clear basophilic stippling in several erythrocytes. In addition to basophilic stippling of red cells, there were also many immature and nucleated cells. Haematological findings are presented in Table 1. Electro-encephalographic (EEG) examination (performed under general anaesthesia) and examination of cerebrospinal fluid on the second day revealed no abnormalities.

On Day 3, a blood lead concentration of 0,04 mg 100 ml^{-1} was established. On Day 4, examination of peripheral blood-smears still revealed evidence of regenerative anaemia and basophilic stippling. Radiographic examination of the skull and abdomen clearly demonstrated 2 roundish, highly radiodense objects of differing size in the stomach region (Fig. 1).

A presumptive diagnosis of lead poisoning was based on the clinical and radiographic findings, the haematological picture and the suspicious blood lead concentrations. A gastrotomy was performed and the diagnosis confirmed when a lead roof-washer was removed from the stomach, together with a one-cent and a five-cent coin (Fig. 2).

During the first 24 h after admission, the dog was kept under general barbiturate anaesthesia, but thereafter was not sedated apart from a short period of general anaesthesia during which the EEG was performed and CSF collected. On admission, the dog had been rehydrated with a polyionic isotonic fluid and maintained on intravenous fluid. Two injections of atropine sulphate were given on Day 1 in an attempt to control sialosis. On the third day, a parenteral feeding preparation containing multivitamins, electrolytes, essential amino acids and fatty acids was administered in the drip.

When the dog awoke from the barbiturate sedation, continual, slow, head movements to the left and back while lying on her right side, were observed. No muscle twitches or head movements were observed while the animal was asleep.

By Day 3, there was an improvement in habitus, head movements were less marked and the dog seemed to be aware of its surroundings. By Day 4, it was able to lie in sternal recumbency. At this stage the drip was removed and a milk powder preparation administered per stomach tube although the patient did try to lap from a bowl.

Following the gastrotomy performed on Day 4, the dog recovered well from anaesthesia and sat up on her own during the same evening. She was treated with intravenous fluids, whole fresh blood and an antibiotic injection, but died unexpectedly during the night. At no stage was CaEDTA treatment given, because this was not considered to be necessary because of blood lead concentrations which had not seemed remarkably elevated.

Necropsy findings included mild generalised cyanosis; slight diffuse meningeal congestion of the brain; acute, diffuse hepatic degeneration; moderate splenomegaly, with focal disseminate splenic parenchymal suggilations; and severe focal pulmonary haemorrhage with focal disseminate alveolar emphysema. There was mild serosanguinous ascites. The small intestines showed a few scat-

Table 1: **Haematological changes in a dog with lead intoxication**

	DAY 1	DAY 3
Haemoglobin g ℓ^{-1}	164	70
Red cell count $1x10^{12}$ ℓ^{-1}	6,57	3,28
Haematocrit	0,43	0,22
Mean cell volume fl	67	66
White cell count $1x10^{9}$ ℓ^{-1}	37,5	12,1
Neutrophils % (mature)	0,65	0,76
(immature)	0,27	0,12
Lymphocytes %	0,08	0,07
Monocytes %	0	0,05
Eosinophils %	0	0
Thrombocytes $1x10^{9}$ ℓ^{-1}	Normal	Normal
Anisocytosis	2+	
Polychromatophilia	4+	
Normoblasts	3+	
Basophilic stippling	2+	+
Reticulocytosis	4+	4+

tered areas of mucosal hyperaemia and there was severe hyperaemia of the colonic mucosa and the presence of meleana.

Toxicological examination of samples of liver and kidney were found to have 129,5 and 390 μg of lead respectively per g of tissue, determined on a wet basis by atomic absorption (Perkin-Elmer BP5000 Instrument).

Histologically, the kidney glomeruli showed moderate widening of Bowmans' spaces with aggregated globules of eosinophilic material lining the periphery in many glomeruli. Some tubules were lined with degenerating epithelial cells which had granular eosinophilic cytoplasm and showed variable nuclear pycnosis. Other tubules had dilated lumens with flattened epithelial cells. Aggregated eosinophilic globules were present in the lumen of some tubules. Many of the tubule epithelial cells had enlarged, vesicular nuclei with chromatin margination. These nuclei contained one to a few large, pale, eosinophilic inclusions which were often surrounded by a number of small purplish chromatin granules. The inclusions were round, oval or irregular in shape (Fig. 3). A few large regenerating cells and mitotic figures were present. Inclusions were absent in the regenerating cells. In Ziehl-Neelsen stained sections, acid-fast inclusions of varying size and shape were observed in the nuclei and cytoplasm of tubule epithelial cells and were larger in the nucleus (Fig. 4). These inclusions were noted in especially the dilated tubules with flattened epithelium.

Hepatocytes in the liver sections showed varying degrees of acute cellular swelling and many of them had small nuclear inclusions which were acid-fast, but could also be seen in Haematoxylin-eosin stained sections.

A few small foci of myofibre vacuolation and necrosis were observed in the myocardium. Mild fibrosis accompanied these lesions. There was moderate congestion of meningeal blood vessels, and widespread, variable neuronal degeneration and scattered necrotic neurons were found in the brain. No significant lesions were found in the lung and spleen.

DISCUSSION

In this case there was a poor correlation between lead concentrations in the blood and lead concentrations in kidney and liver samples. Blood concentrations were in the lower positive range while large concentrations of lead were present in the kidneys and liver. This would suggest that specific chelation therapy should be instituted from the outset, even if the patient shows clinical and haematological improvement with conservative treatment. This applies also to cases (as in this one) where the source of poisoning has been removed.

Such high organ-concentrations of lead would probably have required several courses of chelation therapy to eliminate most of the lead. Ideally, monitoring of urine and blood lead concentrations before and after chelation therapy should be done to ascertain efficacy of the treatment. Basophilic stippling of red cells is not very specific in humans and certain other animal species, but it appears to be virtually diagnostic of lead poisoning in dogs[7]: hence if laboratory facilities are not available to determine blood levels of lead, stippled red cells, together with other suggestive clinical signs, allows for a presumptive diagnosis of lead poisoning to be made and specific (and supportive, if necessary) treatment to be instituted.

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POLYCYTHAEMIA VERA IN A DOG

J M WYSOKE* and J VAN HEERDEN*

ABSTRACT:
An 8-month-old Dachshund showed signs of severe depression and intermittent, mild generalised seizures. A diagnosis of polycythaemia vera was made and the clinical signs ascribed to the hyperviscosity associated with this condition. Treatment by phlebotomy was initiated before the dog died.
Key words: Polycythaemia vera, seizures, nervous signs, hyperviscosity.

Wysoke J.M.; Van Heerden J. **Polycythaemia vera in a dog.** *Journal of the South African Veterinary Association* (1990) 61 No. 4, 182-183 (En.) Department of Companion Animal Medicine and Surgery, Faculty of Veterinary Science, Medical University of Southern Africa, PO Box

The term polycythaemia refers to an increase in the number of red blood cells per unit volume of blood[1 4]. A red cell count of greater than 10 x 10^{12} ℓ^{-1} and an haematocrit greater than 0,65 is indicative of an increased red cell mass and polycythaemia[1]. Polycythaemias are further defined as being relative or absolute according to the presence or absence of a red cell production abnormality[1 3 6]. Relative polycythaemias have no underlying red cell production abnormality, but result from excessive loss of body fluids due to vomition, diarrhoea or hyperthermia or as a result of catecholamine-induced splenic contraction[1 3 4]. Relative polycythaemia is usually confirmed from the history and the response to rehydration therapy.

Absolute secondary polycythaemia is associated with organ pathology and an increase in erythropoietin secretion or the secretion of erythropoietic factors[1 3 6]. Erythropoietin secretion may be appropriate or inappropriate. Excessive appropriate secretion of erythropoietin may occur in response to systemic hypoxia secondary to pulmonary or cardiac disease[1]. Congenital heart disease in dogs such as tetralogy of Fallot, reversed ventricular septal defects with Eisenmenger's complex and reversed patent ductus arteriosus with right to left shunting of blood, result in venous admixture of oxygenated blood, with resultant systemic hypoxia and increased erythropoietin synthesis and release[1]. A similar sequence of events follows the hypoxaemia as a result of pulmonary emphysema, bronchiectasis or alveolar hypoventilation due to marked obesity[1]. The inappropriate secretion of excessive quantities of erythropoietin in man, has been associated as a paraneoplastic syndrome with renal tumors, hepatomas, uterine leiomyomas and cerebellar haemangioblastomas[5]. A similar syndrome has been reported in dogs with renal carcinoma and lymphosarcoma[5 6].

Polycythaemia vera or absolute primary polycythaemia is defined as an absolute increase in the number of red blood cells per unit volume of blood, with associated low levels of erythropoietin, due to a rare myeloproliferative disorder in dogs and cats[1 3]. Polycythaemia vera is thought to be due to the proliferation of clonal cells excessively sensitive to stimulation by erythropoietin[2]. The origin of these clonal cells is unknown and a viral agent, which causes a similar syndrome in certain strains of mice, has been speculated upon[2]. The evolvement of primary polycythaemia into myelofibrosis with myeloid metaplasia or into acute myeloid leukaemia, has been reported in man[2]. Changes in the manifestation of myeloproliferative disorders have also been reported in other species, which should encourage constant re-evaluation of a patient diagnosed with polycythaemia vera[2]. Hyperadrenocorticism may result in an elevation in the red cell parameters, but not to the extent that is found in polycythaemia vera cases[4].

This paper describes a case of absolute primary polycythaemia (polycythaemia vera) and symptoms of the associated hyperviscosity syndrome.

An 8-month-old male Dachshund was presented with a history of having had 3 mild generalised seizures in one day. Prior to presentation, the dog was treated with 30 mg of phenobarbitone orally (Lethyl, Lennon) once only, but remained severely depressed, anorexic and adipsic. Initial examination of the dog revealed that the dog was somnolent and unresponsive to external stimuli. Despite the profound depression, the hydration status assessed by skinfold pliability and oral mucosal moistness, appeared to be normal. The rectal temperature was 38°C and the femoral pulse could not be palpated. Auscultation revealed the heart rate to be 165 beats per min with very low-intensity heart sounds. The ocular and oral mucosae were extremely hyperaemic and opthalmoscopic examination of the retina revealed severely dilated and tortuous retinal blood vessels. On abdominal palpation, splenomegaly was detected. Only one testis had descended into the scrotum. It was not possible to palpate the second testis abdominally. Continuous muscle tremors were a prominent clinical sign.

Blood was submitted for haematological and chemical analysis, with the results of these tests reported in Table 2. The most striking result was an haematocrit of 0,81 and a tentative diagnosis of polycythaemia was made.

Further investigation was deemed necessary to rule out possible causes of a secondary polycythaemia. Under general anaesthesia a bone marrow biopsy, a cisternal cerebrospinal fluid (CSF) tap, laparotomy and orchidectomy were performed. Examination of the bone marrow biopsy revealed areas of erythroid hyperplasia. There were very few megakaryocytes. The total protein of the cerebrospinal fluid was normal at 0,24 g ℓ^{-1}. Direct visualisation of the abdominal organs detected no obvious neoplastic or cystic structures as a possible extraneous source of erythropoietin. Both the intra-

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Table 1: **Classification of polycythaemia+**

Relative
Normal red blood cell mass with increased haematocrit due to decreased plasma volume or splenic contraction.
Absolute
Increased circulating red blood cell mass due to sustained increased cell production.
Primary (polycythaemia vera)
Myeloproliferative disorder of erythroid series in presence of subnormal serum erythropoietin.
Secondary
Increased erythrocyte production due to excess erythropoietin or erythropoietin-like factor.
- appropriate
associated with systemic hypoxia, e.g. cardiac or pulmonary disease
- inappropriate
absence of systemic hypoxia, e.g. renal disease, various neoplasms

+ Adapted from Waters & Prueter[6]

Table 2: **Laboratory variables measured on initial presentation of a case of polycythaemia vera**

Haemoglobin (g ℓ^{-1})	285
Red cell count (x 10^{12} ℓ^{-1})	16
Haematocrit	0,81
White cell count (x 10^{9} ℓ^{-1})	15,0
Platelets (x 10^{9} ℓ^{-1})	56,0
Urea (mmol ℓ^{-1})	3,3
Creatinine (umol ℓ^{-1})	33
Sodium (mmol ℓ^{-1})	152
Potassium (mmol ℓ^{-1})	5,5
Total serum protein (g ℓ^{-1})	59
CSF protein (g ℓ^{-1})	0,24
Arterial pO_2 (mmHg)	84
Arterial pCO_2 (mmHg)	42

and extra-abdominal testes were removed and submitted for histopathological examination. The intra-abdominal testis was macroscopically small and hypoplastic when compared to the descended testis. No evidence of spermatogenesis or abnormal endocrine tissue proliferation was found on microscopic examination of the testes. The blood gas analysis of blood obtained from the femoral artery, revealed no abnormality (Table 2). On the basis of these results, a diagnosis of absolute primary polycythaemia (polycythaemia vera) was made.

Facilities to carry out erythropoietin assays were not available at local laboratories and the diagnosis was made by attempting to exclude all other possible causes of secondary polycythaemias.

A therapeutic phlebotomy was performed and 80 ml of blood was withdrawn, which was replaced with an equal quantity of a balanced, electrolyte solution (Balsol, Labethica).

The haematocrit following the phlebotomy was 0,62. The habitus and appetite improved over the next 3 d and the dog was discharged into the care of the owner with instructions that he be returned for weekly examinations. The dog suffocated to death at home 2 d after discharge when a piece of meat, which the owner had fed it, lodged in its pharynx. The owner reported a good habitus and appetite until the time of death.

Very few cases of absolute primary polycythaemia (polycythaemia vera) have been reported in the veterinary literature and as a result, it is difficult to report on a breed, age or sex predisposition[4,5]. Most of the clinical signs are related to the increased red blood cell mass and the concomitant hyperviscosity[1,4]. As a result of sluggish blood flow through the capillary network, a larger than normal proportion of haemoglobin is deoxygenated[3]. Neurological signs are therefore possibly due to central nervous system hypoxia[3]. It is therefore difficult to relate the clinical signs to a specific disease process, as central nervous system hypoxia has a varied clinical manifestation. The clinical signs that should alert one to the presence of a polycythaemia, are the profound redness of the mucous membranes and the dilated, tortuous appearance of the retinal blood vessels. No suitable explanation for the thrombocytopaenia could be found. Consumption of blood platelets as a result of hypercoagulability, due to the sluggish blood flow, may contribute to thrombocytopaenia[6], but this would not explain the decreased number of megakaryocytes in the bone marrow of this case.

Various therapeutic approaches have been used, but the common goal is to maintain a packed cell volume of 0,60. This can be achieved by removal of 20 ml kg^{-1} of blood at 3 d intervals until the desired packed cell volume is achieved, or by combining phlebotomy and cytotoxic agents for inducing bone marrow suppression[1,3]. Chlorambucil, busulphan and hydroxyurea have been used[4,5]. Chlorambucil is preferred to busulphan as it is less toxic and safer in thrombocytopaenic patients[3]. Hydroxyurea has been preferred as it inhibits DNA synthesis, but does not affect RNA and protein synthesis, resulting in a reversible bone marrow depression[5].

Although the definitive test for the diagnosis of absolute primary polycythaemia is the presence of decreased levels of erythropoietin, this assay was unavailable when the case was presented. The diagnosis is therefore made by exclusion of all other causes of a polycythaemia. The diagnosis and management of a patient with polycythaemia presents a challenge to the veterinarian but more important, it affirms the importance of a routine haematological examination of all patients with non-specific clinical signs and neurological disease.

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GOLD MEDAL OF THE SAVA : 1990
GOUE MEDALJE VAN DIE SAVV : 1990

PROF D R OSTERHOFF

The Gold Medal of the Association is awarded annually to any person resident in South Africa (RSA and TBVC - countries), or to a veterinarian who is not resident in South Africa, but who is a member of the SAVA, in recognition of outstanding scientific achievement with relevance to Veterinary Science or of a meaningful contribution to the advancement of Veterinary Science. The 1990 Gold Medal is awarded to Prof D R Osterhoff for his active and enthusiastic involvement in teaching, research and community service over the last almost 3 decades, during which time he has given of his time and energy in an unstinting way to promote the cause of the profession both nationally and internationally. In particular, we recognise his scientific contribution in the field of genetics and animal blood groups and the national and international recognition he achieved through his endeavours as evidenced by the many invitations to international meetings and the large number of scientific publications to his credit.

Diedrich Richard Osterhoff was born on 14 March 1925 in Hemer, Westfalen, West Germany. He completed his schooling in Germany and obtained the Agricultural College Certificate in 1947, a Diploma in Agricultural Engineering from the University of Giessen in 1951 and a doctorate in Agriculture under Prof I Johansson of Uppsala, Sweden in 1956. He moved to South Africa in the same year to take up a position as Professional Officer on contract in the Division of Animal Breeding and Dairy Products where he was responsible for the establishment of the South African Blood Group Laboratory at Onderstepoort.

In 1961, Prof Osterhoff was appointed as Professional Officer on a permanent basis at the Research Institute for Animal Breeding and Dairy Products. During 1963, he joined the University of Pretoria as Professor and Head of the Department of Zootechnology in the Faculty of Veterinary Science, Onderstepoort, a position he held until his retirement earlier this year.

Prof Osterhoff is well-renowned in academic circles, both nationally and internationally. He has devoted the last 27 years of his life to a teaching career in Zootechnology and more recently in Ethology. Over the years he has built up a department, which has provided the major portion of our country's veterinary graduates, with a sound grounding in veterinary zootechnology and related subjects. Apart from maintaining a high academic standard in his formal lectures, Prof Osterhoff has also ensured that his students have been exposed to the realities of livestock production.

Further evidence of his international recognition as a teacher, can be found in the many invitations he has received to act as guest lecturer at other universities over the years. In 1972 he was guest professor in Lourenco Marques, Mozambique; in 1974 at the University of Gent, Belgium; in 1976 at the University of Berlin and the University of München in Germany; in 1981 in Paraguay and Uruguay; and in 1984 in Helsinki, Finland. In addition to the above, Prof Osterhoff has also been involved in several refresher courses for veterinarians as well as in a number of courses presented to outside organisations, farmer's groups and breeder's associations.

Over the years Prof Osterhoff attended many postgraduate courses ranging from genetics, artificial insemination, immunology and biotechnology, to statistics and computerisation. He also participated in formal post-graduate teaching, acting as promotor and co-promotor for several post-gradute students in other university departments and at other universities. Under his guidance and leadership, 2 doctorates and 2 master's degrees have been awarded to date in his department, while a further 3 doctorates and one master's degree are nearing completion.

Community service has been a hallmark of the endeavours of Diedrich Osterhoff during his entire career. In addition to his contributions to continuing education mentioned above, he has also served the livestock industry with dedication. As an animal scientist by basic training, he has served on many committees and breed societies and has contributed to the activities of many scientific and other societies, either as active member, or as chairman. He has, at all times in this capacity, promoted the veterinary profession in these committees and societies and has strived towards better relations between the veterinary and animal science professions. This was done at all levels of contact and it has made a significant contribution towards bringing the two professions closer together. This component of his contribution to veterinary science and the profession alone, deserves full and appropriate recognition.

The research interests of Prof Osterhoff have essentially centered around the blood groups of domestic and wild animals. He was responsible for the establishment of the first blood group laboratory in the country. It now renders an essential service to the community at large; from researchers to breed societies, to the thoroughbred industry. His work carries national and international recognition as a result of which he has received innumerable invitations to present his findings. He attended no fewer than 14 large congresses overseas, participated in 2 specialist workshops on blood typing in Thoroughbred horses in the USA and Australia and received no fewer than 15 invitations to international meetings, conferences, etc. during the period 1970 - 1989 with venues ranging from Europe, Scandinavia, South America and the USA to Japan, Taiwan and Russia.

In pursuing his research activities, Prof Osterhoff was also instrumental in the development of the department of Zootechnology as it was formerly known and of the Department of Veterinary Ethology as it is currently known of the Faculty of Veterinary Science at the University of Pretoria. This included the creation of stabling facilities, animal handling facilities, a milking parlour and many more. Under his guidance a cytogenetics laboratory was also established. In more recent years, his dedication and enthusiasm have led to the establishment of the Epol Professorship in Companion Animal Zootechnology and Nutrition in the department as well as the establishment of a nutrition laboratory.

Research conducted by or under the guidance of Prof Osterhoff can be broadly divided into 5 fields of study:

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1. Blood groups and genetic markers in animals
2. Chromosome studies in animals
3. Ethological studies in animals
4. Veterinary nutritional problems
5. Human/animal interactions

No fewer than 182 scientific publications and 58 popular and review articles have flowed from the pen of Prof Osterhoff over the last 36 years. Many of these were directed at identifying new blood groups and other genetic markers in domestic animals, resulting in the inclusion of specific aspects of the identification and correct registration of calves born after importation of semen from other countries and those born after embryo transfer in the appropriate legislation.

Breed comparison on the basis of genetic markers led to an agreement that an infusion of Gelbvieh blood into the South Devon breed was accepted, resulting in the importation of semen of Gelbvieh bulls for the improvement of South Devon cattle from 1977 onwards. Blood group studies have proved that the Drakensberger breed originated from an early cross between Friesland and Afrikaner cattle while determination of genetic markers of the Dohne and Walrich-Merino sheep breeds led to the amalgamation of the two breeds in 1987.

Another very important aspect of the research conducted by Prof Osterhoff has been the development of an agreement with the Jockey Club of South Africa concluded in 1989, whereby yearly blood typing of 4 000 Thoroughbred horses will be performed by the blood typing laboratory.

Other research projects deal with nutritional aspects of cattle, horses and sheep. Feed additives have been tested and rations with growth stimulants have been developed. Feeding in relation to sperm production has been investigated; anticoccidia compounds have been envestigated; calf raising trails have been performed to provide practical techniques for the cheap raising of calves and many other animals.

In addition, Prof Osterhoff has succeeded in maintaining contact with scientists from all over the world. This has been achieved notwithstanding the relative isolation of South Africa in the international community. Several internationally recognised scientists from as far afield as Germany, France, Sweden, Holland, Denmark and Belgium accepted invitations to visit South Africa and contributed to the activities of the laboratories under the control of Prof Osterhoff, as well as to the improvement of the veterinary course offered by the University of Pretoria. Several other scientists also visited these laboratories at various times; some of them spending several weeks and even months in the various laboratories, participating in the research activities or gaining experience in techniques and procedures.

Future immunogenetical research, based on the initial work of Prof Osterhoff, will continue and will concentrate on lymphocyte antigens. This research is directly aimed at the identification of marker genes and the identification of disease resistance in domestic animals.

Prof Osterhoff is also a man of many other talents. He is a family man with wide-ranging interests besides blood groups and animal genetics. He has the specific talent of being able to speak, besides English and Afrikaans, 5 other languages, viz. German, Swedish, French, Norwegian and Danish.

Prof Diedrich Osterhoff is a recognised leader in his field. As a direct result of the achievements listed above, Prof Osterhoff has already received recognition by having been awarded, amongst others, the Havenga Prize for Agriculture in 1969, the Faculty medal of the Faculty of Veterinary Science of the University of Gent, Belgium in 1974 and the Silver Medal for Research of the South African Society for Animal Production in 1978. He has undoubtedly also made a signal contribution towards the growth, promotion and development of veterinary science over many years. As an honorary Associate member of the SAVA he has served both our Association and the profession in an excellent manner. His positive attitude towards the profession, and the unbounding energy and considerable effort with which he has served our interests are most praiseworthy. It is therefore a privilege for the South African Veterinary Association to award the 1990 Gold Medal to Prof D R Osterhoff.

SILVER MEDAL OF THE SAVA : 1990
SILWER MEDALJE VAN DIE SAVV : 1990

PROF C M VEARY

The Silver Medal of the Association is awarded annually to any veterinarian registered with the South African Veterinary Council in acknowledgement of outstanding service to and advancement of the veterinary profession or calling in South Africa. The 1990 Silver Medal is awarded to Prof C M "Tubby" Veary for his outstanding service to the profession in the field of public health and more specifically in meat hygiene. Not only has he succeeded in promoting the ideals and the image of the profession in public health, but also the veterinary profession's role in providing non-private practice services in meat hygiene. He is furthermore also honoured for his service to the profession in his capacity as an academic and a scientist in teaching, research and community service.

Courtney Martin Veary was born in Johannesburg on 21 September 1939 and matriculated from the St John's College in Johannesburg in 1956. He obtained the BVSc degree in 1963 and the Diploma in Veterinary Public Health in 1978, both from the University of Pretoria. He started his professional career as an assistant in predominantly small animal practices in Johannesburg and Durban during the years 1964 and 1969. He also held the position of race course veterinarian for the Jockey Club of South Africa for a short time in 1968/69. In the same year he moved "overseas" to Durban where he was employed by the Durban City Council as a Veterinary Meat Hygiene Officer, a post he held until 1975 when he became State Veterinarian at the Durban Abattoir. During 1979 "Tubby" was transferred to the Natal Regional Office in Pietermaritzburg as Regional Meat Hygiene Officer for Natal.

Due to his dedicated committment, professionalism and integrity, promotion came in 1982 when he was appointed as Assistant Director, Meat Hygiene in the Transvaal Regional Office. He held this position for 3 years and then transferred to the Faculty of Veterinary Science of the University of Pretoria in 1985 where he currently still holds the post of Associate Professor in the Department of Veterinary Public Health.

During his career in Veterinary Public Health, "Tubby" has served the interests of the profession with dedication and enthusiasm. He has been active in veterinary meat hygiene for 21 years during which time he has established himself as a leader in this field in the RSA. He has made a considerable contribution towards veterinary education in the field of meat hygiene. He has attended numerous congresses and workshops and has freely given of his time and energy to various continuing education courses, farmer's days, and research programmes.

In this capacity, his work on the culling of springbok and the export of venison, was enthusiasitically received at the International Symposium of the WAVFH in Stockholm in 1989. Considerable interest in this project has also been shown by De Beer's Consolidated Mines and the Department of Agriculture. In addition, Prof Veary is also involved in research on arthritis in slaughter pigs and the microbiology of goose carcasses.

In addition to his participation in formal post-graduate education and various continuing education courses, Prof Veary also acted as chairman and convenor of a mastitis workshop during 1988. This workshop was particularly successful in bringing together for the first time in South Africa, veterinarians, producers, farmers and members of the trade, in a discussion on mastitis and its prevention. It led to the formulation of a policy on the modular herd control of mastitis, the proceedings of which now serve as a reference work for the effective control of mastitis in South Africa.

Notwithstanding the activities described above, Prof Veary also found the time to be active in community service, a typical example of his unselfish and enthusiastic endeavours to serve the profession. He currently serves on the Abattoir Training Control Committee through ministerial appointment; he is a member of the Departmental Committee of Investigation into Condemnations; he is the faculty representative on the Red Meat Producer's Organisation of the SA Agricultural Union Committee on Condemnations; he also sits on the Executive Committee of the National Game Organisation of the SAAU and serves as consultant on poultry abattoir and quality control matters to a leading poultry concern. He has also served as SAVA representative on the Community Health Association of Southern Africa for several years and was co-opted onto the Executive Committee in 1989. In all these activities, Prof Veary has contributed significantly to the role of the veterinarian in public health and veterinary meat hygiene and the promotion of the interest of the profession.

"Tubby" Veary has also excelled in his service to the profession in his activities within the SAVA. His committment stretches over 26 years. He joined the Association in 1964 and served the Natal branch uninterruptedly in various capacities from secretary to chairman during the period 1968 - 1981. On moving to Pretoria in 1981, he joined the Northern Transvaal branch of which he is still a member. As branch representative, he served on Federal Council from 1970 to 1978 whereupon he became a nationally elected member from 1978 to 1982. He seved as Vice-President from 1982 to 1984 and as President from 1984 to 1986.

As can be expected from a man of his calibre, "Tubby" did not disappear from the scene after his term of office as President. He stayed on and he wanted more. He served as Immediate Past President in 1986/87 and took over the chairmanship of the Federal Council's Finance Committee as well as of the Public Health group in the same year. As "Minister of Finance", a post he held until 1989, he once again gave excellent service both to the Association as well as to the profession. During this time he also agreed to serve on several other committees of the Federal Council, notably the Awards Committee, Editorial Committee, Education Committee and Executive Committee. He has also been an active Trustee of the Veterinary Foundation since 1974.

Prof Veary is also a man of diversity. In addition to the above, he has also participated in many other activities, notably the Whirling Wheels Paraplegic Club of which he was the President in 1972/73, the La Lucia Town Board and Licencing Board, the Management Board of Umhlanga and the Umhlanga Town Council where he was honoured by the Umhlanga Rotary Club in 1980 for services rendered to the community. "Tubby" is also a keen sportsman having

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played hockey at university and at provincial level, and being a member of the Northern Transvaal Masters hockey team in 1989.

Due to his very nature, his unselfish service, professionalism, integrity, loyalty and deep sincerity, "Tubby" has established himself as a leader in his field and, in addition, has served the SAVA and the veterinary profession with unequalled distinction as a scientist, a colleague and as a friend of the profession. In an attempt to pay tribute to a colleague and friend, the SAVA honoured him with the Boswell award for dedicated service to the Association in 1987 and elected him unanimously as Honorary Life Vice-president of the Association in 1988. It is our privilege to do so yet again, by awarding the 1990 Silver Medal to Prof C M Veary.

To the editor/Aan die redakteur

AURICULOTHERAPY IN CANINE THORACOLUMBAR DISC DISEASE

In "A clinical study of auriculotherapy in canine thoracolumbar disc disease" (JSAVA 61: 102-105), the author reports on a series of cases where pain caused by disc disease was treated by acupuncture. The role of pain in disc disease however, warrants further discussion.

The emphatic viewpoint of Prata[8] is that pain associated with thoracolumbar disc disease is consistently associated with nerve root attenuation. I support the statement in Pead's[7] letter referring to a similar article[9]: "I feel that the author has failed to stress that the treatment of pain is a secondary objective in thoracolumbar disc disease and that any type of analgesia must be coupled to procedures which are designed to limit the effect of the protrusion on the nervous system".

I would like to emphasise the fact that disc disease is a compressing lesion of the spinal cord and nerves. I therefore believe that disc disease is a surgical problem unless proven otherwise. Hoerlein states the following: "it is best to remove the lesion (which may be massive) even if pain is the only clinical sign. If the protruding mass is left in the canal, organisation of the mass into a fibrotic, calcific, or osseous lesion can cause permanent compression"[4]. This philosophy is supported by Prata[8].

The concept proposed by Brown et al.[3] that "if an animal is presented with marked paresis, or if a mildly disabled animal fails to respond to conservative treatment, surgical intervention is recommended" is no longer valid. This is because of safe and precise myelography and the minimal invasive, minimal destabilising decompressive surgery of pediculectomy (mini hemi-laminectomy)[1 2 5]. Such procedures give immediate and lasting relief from pain by addressing the primary problem by decompression.

The stated recovery rate by auriculotherapy[10] of 50% is less than the accepted recovery rate with conservative treatment[6] or the recovery rate after laminectomy and fenestration[6].

It is vital to realise that pain is just the symptom of annulus fibroses trauma and nerve compression[8]. Treatment of disc disease especially without myelographic confirmation, by means of conservative, medical or acupuncture measures may remove the restraint of discomfort and pain and thus the protection against further damage.

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NURSING CARE OF THE SMALL ANIMAL NEUROLOGICAL PATIENT

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ABSTRACT

Nursing care of long-term recumbent small animals, with emphasis on the neurological patient, is described. Principles of general nursing care, particularly nutritional support and the prevention and treatment of urinary complications, are of major concern in any weak or recumbent patient. The estimation of nutritional requirements and adjustments, information on South African commercial liquid diets and practical rehabilitation are described.

Key words: Nursing care, neurological patient, nutritional support, urinary complications, rehabilitation.

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INTRODUCTION

Small animal neurological patients are frequently seen in clinical practice. These patients may require expensive, time-consuming surgery and after-care. A well-managed rehabilitation programme can expedite recovery from a neurological insult, be it as a result of disease, trauma or surgery. Successful rehabilitation relies on team-work which involves the veterinarian, the veterinary nurse and the owner.

Most dietary information available to South African veterinarians is of little application, as local products differ in both name and composition from those available in the United States of America and the United Kingdom. This article aims to help practitioners develop an appropriate rehabilitation programme, utilising available techniques and products.

HANDLING

The simple procedures of handling, turning and carrying a neurological patient are often conducted with little thought about the possibility of exacerbating neurological deficits. Anaesthetised spinal patients are particularly at risk, as normal epaxial muscle support is absent. Moving these patients whilst keeping the neck and back as straight as possible, is imperative. Small patients may be cradled in the arms, ensuring adequate neck and thoracolumbar support, whereas larger dogs should be strapped to a rigid, flat surface[25]. For small patients, the use of a flat tea-tray may be advised, which can be slid under the recumbent patient and secured by means of elastic gauze or adhesive bandages. Larger dogs could be strapped to an ironing-board for transport to a veterinary hospital.

Patients exhibiting personality changes, should be handled with care as their behaviour is unpredictable and patient and handlers may be at risk. Cages with padded walls should be used to house patients with vestibular or cerebellar disease to reduce the likelihood of trauma. It may be necessary to tranquilise such patients with 0,5 to 1 mg kg^{-1} diazepam (Valium, Roche Products) or 1-5 mg kg^{-1} phenobarbitone (Lethyl, Lennon Limited). Both these drugs may be administered 3 times daily.

Although the treatment of brain trauma is not within the scope of this article, certain nursing procedures require attention, particularly in comatose patients. The head should be elevated and compression of the veins of the head avoided. Maintenance of a low-normal body temperature and adequate ventilation, will reduce excessive metabolic demands on cerebral tissue[21].

Decubital ulcers and hypostatic pneumonia are serious potential complications in long-term recumbent patients. Turning paretic patients every 2-4 hours, will minimise these complications[22]. Soft, supportive bedding is imperative to minimise decubital ulcer development. Either foam rubber, air or water mattresses should be used. Keeping the patient clean, dry, and free from urine and faecal contamination is important as moisture contributes greatly to the development of decubital ulcers[16]. An artificial sheepskin material (Drybed, Chattaronga, P.O. Box 191, Rivonia 2128) will help keep the patient dry.

Hypostatic pulmonary congestion predisposes a patient to pneumonia, and regular turning of the recumbent patient aids postural drainage. Pulmonary physiotherapy by means of chest percussion (coupage) and manual vibration, will augment postural drainage[3]. Signs of respiratory infection should be attended to immediately. In these cases, it is preferable to perform a trans-tracheal aspirate for cytological examination and culture, prior to instituting antibiotic therapy. A respiratory complication is debilitating and costly to treat, which would be exacerbated if the causative organism is resistant to the antibiotic. Whilst awaiting culture and antibiogram results, amoxycillin (Clamoxyl, Beecham Pharmaceuticals) and gentamicin (Genta 50, Phenix SA) antibiotic therapy may be administered. Preventing this complication is easier than attempting to cure it.

HYDRATION

Water should be offered ad lib for those able to drink. Some animals may have to be supported in a sternal position, and offered water every 2 hours. Forced oral intake of water, by means of a syringe or nasogastric/pharyngostomy tube, may be necessary to maintain normal hydration. An adequate water intake will not only prevent dehydration, but will also minimise the development of retention cystitis. The hydration status should be monitored twice daily by means of skin turgor, capillary refill time, and the appearance of mucous membranes. If normal hydration cannot be maintained by these methods, intravenous fluid administration must be instituted. Resuscitative fluids include the polyionic

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crystalline solutions Plasmalyte B (Sabax) or Balsol (Labethica). Maintenance solutions (Maintelyte, Sabax, Post Surgisol, Labethica) have high potassium concentrations (25 mmol ℓ^{-1}) and include 100 g dextrose. Paediatric fluid infusion/administration sets deliver 60 drops per ml, allowing the clinician to calculate the rate of infusion. Maintenance fluid solutions are hypertonic and require strict intravenous administration via the jugular vein. A jugular catheter (Venocath, Abbott laboratories) is ideally suited for this purpose.

NUTRITION

Patients with pharyngeal dysfunction or those too weak to eat, rapidly become protein-energy deficient. Injury, surgery and systemic disease result in neuroendocrine activation, causing an increase in the metabolic rate[6]. If this is accompanied by deficient nutrient intake, then endogenous sources of energy are utilised. Liver and muscle glycogenolysis, fat oxidation and lysis of tissue protein rapidly occurs. Intravenous glucose infusion has little sparing effect on body proteins; it further increases the metabolic rate and insulin release. The antilipolytic action of insulin also results in less utilisation of endogenous fat reserves. As a consequence, protein catabolism is increased[3 6 10].

Stress (particularly following surgery, serious infections, burns, trauma and head injuries) results in metabolic alterations in fuel utilisation. An "ebb" and "flow" phase after trauma is described, characterised by hypometabolism for 24 to 48 h, and then hypermetabolism. The latter phase peaks at 4 d and lasts 2-4 weeks[10]. A "diabetes of injury" also occurs, characterised by peripheral insulin resistance, increased hepatic gluconeogenesis and glycogenolyis. This hyperglycaemia and insulin resistance, limits the use of intravenous glucose administration as a fuel source in trauma patiens[3 10]. Significant amounts of tissue protein are metabolised during stress; the majority of amino acids are deaminated and utilised in the synthesis of glucose. In addition, amino acids are required during stress for the production of antibodies and blood cells[10].

Nutritional support therefore becomes important in order to minimise the utilisation of endogenous energy, especially that derived at the expense of tissue protein[6 10]. Any depletion of body protein, however small, is regarded as the start of a detrimental process that can have devastating effects on patients[10].

In order to calculate the approximate requirements, the Veterinary Hospital of the University of Pennsylvania Nutrition Support Service (VHUP - NSS) utilise the mean maintenance [M] of healthy adult dogs and cats as a starting point, and make adjustments for certain clinical conditions[10]. The mean maintenance [M] is used because it represents the resting energy expenditure (REE) plus increments for physical activity, assimilation, waste formation and excretion. It approximates the energy expenditure in hospitalised dogs and cats more closely than basal metabolic rate (BMR) and resting energy energy expenditure (REE)[10].

Table 1: **Maintenance energy requirements* (M) for dogs[10]**

Body mass (kg)	Kcal ME d^{-1}	Body mass (kg)	Kcal d^{-1}
1	132	26	1 520
2	222	27	1 563
3	300	28	1 607
4	373	29	1 650
5	440	30	1 692
6	506	31	1 734
7	568	32	1 776
8	628	33	1 817
9	686	34	1 859
10	742	35	1 899
11	797	36	1 940
12	851	37	1 980
13	903	38	2 020
14	955	39	2 060
15	1 006	40	2 100
16	1 056	41	2 140
17	1 105	42	2 177
18	1 153	43	2 216
19	1 200	44	2 255
20	1 248	45	2 293
21	1 295	46	2 332
22	1 340	47	2 369
23	1 368	48	2 409
24	1 430	49	2 445
25	1 475	50	2 482

*M = 132 $BW^{0,75}$ kcal ME per day[10]
BW = Body mass in kg

Table 2: **Maintenance energy requirements* (M) for cats[10]**

Body mass (kg)	kcal ME d^{-1}
1,0	60
1,5	90
2,0	120
2,5	150
3,0	180
3,5	210
4,0	240
4,5	270
5,0	300
5,5	330
6,0	360

*M = 60 W kcal ME per day[10]
W = Body mass in kg

Tables 1 and 2 list the maintenance energy requirements, in metabolisable energy (ME) per day for a specified body weight. Adjustments for the particular clinical condition are then made, expressed as a multiple of M (Table 3). In this estimation of the mean maintenance requirement, additional adjustments are made for hyperactivity, steroid therapy (upward), or downward with inactivity due to confinement, multiple trauma, paralysis, coma[10].

After calculating the energy requirements, the diet and route of administration must be selected. Enteral nutrition is preferred to parenteral nutrition. Total parenteral nutrition is more expensive, more dangerous and requires greater attention to formulation, preparation, sterility of solutions and administration systems, and patient monitoring[10]. Total parenteral nutrition is beyond the scope of this article and the reader is referred to recent reviews[1 19]. Dietary options are those of canned pet foods, commercial liquid diets, and home-made gruels. The method of administration is determined by the type of diet selected.

Initially the patient should be encouraged to voluntarily consume a good quality low bulk meal 4 to 6 times daily[22]. It may be necessary to prop the animal in a sternal position with sand bags. Increasing the

Table 3: **Individual adjustments as multiples of M***

Condition	Adjustment x M
Physical inactivity	0,7 - 0,9
Hypometabolism	0,5 - 0,9
Elective surgery	1,0 - 1,2
Trauma : mild	1,0 - 1,2
moderate	1,1 - 1,5
severe	1,1 - 2,0
Sepsis	1,2 - 1,5
Head injuries : mild	1,0 - 1,2
severe	1,2 - 2,0

*Modified from Donoghue[10]

palatability of food by enhancing the flavour and aroma, may encourage voluntary eating. Additives such as peanut butter, garlic salt, canned tuna, strained baby food, and chicken or beef stock, are suggested[9]. High protein supplements may be necessary to reduce the bulk of canned food required to maintain the energy intake. Examples of such supplements are listed in Table 4. Diazepam (Valium, Roche Products) administered at 0,05 mg kg^{-1} intravenously has an immediate appetite-stimulating effect, particularly in the cat[9]. However, patients are often left sedated and depressed, and the appetite-stimulating effect decreases with subsequent doses. Forced oral feeding with canned food boluses is usually too stressful, and requires large quantities to meet energy requirements due to the low caloric density and high water content. Short-term (1-2 d) support may be achieved with this method, but it is usually not well-tolerated.

Semi-solid, sticky materials such as peanut butter and commercial nutrient gel (Nutrijel, Centaur Labs) may also be used. Peanut butter (Yum-Yum, Epic Oil Mills) contains approximately 95 kcal per table spoon (JB Bloch 1989 Epic Oil Mills, Johannesburg, personal communication). Nutrijel contains approximately 2,8 kcal g^{-1} (2,8 kcal per 25 mm) (A. Edgson 1988 Centaur Labs, Johannesburg, personal communication). These are best used as supplements to other nutritional sources, as only a limited quantity can be given at one time.

Syringe-feeding may be effective in some patients. However, the stress of handling and the risk of aspiration, make this an undesirable method of forced-feeding[10]. When syringe-feeding is indicated, commercial liquid diets should be used, which may be fortified with additional blended, high quality protein (cottage cheese, eggs).

Tube-feeding is necessary in patients with pharyngeal paralysis, severe depression, or those unco-operative with forced-oral-feeding. Repeated oral intubations are very stressful and are to be avoided.

Table 4: **High protein supplements**[a]

	Energy (kcal/100 g)	Protein (g/100 g)	Fat (g/100 g)
Egg[b] (whole cooked)	163	13	12
Minced beef (regular)	270	18	22
Cheddar cheese	400	25	32
Cottage cheese	100	14	4

[a]Modified from Lewis et al[18]
[b]1 large = 50 g

As anaesthesia is required for pharyngostomy tube placement, a nasogastric tube may be used in patients which are regarded anaesthetic risks[6,7]. Small bore (5 French) nasogastric tubes (Feeding Tubes, Sabax; Safeed, Terumo Corporation) are used in cats, whereas slightly larger bore (8 French) are usually well-tolerated by dogs. Due to the small diameter of the nasogastric tube, only liquids and commercial liquid diets may be administered. Use of a pharyngostomy tube (Leven's Type Stomach Tube or Ryles Type Duodenal Tube, Sabax) allows the use of gruels, made from liquidised canned food and water. Gruels usually require supplementation with a source of fat (eg. maize oil) and protein of high biological value (eg. eggs, cottage cheese)[8]. The techniques of nasogastric and pharyngostomy tube passage are well described elsewhere[6,7,9].

It is preferable to tube-feed a diet low in lactose, as lactose often leads to diarrhoea[8]. For this reason, most infant milk formulas should be avoided. Various commercial liquid diets, which may be used for tube-feeding, are listed in Table 5. A period of adaptation is required when feeding commercial liquid diets. On the first day, 50% of the calculated daily requirement should be fed, increasing to 75% on Day 2 and 100% on Day 3. Gastric reflux may occur in recumbent patients fed by means of nasogastric or pharyngostomy tubes. To avoid reflux oesophagitis, metoclopramide (Maxolon, Beecham Research Laboratories) should be added to an intravenous infusion at 1 to 2 mg kg^{-1} 24 h^{-1}, and cimetidine (Tagamet, Smith Kline & French) administrated intravenously 3 times daily at 5 mg kg^{-1} (M Rosenzweig 1990 Department of Clinical Studies, University of Pennsylvania, personal communication).

Homemade blended diets may also be used for pharyngostomy tube-feeding. A blended mixture of one 125 ml jar of strained meat and vegetable baby food (Purity, Coleman Foods), two cooked eggs, one tablespoon of maize oil, and 100 ml of water provides a liquid diet of approximately 1 kcal ml^{-1}.

URINATION

Recumbent patients with intact voluntary urinary control, should be taken onto a grassed area to urinate, at least 3-4 times daily. Patients which have lost voluntary control over urination, require gentle manual bladder expression every 4-6 hours.

Spastic neuropathic bladder

A spinal cord lesion cranial to the sacral spinal cord segments (S1-S3), results in the external urethral sphincter and perineal muscles becoming spastic, with resultant increased resistance to urine flow[20]. Voluntary control and the perception of a full bladder is lost, and the residual volume increases. In these cases, manual expression of urine is usually not possible, and the animal must be catheterised whilst instituting pharmacological therapy.

This functional urethral obstruction (somatic dyssynergia), should be treated with muscle relaxants until the bladder spinal reflexes take control, and the bladder becomes "automatic". When the threshold capacity is reached, non-voluntary (reflex) micturition will then occur[20]. Two muscle relaxants are recommended for use until reflex micturition occurs. Diazepam (Valium, Roche Products) may be administered at 2 to 10 mg 3 times daily[24]. The main side effect is sedation. A more specific (peripheral) skeletal muscle relaxant, is dantrolene (Dantrium, Smith Kline & French), recommended at a dosage of 3 to 15 mg kg^{-1} divided into 2 daily doses[24]. The dosage should be titrated and the lowest optimal dose administered, as generalised muscle weakness is a potential side effect.

Flaccid neuropathic bladder

A distended, areflexic neuropathic bladder is encountered in patients with lower motor neuron lesions interrupting the sacral reflex arc[20]. This is most often encountered in cauda equina syndrome,

Table 5: **Commercial liquid formulas**

Product (Manufacturer)	Energy (kcal ℓ^{-1})	Protein (g ℓ^{-1}) and sources	Fat (g ℓ^{-1}) and sources	Carbohydrate (g ℓ^{-1}) and sources	Lactose (g ℓ^{-1})	Osmolality (m0sm kg^{-1} H_20)
*Ensure (Abbott Lab)	960	33,6 Na and Ca caseinates, Soy protein isolate	33,6 Maize oil	130,8 Hydrolised maize starch, Sucrose	0	470
+Ensure Plus (Abbott Lab)	1 440	50,4 Na and Ca caseinates, Soy protein isolate	50,4 Maize oil	196,2 Hydrolised maize starch, Sucrose	0	690
**Ensure Plus HN (Abbott Lab)	1 500	65,4 Na and Ca caseinate, Soy protein isolate, Whey protein (Pro-Mod, Abbott Lab)	50,4 Maize oil	196,2 Hydrolised maize starch, Sucrose	0	720
Vital High Nitrogen (Abbott Lab)	1 000	41,7 Partially hydrolised whey, Caseinate and Soy protein	10,8 Safflower oil, Medium chain triglycerides	184,67 Glucose polymers	0	500
Osmolite (Abbott Lab)	1 060	37,3 Na and Ca caseinates, Soy protein isolate, Maize oil	38,6 Medium chain triglycerides,	145,3 Glucose polymers	0	300
Nutramigen (Bristol/ Mead Johnson)	670	19 Hydrolised casein, Maize syrup solids	26 Maize oil	91 Hydrolised maize starch	0	480
Portagen (Bristol/ Mead Johnson)	670	22,4 Na caseinate, Maize syrup solids	30,5 Maize oil, Medium chain triglycerides	73,6 Hydrolised maize starch, Sucrose	0,2	220
Replace (Sabax)	1 000	37,4 Na and Ca caseinate, Soy protein	33,6 Maize oil	137,6 Hydrolised maize starch	0	465

*212 g Ensure per litre water
+318 g Ensure per litre water
**318 g Ensure + 19,8g Pro Mod per litre water

where the sacral spinal cord segments (S1-S3) or the cauda equina, are damaged. The clinical signs of cauda equina syndrome include lumbosacral pain, urinary and/or faecal incontinence, and flaccid paralysis of the tail. Paresis in one or both pelvic limbs may or may not be present. Paresthesia (defined as abnormal spontaneous sensation and usually manifest as a self-inflicted dermatosis) is also described[17].

Interruption of the reflex arc, prevents contraction of the bladder and reflex relaxation of the urethra, resulting in an excessively distended bladder. Disrupted tight junctions of the bladder (due to excessive distention) contributes to a weakened detrusor muscle[20]. Regular manual expression of the bladder is therefore extremely important. Bethanecol (Urecholine, Merck Sharpe and Domme) is the drug of choice for the treatment of detrusor atony[20]. It has been used successfully to stimulate detrusor contraction in neurogenic and non-neurogenic bladder atony[24]. Recommended oral dosages begin at 5 mg 3 times daily in the dog, and 1,25 mg 3 times daily in the cat with titration until adequate bladder contraction is attained[20].

Successful therapy depends upon the type of lesion and the extent of detrusor decompensation. Bethanecol is not specific for bladder smooth muscle, and potential side effects are lachrymation, diarrhoea and abdominal discomfort, for which atropine is antidotal[24]. The use of bethanecol may cause increased urethral resistance, due to parasympathetic smooth muscle stimulation. Bladder emptying then becomes dependent upon the detrusor exceeding the effect of the urethra. If this outlet resistance (or dyssynergia) is suspected, phenoxybenzamine (Dibenyline, Smith Kline & French) should be used concurrently with bethanecol. This combination allows for increased bladder contraction (effect of bethanecol) with lowered urethral resistance (phenoxybenzamine effect)[24].

Reflex dyssynergia

A partial spinal lesion may result in reflex dyssynergia. This describes a lack of synchronism between bladder contraction and urethral relaxation, such that detrusor contraction occurs without decreased urethral outflow resistance[20]. Clinically, the animal initiates micturition followed by sudden interruption of the urine flow, whilst straining continues. Phenoxybenzamine (Dibenyline, Smith Kline & French), an alpha-adrenergic blocking agent, will decrease urethral pressure, whilst having no effect on intravesical pressure. The recommended oral starting dose is 5 mg, titrating to a maximum of 30 mg once daily in dogs, and 2,5 mg, titrating to a maximum of 10 mg once daily in cats[20]. Response to therapy is dependent upon the type and extent of the lesion. Several days may be required before therapeutic blood levels are attained and improvement occurs. Phenoxybenzamine when combined with bethanecol, is useful in the therapy of

decompensated (myogenic) or atonic bladders[20]. Until voluntary micturition returns, it is important to assist with emptying of the bladder.

If catheterisation is unavoidable, then an aseptic technique is of vital importance, as there is a very high risk of iatrogenic urinary tract infection. Intermittent catheterisation is preferred over the use of long-term indwelling catheters[5 23]. If circumstances necessitate the use of an indwelling catheter, then a closed system of urine collection is imperative. Connection of a urine drainage bag (Cystoflow, Sabax) to a Foley catheter in a bitch will ensure the system is closed. A male dog urinary catheter may be connected to a sterile fluid administration set and empty intravenous fluid bottle, to create a closed system. Paediatric nasogastric tubes (Safeed, Terumo Corporation) may be used as urinary catheters in male dogs. They are softer, cheaper, and although a little more difficult to insert, appear to be better tolerated by patients requiring indwelling urinary catheters. Periodic urine examinations are necessary in all recumbent patients, but especially those that are catheterised, to timeously identify urinary tract infections.

To prevent urine scald, the bedding must be changed frequently[22]. A special fabric, such as an artificial sheepskin (Drybed), is recommended as bedding, to help keep the patient dry. Patients that are contaminated with urine and faeces should be bathed and thoroughly dried. The hair around the perineum may be clipped so that a barrier cream (Fissan paste, Group Laboratories) may be applied to prevent urine scald. Talcum powder applied to the inguinal area, and the placement of absorbent padding between the legs, will also decrease skin irritation[22].

DEFECATION

Patients with faecal incontinence, can be stimulated to defecate either with a rectal thermometer or a gloved finger. If this is unsuccessful, then a warm soapy enema should be administered every 2-3 d[14].

Although convenient, sodium phosphate enemas (Fleet Enema, Lennon Laboratories) should not be used. Hyperphosphataemia, hypernatraemia and hypocalcaemia are potential serious side effects[2]. This is particularly important in chronically constipated and recumbent patients. Firstly the colonic mucosa is disrupted and more permiable, and secondly recumbent patients are more likely to have longer retention of the enema solution due to immobility and an atonic colon. Glycerol enemas (Babylax, Restan Laboratories) and sodium citrate enemas (Microlax, Pharmacia) may be used in these patients. It is preferable to use either luke-warm tap water or a normal saline enema first, as these are non-irritative. If unsuccessful, a mild soapy enema should be administered. A diluted solution (1:5 parts water) dioctyl sodium succinate (Surfactol, Centaur Labs) may also be used. Small volumes administered frequently, prove to be more successful than a large volume single enema where most of the fluid is expelled with little faecal material.

The use of hexachlorophene soap in an enema is contraindicated, especially in cats. Clinical and experimental neurotoxicosis has been reported with hexachlorophene enema administration[4 27].

REHABILITATION

Immobile, recumbent patients become bored and depressed if they are not stimulated. These patients should be positioned so that they can watch activity within the hospital. Petting, grooming and talking to the recumbent patient provides stimulation, and where possible, the owners should be encouraged to visit. Physical therapy also results in psychological stimulation.

PHYSICAL THERAPY

Physical therapy in the rehabilitation programme utilises various techniques to improve circulation and healing, and help restore normal function. Physical therapy results in an improved mental state, increases physical strength and, by accelerating the return of normal function, reduces the period of hospitalisation[11].

Table 6: **Aims of physical therapy**

1. Increase circulation
2. Maintain joint mobility
3. Maintain muscle mobility
4. Stimulate vital functions
5. Psychological stimulation

The aims of physical therapy are listed in Table 6. The maintenance of joint and muscle mobility will prevent contractures of joints and muscles, and stimulate circulation. During physical therapy, other vital functions such as cardio-respiratory, excretion and psychological stimulation take place[12]. In order to achieve these aims, the following modalities may be used:

* Massage
* Exercise therapy
* Hydrotherapy
* Electrotherapy

Those procedures which are easy to perform in private practice, will be emphasised.

Massage

Massage is the simplest form of physical therapy and involves finger and hand manipulation of soft tissues. Massage improves circulation and stimulates venous and lymph drainage, thereby removing metabolites and increasing nutritive elements[26]. The mobilisation and loosening of tissues, decreases the likelihood of adhesions, stretches tendons and relieves spasm and pain[11 13 26].

The massage techniques used on the neurological patient are effleurage (stroking) and petrissage (kneading and compression)[13].

Effleurage: This is a gentle, stroking technique directed from the distal section of the area being treated to the proximal section. The direction aids venous and lymph return. For soothing, sedative effects the stroking is done slowly and rhythmically. In order to increase circulatory effects, the strokes are firmer, faster and rhythmical. This technique is usually the first to be used when performing massage, as it accustoms the patient to the therapist's touch, and is reassuring and soothing.

Petrissage: The manipulation of kneading and compression is primarily used on muscle groups or individual muscles, to enhance circulation and stretch muscles. The technique is applied by moulding the palm of the hand and fingers as a unit to the area. Compression is then applied between fingers and palm, as the hand moves proximally in the direction of venous return. The technique should be gentle but firm, and never vigorous enough to cause protective muscle contraction[11].

The duration of a treatment period will vary according to the number of joints and muscles to be masssaged, as well as the size and condition of the patient.

Exercise therapy

Exercise therapy is another form of simple physical therapy (involving both passive and active exercise) to improve blood and lymph flow, sensory awareness and voluntary movement[12]. Passive and active exercise leads to improved conduction of nerve impulses, and initiates proprioceptive reflexes which induce muscle contraction and improved muscle strength[26].

Passive exercise is any exercise that does not require patient participation. This form of therapy consists of passive movement of the paretic or paralysed limb by the therapist. By stretching muscles and tendons, disuse atrophy is also minimised[12].

To ensure maintained mobility, the joints must be flexed and extended to the limit of their range of motion, unless the movement is painful. If pain is expe-

rienced, the procedure must still be performed but must then be limited to that which is tolerable to the patient[26]. Muscles which pass over more than one joint, must be stretched over both joints simultaneously. To effectively stretch the biceps femoris, semimembranosis and semitendinosis muscles, hip flexion and stifle extension must be performed simultaneously. In addition, when conducting passive exercise, all the physiological movements must be performed; for example the hip joint: flexion, extension, abduction, adduction, internal and external rotation[12].

Active exercise entails patient participation. This form of exercise in the neurological patient, usually necessitates support of part of the body. This is achieved either by means of a sling, or by the patient being held by the therapist. The "towelling" method, whereby a paraplegic/paraparetic patient is supported by means of a towel slung around the abdomen, provides support and allows voluntary movement of the limbs. A similar effect can be achieved by the "tailing" method, care being taken to grasp the tail at the base to prevent injury.

Active exercise should be tailored to the physical capacity of the patient, and care be taken to avoid abnormal stress to the spine, particularly after prolonged immobility and spinal surgery. The length of treatment should be from 20 to 30 min, depending on the patient. Endurance increases with repitition.

Hydrotherapy

Exercise in water (swimming) is an additional form of physical therapy requiring patient participation. Being placed in water is a greater stimulus for voluntary muscle movement than being supported on grass. The buoyancy creates a lifting effect and free voluntary movement becomes possible in an animal which, otherwise is unable to stand[11 26].

In addition, the warm water stimulates the circulation, cleanses the skin of urine and faeces, and soothes muscle spasms. Sutured surgical wounds have to have sealed before hydrotherapy is initiated, and draining wounds should not be immersed in water. Febrile patients, or those with cardiac or respiratory disorders, should not be placed in water[11].

The optimum water temperature should be 38-40°C, and the room comfortably warm and draught free[11]. The length of the treatment period depends on the reaction of the patient to hydrotherapy. Commence with a 5 min session, extending each subsequent session by 5 min, to a maximum period of 20 min. If the patient shows undue fatigue at any stage of the hydrotherapy, treatment should be stopped for that day. Never leave a patient unattended in the water. Ensure the patient is thoroughly dry after the hydrotherapy, making use of a hair-dryer if necessary. A recumbent, damp patient is predisposed to the development of decubital ulcers.

At the end of the session, position the paretic or paralysed limbs in the mid-position, on the appropriate surface (sheepskin, dry bed).

Electrotherapy[15]

The only mode for electrical stimulation with denervation, is interrupted direct current ("pulsed galvanic" current). Stimulation with this type of current will facilitate the relaxation of muscle spasm, and stimulate circulation by the "pumping action" of the musculature. It is the preferred electrotherapeutic modality for neuromuscular stimulation in cases of reaction of degeneration, (i.e. the reaction to electrical stimulation of muscles, the nerves of which have degenerated). High-voltage, direct current should not be used, as the pulses are too short in duration to be effective. Low-voltage, direct current (galvanic) is the only mode for stimulation in denervated musculature. The therapeutic effects of faradic current ("Faradism") are lost in denervated musculature and with reaction of degeneration due to nerve damage; it is usually an irritating stimulant to the patient. This is important in polyradiculoneuritis, where hyperasthesia to sensory stimuli is often present.

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