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ALL CORRESPONDENCE: Manager, SAVA, Jl. S Afr. Vet. Ass., P.O. Box 25033, Monument Park, 0105 Pretoria. (Tel. (012) 346-1150)

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EDITORIAL **REDAKSIONEEL**

THE PHILOSOPHY OF CONTINUING EDUCATION

The perceived need for continuing education within the veterinary profession may be ascribed to a number of factors. Not least of these is the growing realisation that the almost logarithmic knowledge explosion over the last several decades has rendered the basic veterinary degree inadequate to equip either the veterinary scientist, or the practitioner, for a lifetime career. For some, continuing education stems from the desire to specialise in a particular field or discipline, whilst for others it signifies a need to reinforce the scientific and technological basis of veterinary practise. To this end, veterinary educators have endeavoured to impress upon the undergraduate the concept of embarking on a lifetime of learning[3].

In its broadest sense continuing education implies the concerted attempt to make use of all available means to acquire new knowledge and master relevant technology. As there is no monopoly on knowledge, it merely remains for the individual to make use of the avenues such as publications, videocassettes, videodiscs, tapes, libraries, non-formal and formal offerings to meet the challenge: the lifelong learning referred to above. The advent of relatively inexpensive computerisation has added a new dimension to both professional competence and continuing education. Whilst training in the use of computerised data is rapidly being included in the curricula of undergraduates, earlier generations of veterinarians need to master the technology in their own time. In this regard it has been said that as scientific knowledge doubles every six years, factual recall may be contrued as malpractice[2]. Properly computerised data can ensure rapid access to current information without the patient, or the client having to be solely at the mercy of the individual's memory.

Generally, self-study in the form of continuing education, has been largely disadvantaged by the very nature of the undergraduate tuition offered by universities where emphasis has been placed on training rather than on education. The former concentrates on problems with known solutions, whilst the latter is concerned with problems of unknown solution. It is estimated that over 90% of instruction, is at the lower cognitive level in some disciplines[1], that is training rather than educating. The expected change to greater self-reliance in formal education should facilitate non-formal continuing education by future graduates.

The commitment to a lifetime of learning stems from the service nature of the veterinary profession and the special responsibility which the profession has in society. This responsibility is embodied in the Veterinary and Paraveterinary Professions Act No 19, of 1982[4]. Parliament has in its wisdom, seen the profession as consisting of educated, knowledgeable persons, adequately qualified to uphold the interests of society in the field of veterinary matters. This trust which has been placed in the profession, should be a source of pride and foster the motivation of each and every member to update his knowledge and insight by way of continuing education — veterinary and otherwise.

Apart from the individual's responsibility, a responsibility to promote and encourage continuing education also falls to the veterinary institutions such as the veterinary faculties, the South African Veterinary Council, research and service establishments, the South African Veterinary Association and its subgroups; in fact wherever veterinarians are active in society.

Most, if not all of these organisations, have to a greater or lesser extent addressed their respective responsibilities regarding continuing education. Thus both veterinary faculties are involved in the provision of refresher and short courses, and contribute indirectly to the success of courses offered by the SAVA by encouraging active participation by staff. The funding of universities is, however, such that more direct involvement must be financed outside of the current state subsidy system. According to the report on the subsidisation of universities (SAPSE-110)[5], "the diversity of activities that may be included under the heading of community instruction, render it impossible to formalize public support via a formula in an uncontentious manner, especially in cases where the courses offered appear to be on the fringe of a university's normal activities. The benefits ... also tend to accrue to persons in their private capacities on account of the type of course generally provided, rendering subsidization unnecessary". Universities have attempted to have continuing education excluded from this restriction, but without success thus far. Given the above restriction, it is obvious that the faculties are largely unable to expand their continuing educational offerings unless private funding can be secured, or by charging the participants the full cost thereof. The formal post-graduate degree programmes are obviously subsidised under the formula and offer the most effective avenue for furthering veterinary education.

The South African Veterinary Council has a juristic responsibility to ensure veterinary educational standards for registration purposes and encourages continuing education for the good of the profession. The latter consideration assumes particular significance where veterinarians seek reregistration after a period of absence from the profession. In this regard the question of compulsory continuing education "credits" has been mooted, but would appear to be impractical at present. In particular, the relative weighting of various courses, self-study, attendance versus examination and similar considerations, present difficulties. Nevertheless, one may assume increasing pressure within the profession for some form of certification in time to come.

The South African Veterinary Association, being a voluntary association of veterinarians, has a responsibility to cater for the needs of its members. The Association has to date played the major role in providing non-formal continuing educational courses and

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programmes for its members and may be expected to expand this activity commensurate with the demand. The speciality groups have been particularly active in this regard and have done much to broaden the educational input by bringing prominent overseas tutors to this country.

The appearance of a number of veterinary textbooks by local experts has added to the pool of pertinent local scientific information available to veterinarians for self-study purposes. This new phase of veterinary scientific development is most encouraging.

The recognition of the South African Veterinary Foundation as an educational organisation offering tax incentives to potential donors is also a significant consideration in that it could play a pivotal role in continuing veterinary education funding in future.

The lead taken by the SAVA in promoting veterinary education, is in keeping with the philosophy of the association whereby resources are pooled to better serve the interests of the members. It would therefor seem that the Association is best placed to co-ordinate and expand continuing education for its members in the first instance and for the profession as a whole.

Ideally, the Association should appoint a full-time continuing education officer whose function would be to identify topics and suitable presenters; co-ordinate input and organise meaningful courses and material in an ongoing and enthusiastic manner. At present, however, the resources of both the Association and the Foundation are inadequate to finance such an appointment. The Foundation, notwithstanding supreme efforts by individual members, has barely kept pace with inflation and the financial state of the Association has recently been the subject of intense debate. What is required, is a commitment by all members of the Association, to raise monies for continuing education under the auspices of the Foundation, which is now legally registered for this purpose. These funds should be used to purchase expertise whereby the abovementioned ideal may be realised. In time, the courses and other educational material should generate sufficient funds for the programme to become self-supporting. The magnitude of this task should not be underestimated as it will include offices, a secretariat, reprographic facilities and payment for lecturers. The latter have for too long offered their professional expertise free of charge in this regard.

Continuing education is the responsibility of the individual in the first instance, but the Association can facilitate the presentation of learning material, given the dedicated support of all its members. It remains to be seen whether the concept of continuing education is merely rhetoric or a well established need within the profession.

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N C Owen, Dean, Faculty of Veterinary Science, Medical University of Southern Africa, 0204 Medunsa, Republic of South Africa

ARTICLE ARTIKEL

ACUTE *LANTANA CAMARA* TOXICITY IN CATTLE

N. FOURIE*, J.J. VAN DER LUGT**, S.J. NEWSHOLME** and P.W. NEL*

ABSTRACT: Fourie N.; Van der Lugt J.J.; Newsholme, S.J.; Nel P.W. **Acute *Lantana camara* toxicity in cattle.** *Journal of the South African Veterinary Association* (1987) 58 No. 4,173-178 (En) Section of Toxicology, Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa.

An outbreak of acute *Lantana camara* poisoning in cattle is described in which 10 out of 91 animals died. The affected cattle became icteric and voided soft, black faeces. Necropsies were performed on three steers, and the macro- and microscopical changes in their livers and kidneys were compatible with those of *L. camara* poisoning. Changes were similar in two steers that developed typical signs after being dosed with fresh *L. camara* collected in the toxic camp. Clinical pathological changes in experimental animals included elevated serum urea and creatinine concentrations.

Key words: *Lantana camara* toxicity, cattle, hepatosis, nephrosis, gastrointestinal disturbance.

INTRODUCTION

Lantana camara is an exotic shrub which has become a widespread weed, particularly in the moist, eastern parts of South Africa[19]. Ingestion of the plant by ruminants can cause a disease which is usually clinically characterized by icterus, photosensitivity and constipation[11-14, 17] and by distinctive hepatic and renal pathology[12-14]. In some field outbreaks of acute *L. camara* poisoning in cattle, however, severe gastrointestinal disturbance and intestinal haemorrhage have been reported[11], and severe dysentery can frequently occur (AA Seawright, University of California, Davis, personal communication).

The toxic principle, Lantadene A, which is a pentacyclic triterpene acid, was isolated from *L. camara* in South Africa by Louw[3,4] and has subsequently been confirmed as the most significant toxin in the plant[15].

Although sporadic field outbreaks of *L. camara* poisoning occur in South Africa, the clinical and pathological features of these outbreaks have not been described. Herein we report clinical and pathological findings in an outbreak of acute disease in cattle which was attributed to the ingestion of *L. camara*, and the results of a dosing trial with collected plants.

HISTORY OF OUTBREAK

During July 1984, 10 out of a herd of 91 crossbred-Brahman cattle died near Pretoria about five months after they were introduced from another area. Although the veld was very dry, there was still a substantial amount of dry forage *(Themeda trianda)* available to graze. A thick stand of *L. camara* grew on the banks of a rivulet, amongst other vegetation. It was evident that the *L. camara* plants had been extensively eaten.

The cattle had grazed in the camp for two weeks when the farmer noticed that some had developed a blackish diarrhoea. They were immediately withdrawn from the camp but deaths nevertheless occurred a few days later. The farmer suspected anaplasmosis, and treated the sick animals parenterally with oxytetracycline (Terramycin L.A., Pfizer), to which they failed to respond.

The sick animals showed the following clinical signs: anorexia, severe depression, ruminal stasis, black soft faeces and yellow discolouration of the mucous membranes. No clinical signs of photosensitivity were observed when the senior author visited the farm during the outbreak. Liver-supportive therapy was of no avail, and eventually 10 animals died over a period of six days.

MATERIALS AND METHODS

Field outbreak

Necropsies were performed on three steers which had recently died (Cases 1-3). Tissue specimens of liver, kidney and gastrointestinal tract were collected and fixed in 10% buffered formalin for light microscopy. Selected tissue blocks were embedded in paraffin wax and sections were cut and stained with haematoxylin and eosin (HE) according to routine procedures. Duplicate sections were stained by a modified Masson's trichrome method[5].

Dosing trial

Fresh sprouting plant material was milled and dosed per stomach tube to a 205 kg Afrikaner steer (Case 4; Table 1) and a 207 kg crossbred-Jersey steer (Case 5; Table 2). Both animals were kept in the sun and examined daily. The following routine chemical pathological determinations were done on the sera (S) of both steers: aspartate transaminase (AST), gamma glutamyltransferase (GGT), urea (SU), creatinine, total bilirubin and phylloerythrin[10]. In addition, blood haemoglobin (B-Hb), haematocrit (B-Ht) and total plasma proteins (TPP) were determined in Case 5. Enzyme activity was measured at 25°C using the colorimetric method (Monotest, AST-optimised, GGT-New, Boehringer Mannheim). Serum urea was determined using the Berthelot method[7], creatinine by a colorimetric method (Boehringer Mannheim) and bilirubin by the azobilirubin method[18]. Blood haemoglobin was determined by the Hemiglobin cyanide method[7] and TPP by

* Section of Toxicology, Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa.
** Section of Pathology, Veterinary Research Institute. The current address of Dr Newsholme is: Department of Veterinary Pathology, University of Liverpool, P.O. Box 147, Liverpool, L69 3BX, United Kingdom.

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Table 1: Clinical observations and clinical pathology of Case 4 dosed with fresh *L. camara*

Day	Dose g/kg	Clinical observations	Clinical pathology S-bilirubin (total) µmol/ℓ	Clinical pathology S-phylloerythrin µg/100 mℓ
0	10	none	–	–
1	5	weakness, anorexia, mild icterus, decreased ruminal movements, diarrhoea	–	37,9
2	0	weakness, anorexia, moderate icterus, ruminal stasis, constipation	–	–
3	7,5	weakness, moderate icterus, mild photosensitivity, dehydration, ruminal stasis, dry and hard faeces	54,7	39,1
4	0	severe depression, mild photosensitivity, dehydration, ruminal stasis, dry and hard faeces	73,5	32,4

– Not determined

Table 2: Clinical observations and clinical pathology of Case 5 dosed with fresh *L. camara*

Day	Dose g/kg	Clinical observations	Clinical pathology B-Hb g/ℓ	B-Ht ℓ/ℓ	TPP g/ℓ	S-bilirubin (total) µmol/ℓ	S-phylloerythrin µg/100 mℓ
–3	0	none	108	0,31	73,7	4,2	1,48
0	15	none	109	0,34	78,7	4,8	1,44
1	0	depression, decreased ruminal movements	115	0,35	82,8	6,5	20,9
2	10	Anorexia, listless, decreased ruminal movements, dry and hard faeces	134	0,40	90,3	22,6	34,6
3	10	listless, mild icterus, decreased ruminal movements, dry and hard faeces	111	0,33	104,5	42,7	31,1
4	0	severe depression, mild photosensitivity, marked icterus, dehydration, black and soft faeces, ruminal stasis	94	0,28	89,2	56,4	19,6

Section of Toxicology, VRI, Onderstepoort normal values:
B-Hb : 80 - 140 g/ℓ
B-Ht : 0,26 - 0,42 ℓ/ℓ
TPP : 60 - 90 g/ℓ
S - bilirubin (total) : <8,6 µmol/ℓ
S - phylloerythrin : c. 2µg/100 mℓ

the Biuret method[7]. Microhaematocrit capillary tubes were used for the B-Ht determination (Damon IEC MB Micro Hematocrit).

During necropsy, various tissues were collected for examination by light microscopy as described.

RESULTS

Field cases

Macroscopical pathology: At necropsy, marked icterus, moderate hydrothorax, mild dehydration and ascites were evident in all three steers. The livers were swollen, friable and yellowish-brown to orange with a distinct lobular pattern. The gall bladders were oedematous and contained pale green bile. There was marked, bilateral perirenal oedema. Renal cortices were petechiated and bulged at the cut surface. The abomasal and intestinal mucosa was moderately congested and oedematous. The contents of the abomasum, small and large intestines were dark brown to black throughout. In the abomasum and small intestine the contents were watery and in the large intestine, including the rectum, they were soft and pasty. No ulcers or erosions were seen in the gastrointestinal tracts. In Cases 1 and 2, there was extensive subcutaneous oedema of the ventral abdomen. Scab formation was extensive on the muzzle of Case 2.

Microscopical pathology: Liver: In cases 1 and 2, the hepatocytes were diffusely and mildly enlarged. The cytoplasm was finely vacuolated and 1-3 large intracytoplasmic vacuoles, frequently containing cytoplasmic fragments, were present in several hepatocytes (Fig. 4). Bile canaliculi and cell borders were distinctly delineated. Many of the hepatocytes were binucleated. Nuclei, with prominent nucleoli, varied in size and were hypochromatic with marginated chromatin. A few randomly scattered hepatocytes were necrotic (Fig. 4). The hepatic cell plates were often disrupted and the spaces of Disse were moderately dilated. There was mild bile ductular hyperplasia and

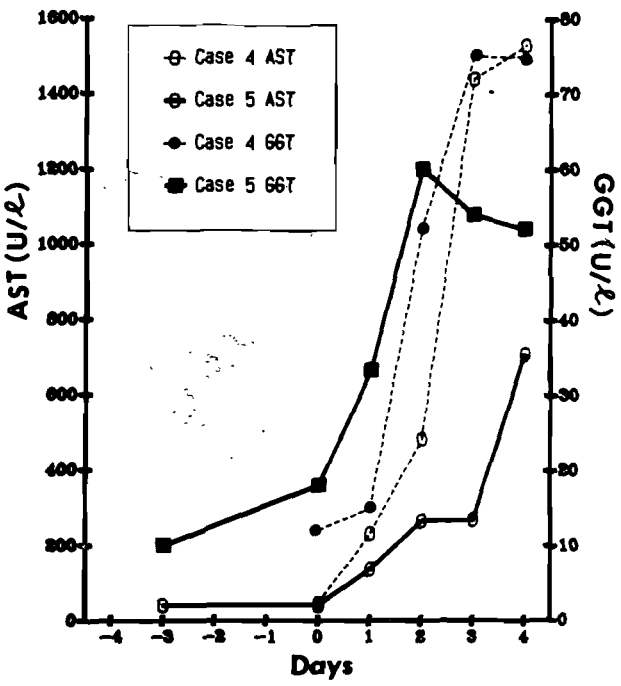

Fig. 1: Plasma activities of AST and GGT of Cases 4 and 5 (normal activities: AST < 80 U/ℓ; GGT < 25 U/ℓ)

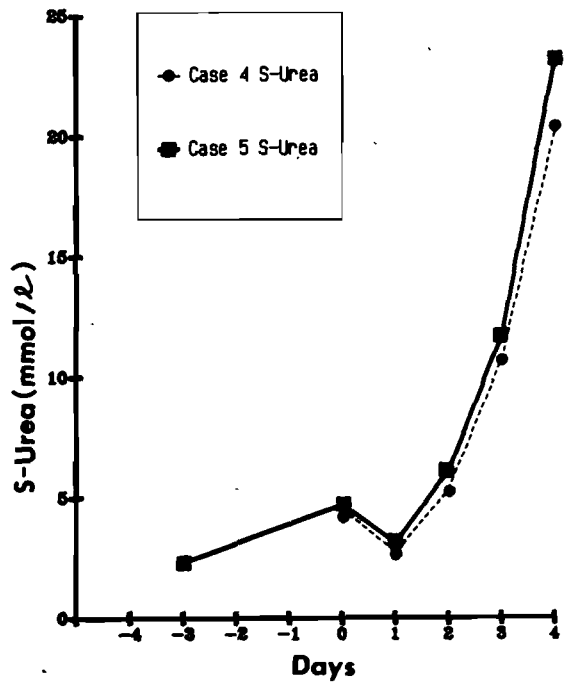

Fig. 2: Concentrations of S-urea of Cases 4 and 5 (normal value: 1-4,5 mmol/ℓ)

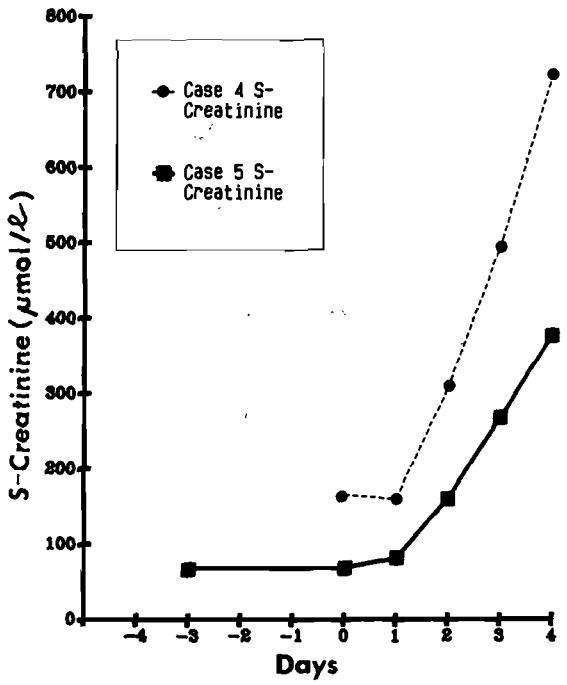

Fig. 3: Concentrations of S-creatinine of Cases 4 and 5 (normal value: 88-160 μmol/ℓ)

portal fibroplasia. Several of the Kupffer cells contained intracytoplasmic, yellowish-green, bile-like pigment globules. In Case 3, in addition to these changes, extensive centrilobular coagulative necrosis and mild fibrosis also occurred.

Kidney: In cases 1 and 3, there was extensive coagulative necrosis of cortical tubular epithelium, as well as widespread dilation of convoluted tubules and glomerular spaces (Fig. 5). The cytoplasm of most of the remaining tubular epithelial cells was vacuolated (Fig. 5). Mild diffuse fibrosis in sections stained with Masson's trichrome, a few focal mononuclear cell infiltrates and scattered haemorrhages were evident in the cortical interstitium. Several tubules at the corticomedullary junction and medulla were slightly distended and contained light pink granular casts. In Case 2 the nature of the changes was similar, but cytoplasmic vacuolation of the epithelial cells was more marked and coagulative necrosis was less extensive than in Cases 1 and 3. In addition to granular casts, some bright red homogeneous casts were present within medullary tubules in Case 2.

Other organs: Post mortem autolysis in the gastrointestinal tract was too advanced for lesions to be identified.

Dosing trial

Clinical signs: Clinical signs in the two steers included anorexia, depression, decreased ruminal movements and photosensitivity accompanied by icterus (Tables 1 and 2). In Case 4 mild coronitis and irritation was noted on Days 3 and 4, while in Case 5 photosensitivity was clinically characterized by irritation and swelling of the ears on Day 4. In both cases the eyes were slightly sunken on Day 4.

Case 4 died suddenly on Day 4, preceded by recumbency and a severely depressed habitus while Case 5 was killed by an overdose of barbiturate on Day 4.

Clinical pathology: The results obtained from the different determinations are given in Tables 1 and 2 and Figures 1-3.

Macroscopical pathology: Necropsies revealed marked icterus in both steers, and mild hydrothorax in Case 5. The lesions in the livers and gall bladders closely resembled those in Cases 1-3. In addition, several scattered petechial haemorrhages occurred in the mucosa of the gall bladders. There was moderate to severe bilateral perirenal oedema, and in Case 5 severe oedema was also present in and around the rectum and urinary bladder.

not been adequately studied under controlled circumstances[16]. Cattle can apparently survive longer periods with non-functional kidneys than non-ruminants. Four bullocks maintained good clinical conditions over an anuric period of up to seven days following bilateral nephrectomy[20]. In ruminants urea nitrogen in the blood and saliva is transferred to the gastrointestinal tract where it is utilized for protein synthesis by micro-organisms[2]. Ruminal and intestinal function is severely impaired in *Lantana* poisoning, and this recycling process may thus be hampered.

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ARTICLE ARTIKEL

A COMPARISON OF HEALTH PARAMETERS IN TWO DIFFERENT CANINE POPULATIONS. PART 1: HAEMATOLOGICAL DATA

G.H. RAUTENBACH*, CHERYL BOOTH** and E.W. HÖHN*

ABSTRACT: Rautenbach G.H.; Booth Cheryl; Höhn E.W. **A comparison of health parameters in two different canine populations. Part 1: Haematological data.** *Journal of the South African Veterinary Association* (1987) 58 No. 4, 179-182 (En). Department of Production Animal Medicine, Faculty of Veterinary Science, Medical University of Southern Africa, 0204 Medunsa, Republic of South Africa.

Blood samples were collected on a random basis from two canine populations. The haemoglobin concentration, erythrocyte count, haematocrit, leucocyte count and differential leukocyte count were investigated in a population of kennelled dogs and a population of dogs of a rural township in a developing country.

The means of five of the nine haematological parameters evaluated were found to be statistically significantly different between the two groups. It is postulated that these differences were due to the difference in diet, type and severity of disease, parasites present in the population, and to a lesser extent to breed and age differences.

Key words: Dogs, health-parameters, haematology, developing country.

INTRODUCTION

The Faculty of Veterinary Science of the Medical University of Southern Africa recently established a clinic in the town of Maboloka in the district of Odi, Bophuthatswana. Maboloka is a rural town with a human population of 26 000 and an estimated dog population of 2 300. The housing is of a moderate to low standard with tin shanty houses being generally present. The average yearly income per household is estimated to be under R3 000,00 (approximately 1 470 U S A dollars). There are no formal sanitary services in the town.

On establishment of the veterinary clinic in the town it was found that the incidence and prevalence of diseases observed were apparently different from those found in the more affluent societies with which the clinicians were more familiar (J van Heerden, Department of Companion Animal Medicine and Surgery, Medical University of Southern Africa, unpublished data). It became apparent that the planning of a health care service to control and prevent disease was a high priority, but no previous data on mortality, morbidity or disease incidence existed for the area. As a sound knowledge of these factors is needed for the planning of preventive programmes, it became necessary to collect information by means of a cross-sectional survey.

This survey was carried out to establish laboratory reference values for a population of dogs whose health was relatively unaffected by veterinary intervention, and also to elucidate the prevalence of certain conditions in the population.

The parameters from the Maboloka dogs were compared to those of a relatively disease-free population and to reference values. For the disease-free population a randomly selected sample of kennelled police dogs was used.

* Department of Production Animal Medicine, Medical University of Southern Africa, 0204 Medunsa, Republic of South Africa.
** Medunsa Animal Hospital, Faculty of Veterinary Science, Medunsa.

MATERIALS AND METHODS

Group I: Two hundred and twenty dogs of the town of Maboloka (Bophuthatswana) were used. The town was divided into 11 areas and twenty dogs were selected from each area by visiting every fourth house on one side of a street. In cases where the home owner had more than one dog, only one dog was selected by drawing lots. Care was taken not to sample dogs from adjacent streets.

The blood samples were collected over a period of 106 days covering the period from the first day of February to the end of May, 1985. Approximately 15 ml blood was collected from each animal using EDTA as a preservative.

Dogs under 3 months of age and those with a body mass of less than 2,5 kg were excluded from the study, as it was considered deleterious to their general health to collect the amount of blood required for the different tests.

The dogs were also examined clinically, external parasites were collected for identification, and faeces were collected to establish the level of internal parasitism.

Group II. One hundred and one dogs of the South African Police Dog School were used as a control group. These animals were all kept in an intensive kennel situation. No selection was done here other than to exclude dogs with a record of chronic disease.

All the animals of Group I were mongrels of a mixed genetic background. The animals from Group II were composed of 57% German Shepherd dogs, 13% Dobermanns, 12% Bloodhounds, 9% Border Collies and 9% Dobermann-Rottweiler crossbred dogs.

The mean estimated age of Group I was 30,8 months with a range of 2 to 96 months. The mean age of Group II was 21 months.

Of the 220 dogs sampled in Group I, 124 (56%) were male and 96 (43,6%) female. None of the females sampled had been sterilised and only four male dogs had been castrated. Group II was composed of 55 male animals (54,5%) and 46 females (45,6%).

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Table 1: Results of haematologal investigation

Test	Group	N	$\bar{x}$	Med	SD	SE	Var	CV%	Act. Range	95% Range	P
Haemoglobin	I	220	113,6	114,0	31,4	2,1	98,6	27,64	28,0-184,0	52,1-175,1	$<10^{-6}$
g/ℓ	II	98	169,4	171,5	21,5	2,2	46,3	12,69	99,0-208	127,3-211,5	
B-RBC	I	220	5,59	5,72	1,52	0,10	2,32	27,9	1,65-8,82	2,61-8,57	$<10^{-6}$
X 10^{12}/ℓ	II	98	7,28	7,29	0,92	0,09	0,84	12,64	9,67-4,76	5,48-9,08	
B-Ht	I	220	0,333	0,335	0,085	0,006	0,714	25,35	0,09-0,53	0,17-0,50	$<10^{-6}$
	II	98	0,487	0,50	0,06	0,006	0,368	12,47	0,30-0,60	0,37-0,61	
B-WBC	I	220	12,25	11,30	5,78	0,39	33885,7	47,18	2,0-48,0	0,92-23,57	0,899
$\bar{x}$ 10^9/ℓ	II	98	12,32	12,00	3,10	0,31	9637,08	25,19	5,70-22,70	6,24-18,41	

$\bar{x}$ — mean; Med — median; SD — standard deviation; SE — standard error of the mean; Var — variance; CV% — co-efficient of variation %; Act. Range — actual range; P — statistical significance of the difference between the means of Group I and II.

In all cases blood was collected from the *vena cephalica antibrachii* or the *vena jugularis externa*; the first being used in the larger dogs and the latter in the smaller dogs. Blood specimens were collected in evacuated tubes (Vac-u-test, Radem Laboratory Equipment) and were transported to the laboratory in a coolbag. The samples were kept in a laboratory refrigerator at approximately 5°C and processed within 24 hours.

The haematological examination consisted of a haemoglobin determination (B-Hb), erythrocyte count (B-RBC), haematocrit determination (B-Ht), leukocyte count (B-WBC) and a differential leukocyte count.

The haemoglobin determinations (g/*l*) were performed on a Coulter Haemoglobinometer (Coulter Electronics, Inc.-Hialeah, Florida, USA). The erythrocyte (x10^{12}/*l*) and Leukocyte counts (x10^9/*l*) were carried out on a Coulter Counter (Coulter Counter Model D N, Coulter Electronics, Inc.-Hialeah, Florida, USA). Controls on the Coulter counter were performed daily with abnormal low and normal Coulter controls (Coulter Electronics, Inc.-Hialeah, Florida, USA). The Coulter haemoglobinometer was calibrated daily with Hb 5, HB 10 and HB 20 calibrators.

The haematocrit determination was performed with a microhaematocrit centrifuge (Heraeus Christ Haemofuge) and reported as a proportion of one.

The erythrocyte indices — mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH) and mean corpuscular haemoglobin concentration (MCHC) — were calculated using the mean B-Hb, B-RBC and B-Ht values as reflected in Table 1.

A quick-stain technique was used to stain bloodsmears. (Diff-Quik, Harleco). The differential eukocyte count was carried out using a microscope and he results reported as a proportion of one.

The mean ($\bar{x}$), median (med), standard deviation SD), standard error of the mean (SE), co-efficient of ariation (CV%), variance (var), the actual range and 5% range (mean ± 1,96 SD) were determined for each f the measured parameters.

The Student's t-test was applied in order to compare he mean values obtained between the two groups. ariance was tested as an integral part of this test to enure that the assumptions of the t-test were met.

The results were also compared to reference values of a healthy group of dogs as given by Schalm, Jain and Carroll[6].

RESULTS

The values of the different parameters are presented in Tables 1, 2 and 3.

The means of five of the nine parameters evaluated in Group I and II were statistically significantly different. The haemoglobin, B-RBC and B-Ht results of Group I deviated markedly from reference values. The mean value and range of results for Group II, on the other hand, compared well with the values described for normal populations, especially as none of the extreme values in this group were excluded.

DISCUSSION

The haemoglobin values of Group I reflected a markedly lower mean than expected. One hundred and twenty four (56%) of the values of this group fell under the lowest value of the given normal range for haemoglobin, while 35 cases in this group had values under 80 g/l, a finding which is consistent with severe anaemia. There was a highly significant statistical difference between the means of Group I and II ($P < 10^{-6}$).

The erythrocyte counts of Group II were generally higher than the mean for a normal population but still within normal range. The mean value for Group I was near the minimum of the normal range and there was a highly significant statistical difference between the values of Group I and Group II ($P < 10^{-6}$).

The blood haematocrit values of Group II compared well with the normal range but those of Group I were again much lower than expected. Thirty four per cent of the blood haematocrit values in Group I were below the minimum of the normal range and 18,6% had values lower than 0,25. There was again a highly significant difference between the values of Group I and II ($P < 10^{-6}$).

The mean corpuscular haemoglobin concentration for both groups were well within normal range. There was however, a marked difference in mean corpuscular

Table 2: Results of haematological investigation

Test	Group	N	$\bar{x}$	Med	SD	SE	Var	CV%	Act. Range	95% Range	P
Neutrophils	I	220	0,384	0,38	0,150	0,01	2,236	39,09	0,10-0,82	0,089-0,678	0,360
Mature	II	98	0,399	0,40	0,104	0,01	1,075	25,99	0,16-0,60	0,196-0,602	
Neutrophils	I	220	0,177	0,160	0,109	0,007	1,188	61,55	0,0-0,56	−0,037-0,39	0,127
Immature	II	98	0,196	0,18	0,089	0,009	0,787	45,19	0,02-0,40	0,02-0,37	
Lymphocytes	I	220	0,329	0,32	0,166	0,011	2,766	50,52	0,0-0,82	0,0-0,655	0,0011
	II	98	0,27	0,26	0,10	0,01	0,989	36,91	0,06-0,54	0,07-0,46	
Monocytes	I	220	0,046	0,04	0,034	0,002	0,117	74,4	0,0-0,20	−0,02-0,113	0,0209
	II	98	0,056	0,06	0,038	0,004	0,144	67,74	0,0-0,16	−0,02-0,13	
Eosinophils	I	220	0,066	0,04	0,063	0,004	0,401	96,35	0,0−0,30	−0,05-0,19	0,085
	II	98	0,079	0,06	0,068	0,007	0,457	85,14	0,0−0,42	−0,05−0,21	

$\bar{x}$ – mean; Med — Median; SD — Standard Deviation; SE — Standard error of the mean; Var — Variance; CV% — co-efficient of variation%; Act. Range — Actual Range; P — Statistical significance of the difference between the means of Group I and II.

Table 3: Calculated erythrocyte indices

Test	Group I	Group II
MCV fl	59,6	66,9
MCH pg	20,2	23,3
MCHC g/dl	34,1	34,7

volume with the Group I mean MCV falling into the microcytic range. The mean corpuscular haemoglobin value of Group I was also on the lower level of a normal range.

The mean blood leukocyte counts for Group I and II both conformed to that of a normal population, but Group I had a very wide frequency distribution.

The mean values for mature neutrophils in both Group I and II were low and the immature neutrophil values were high compared to the collated reference values.

The mean of the lymphocyte differential count of both Group I and II were within the normal range but Group I in particular had a very wide range. There was a highly significant difference between the means of the two groups ($P = 0,0011$).

The proportion of monocytes in the differential count of both Group I and II compared well with the normal range but there was still a significant difference between the means of the two groups ($P = 0,0209$).

The mean value for the eosinophil proportion of the differential count of both Group I and II conformed to the normal.

Comparison of the results of Groups I and II with normal values for the different haematological parameters and comparison of the two groups with each other led to the following deductions: (a) The mean values of the dogs in Group II conformed well to the accepted norm for a healthy population. It must be remembered that no extreme values were excluded from this group. (b) The mean values of the dogs in Group I did not conform to the accepted norm for a healthy population. The range and variation of the data in this group was such that it must be accepted that this was not a homogeneous population. It is clear from the B-Hb, B-Ht and B-RBC values that a large number of dogs in this group were anaemic. (c) The means of five of the nine parameters evaluated in Groups I and II were statistically significantly different. It can therefore be said that the null hypothesis that these two groups come from comparable populations is highly unlikely if judged on haematological parameters.

Differences in the haematological results between groups can be caused by a variety of factors. Certain breeds of dogs have unique haematological features[5]. These factors could play a role in the differences observed between Group I and II. Group I was composed exclusively of mongrel dogs while Group II was composed of 57% German Shepherd dogs, 13% Dobermanns, 12% Bloodhounds, 9% Border Collies and 9% Doberman Rottweiler cross bred dogs. It is known, for instance, that the haematocrit of German Shepherd dogs is usually higher than that of the population average[6].

It is known that the mean age of a group can influence the haematology results[1,7]. Most of the ages of dogs in Group I could only be estimated. The mean age of dogs in Group II, in which the ages of only 8 of the 101 dogs were not known, was 21 months. Only one dog in both groups was under the age of 3 months. In view of the relative similarity in age distribution in these two groups it is doubtful that age played a significant role in the differences observed between group haematology values[7].

Increased blood pressure and release of adrenalin during excitement and fright could cause significant increases in the number of circulating erythrocytes and leukocytes[7]. The dogs in Group I were generally not used to being handled and many of them were frightened. The dogs in Group II were kept in cages and became very excitable when handled. It is doubtful that this factor played a role in the observed differences between the groups since a significant number of dogs in both groups went through stages of either fear or excitement.

Male dogs have a tendency towards higher haematocrit values, haemoglobin concentration and red

blood cell counts than females[6]. There was, however, no appreciable difference in the sex ratio between the two groups.

Pregnancy and lactation can cause an apparent mild to moderate anaemia, probably due to an increase in the plasma volume with resultant dilution of erythrocyte mass in circulation[7]. It is not known how many animals were pregnant or lactating in the two groups but the influence of this factor on the differences seen between these groups is likely to be small.

Technical differences such as the use of different anticoagulants and different counting techniques will obviously play a role in the results obtained[2]. In this exercise the same techniques were used in both groups and therefore it can be safely said that this factor played no role in the differences obtained. The neutrophil counts of both groups showed an apparent shift to the left with an increase in the number of immature neutrophils. This was attributed tc the fact that a medical technologist performed the differential counts. It is known that this can result in an apparently significant left shift in the analysis of canine blood due to more rigid requirements for segmentation in some human classifications[7].

Hinton and Jones[4] showed that the haematocrit of blood samples received by post was significantly higher than that of fresh samples. The samples of Group I were usually analysed within 24 hours after sampling while those of Group II were analysed within 6 hours. As the Group II mean values were much higher than those of Group I, this factor could not have played a role in the differences observed between the groups.

It is known that a variety of nutrients such as iron, protein and the B-vitamins are required for optimal haemoglobin synthesis. Haxhe[3] demonstrated that erythropoiesis is suppressed as soon as food restriction is induced. The animals in Group II were fed a balanced commercial dogmeal while those in Group I were fed mostly on table scraps, and a large percentage were clinically judged to be in a state of malnutrition (Rautenbach, unpublished data.). It would be expected that the anaemic dogs in Group I would suffer to an extent from non-regeneration of red cells if the anaemia were partly caused by malnutrition. It would also be expected that this would cause a hypochromasia. There was, however, no difference in the MCHC between groups. A complicating factor was probably an iron deficiency in Group I. Group I had a mean serum iron level of 18,9 mol/l compared to the 28,7 mol/l of Group II (Rautenbach, unpublished data.). As expected, Group I was affected by this iron deficiency and the value of the MCV was in the microcytic range. It is known that iron deficiency anaemias will be normocytic — normochromic in the early stages, but a fully expressed deficiency will be characterized by a microcytic hypochromic anaemia[6]. It is also known that microcytosis precedes hypochromasia[6].

With the microcytosis present in Group I the MCH was probably a better indicator of haemoglobin synthesis than the MCHC. As can be seen from Table 3, the MCH value of Group I was lower than that of Group II. It is probable that malnutrition and iron deficiency played a major role in the differences seen in the haematology results of Group I and Group II.

Variations in the type and severity of disease would obviously play a role in the differences observed in haematology results between the two groups. Animals housed in large groups are usually more prone to transmission of infectious diseases and parasites than individual pets[5 7]. In this study, however, it appeared from the haematology results that Group I was a disease-ridden population, which indicated that this was not a normal population of individual pets. The study did establish that the Group I population suffered heavily from internal and external parasites, that infectious disease was rife in the population and that even the incidence of neoplasms was high (Rautenbach, unpublished data.).

It is postulated that the differences between Groups I and II as seen from the haematology results were largely a result of differences in diet, type and severity of disease, parasites present in the population; and to a lesser extent, to breed and age differences.

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ARTICLE **ARTIKEL**

THE USE OF A DIFFUSION TEST FOR THE DETECTION OF ANTIBIOTICS IN THE TISSUES OF SLAUGHTER STOCK

DENISE N. LLOYD* and D. VAN DER MERWE**

ABSTRACT: Lloyd D.N.; Van der Merwe D. **The use of a diffusion test for the detection of antibiotics in the tissues of slaughter stock.** *Journal of the South African Veterinary Association* (1987) 58 No. 4,183-186 (En). Department of Infectious Diseases and Public Health, Faculty of Veterinary Science, Medical University of Southern Africa, 0204 Medunsa, Republic of South Africa.

The Brilliant Black Reduction Test Kit (BR Test), which is widely used to detect antimicrobial residues in milk, was adapted to detect residues in the meat and tissues of slaughter stock. The adaptation consisted of placing kidney and muscle tissue samples into 2,5ml diffusion cups containing 0,4ml media plus *Bacillus stearothermophilus* spores and brilliant black indicator. A preliminary trial undertaken to test the lower limits of sensitivity of the adapted BR Test to a number of the more common antibiotics used in food animals, was followed by a survey involving 943 pigs slaughtered at one abattoir.

Samples were tested from 87 suppliers of which 11 regularly marketed pigs with detectable antimicrobial residues. Most of these pigs came from large pig producing units. Three suppliers marketed pigs with suspicious reactions. No residues could be detected in pigs from the remaining 73 suppliers.

The BR Test was found to be a quick, inexpensive, practical screening test which could be utilized for the routine detection of antimicrobial residues in slaughter stock at all South African abattoirs.

Key words: Diffusion test, *Bacillus stearothermophilus,* antibiotics, detection, tissues, meat, pigs.

INTRODUCTION

The discovery and use of antimicrobials has had a profound impact on the life and health of both man and animals. By controlling infectious diseases and promoting health, these drugs have greatly improved the quality of life for the individual and for society at large. However, the presence of antibiotic residues in the tissues of food animals represent potential dangers that cannot be detected during routine post mortal meat inspection. These residues may endanger man's health by producting direct allergic reactions in the consumer of contaminated meat and meat products. These effects range from mostly mild allergic reactions to even fatal anaphylactic shock[2 6 11 12].

The residues of most of the antibiotics used in veterinary medicine are not destroyed by normal storage and cooking procedures[2 5-6 8-10-13 15]. Furthermore, a proportion of the South African population prefers to consume raw or semi-cooked meat and meat products e.g., biltong and smoked and pickled pork products. Meat and organs should therefore be routinely tested to ascertain whether or not antibiotic residues are present.

Bacillus stearothermophilus as a test organism has been used successfully by various authors to detect antibiotic residues in the meat, organs and urine of slaughter animals[3 7]. This test has been further developed and is now available in kit form; The Brilliant Black Reduction Test (BR Test Kit, Enterotox Laboratories, West Germany.)

The test was originally developed to detect antimicrobial residues in milk. This standardised test consists of two vials, one containing *B. stearothermophilus* spores plus brilliant black indicator (lyophilized powder) while the other vial contains synthetic medium. The inoculum size is standardised so that the lowest level of a wide variety of antibiotic residues can be detected (F Muller, Enterotox Laboratories, Krefeld, West Germany, personal communication). Brilliant black indicator is added to the assay medium so that samples containing an antibiotic can be easily identified. If an antibiotic is present, the test organism is inhibited and the assay medium remains blue. If no antibiotic is present, the growth of the test organism reduces the brilliant black indicator to a yellow colour.

Using the B R Test, A Ebrecht (Enterotox Laboratories, Krefeld, West Germany, personal communication) was able to detect antibiotic residues in the meat and tissues of slaughter stock at the following minimum levels: procaine penicillin 0,0035 I U/ml; ampicillin 0,0020 μg/ml; oxytetracycline 0,10 μg/ml; dihydrostreptomycin 3,200 μg/ml; chloramphenicol 2,00 μg/ml; lincomycin 0,20 μg/ml. The conclusion was reached that the *B. stearothermophilus* organisms were sufficiently sensitive to detect the residues of most of the antibiotics administered to slaughter animals. This finding was confirmed by Bielecka et al.[3].

This paper deals with the use of the BR Test to detect antibiotic residues in the meat and tissues of pigs.

MATERIALS AND METHOD

Preparation of BR Test Kits

The agar powder of the kits was dissolved in 80 ml distilled water and autoclaved at 121°C for 15 minutes. After sterilization, the agar was cooled down to and maintained at 60°C to prevent solidification. The lyophilized powder containing *B. stearothermophilus* was dissolved in 5ml warm (60°C), sterile, distilled water and added to the agar.

The mixture was stirred for a further 2 minutes. Standard 2,5 ml polystyrene cups with "plug-tite" caps (N.T. Laboratory Supplies) were filled with 0,4 ml of

* Department of Infectious Diseases and Public Health, Faculty of Veterinary Science, Medical University of Southern Africa, 0204 Medunsa, Republic of South Africa.
** Department of Agriculture,. Economics and Marketing, Krugersdorp Abattoir, Krugersdorp.

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the mixture. The mixture was allowed to solidify and then stored at 4°C. All media were used within one month of preparation.

Animals

In a preliminary trial to test the lower limits of sensitivity of the BR Test to a number of antibiotics used in South Africa, four pigs from an extensive farming enterprise known to be free of antibiotic residues, were used as a source of tissues to which known quantities of antibiotics were added. A survey was subsequently conducted at the Krugersdorp Abattoir by systematically sampling every 10th pig delivered for slaughter during the period July 1985 until the first week of December 1985. A total of 948 pigs from 87 suppliers were sampled.

Sample preparation

A portion of the *pars muscularis* of the diaphragm and a portion of the kidney, approximately 30x30x5mm in size, were removed from each of the sampled carcasses. The capsule was stripped off the kidney and the peritoneum/pleura was removed from the diaphragm. A 2x2x8mm piece of kidney (incorporating both cortex and medulla) and muscle tissue were punched out with a specially designed meat borer (Enterox Laboratories, West Germany). The knife was rinsed and sterilized between carcasses.

Dilutions of antibiotics were prepared using phosphate saline buffer (PBS) (pH 7,0) (prepared according to Bancroft & Stevens[1]), as diluent (Table 1).

Test procedure

Preliminary trial: Each antibiotic was individually tested by soaking tissue and kidney samples from the known negative pigs at room temperature in 0,5 ml of the different antibiotic dilutions for a period of two hours to ensure tissue equilibration. Kidney and muscle samples were then placed directly onto the agar in the sample cups.

Survey samples: tissue and muscle samples were placed directly into sample cups containing 0,4 ml agar mixture. A standard control disc containing 0,05 IU penicillin was utilised to monitor the sensitivity of the organism in each batch of medium used. Negative controls were set up for each antibiotic used. After a pre-incubation period of one hour at 4°C, the sample cups were incubated in a water bath at 62-64°C for 2 hours.

Statistical information

Seventy suppliers in the study were subsequently contacted. They were asked for the following information: number of breeding sows; number of pigs marketed per week and where they were usually marketed; any speculation; feed used; feed additives; injectables containing antibiotics used and when they were last administered; the withdrawal period adhered to: (time interval measured from when the animal was removed from medication until slaughter). Seventeen suppliers could not be traced.

The data were evaluated by means of Chi-square analysis.

RESULTS

Results of the preliminary trial are presented in Table 1.

In the preliminary trial, residues of penicillin, dihydrostreptomycin, chloramphenicol and oxytetracycline were detected in both kidney and muscle tissue down to the lowest dilution of antibiotic tested. Residues of lincomycin could not be detected in kidney and muscle tissue at the lowest dilutions used. Residues of this antibiotic could not be detected to a level of 0,75 µg/ml.

Results of the survey revealed that eleven suppliers marketed pigs containing detectable antibiotic residues, seven of them more than once. Three suppliers marketed pigs with suspicious reactions in the muscles, each on one occasion only (Table 2). Two of the positive suppliers were speculators. They bought large numbers of pigs at sales throughout the country, fattened and marketed them. No antibiotic residues could be detected in pigs from 73 suppliers.

Most of the pigs containing antibiotic residues were marketed from large pig producing units (Table 3). Because the two speculators in the positive study marketed more than 1 000 pigs per month they were classified as large pig producing units. A Chi-square test of the values showed that the proportions of positive carcasses differed significantly depending on the size of the farm. The larger production units showed

Table 1: Antibiotic standards, dilutions used and results of preliminary trial

Antibiotic standards	Dilutions used	Kidney results	Muscle
Penicillin	0,1875 IU/ml	Blue	Blue
(Salcillin; 300 000 IU/ml,	0,05 IU/ml	Blue	Blue
Salisbury S.A.)	0,0375 IU/ml	Blue	Blue
Streptomycin	10,0 µg/ml	Blue	Blue
(Dihydrostreptomycin; 100 g/ml,	5,0 µg/ml	Blue	Blue
Milvet)	3,0 µg/ml	Blue	Blue
Chloramphenicol	10,0 µg/ml	Blue	Blue
(Chloramphenicol 10%; 100 g/ml,	8,0 µg/ml	Blue	Blue
Panvet)	4,0 µg/ml	Blue	Blue
Oxytetracycline	5,0 µg/ml	Blue	Blue
(Liquamycin 100; 100 g/ml,	1,0 µg/ml	Blue	Blue
Pfizer)	0,5 µg/ml	Blue	Blue
Lincomycin	0,75 µg/ml	Blue	Blue
(Lincocin; 100 g/ml	0,50 µg/ml	Yellow	Yellow
Upjohn)	0,25 µg/ml	Yellow	Yellow

Table 2: Number of pigs with detectable residues

Supplier	Sampled	Number of pigs Positive K	Positive M	Positive Both	Suspicious K	Suspicious M	Suspicious Both	Negative
A	46	1	–	4	–	–	–	41
B	33	–	–	–	–	1	–	32
C	34	1	1	2	–	–	–	30
D	3	–	–	1	–	–	–	2
E	9	–	–	–	–	1	–	8
F	17	1	–	–	–	–	–	16
G	51	1	–	2	–	–	–	48
H	41	–	–	5	–	–	–	36
I	36	1	–	3	–	–	–	32
J	9	1	–	1	–	–	–	7
K	48	–	–	–	–	1	–	47
L	154	3	–	7	–	2	–	142
M	4	–	–	2	–	–	–	2
N	11	–	–	1	–	–	–	10
TOTAL	496	9	1	28	–	5	–	453

K = Kidney
M = Muscle

Table 3: Numbers of suppliers classified depending on size of pig producing units and presence/absence of antibiotic residues in respective pig carasses

	Large 1000+ SOWS	Medium 200-500 SOWS	Small Less 200 SOWS	Could Not Trace
Positive Suppliers	5	3	2	1
Negative Suppliers	1	11	48	17

Table 4: Drugs administered to pigs

Drug used	Users
1. Feed additives	
Mecadox (Pfizer)	3
Salpigro (Hoechst)	3
Bayo-n-ox (Bayer)	1
Oxytetracycline	2
Diazol (Chemveld)	1
2. Injectable preparations	
Penicillin/streptomycin	2
Liquamycin (Pfizer)	7
Oxytetra, LA (Phenix), long acting oxytetracycline	1
Kanamycin TAD (Natterman)	1
Peni LA (Phenix), long-acting penicillin	3
Contrabac (Panvet)	1
Chloramphenicol (Panvet)	1
Sulphatrim (Phenix)	1

significantly more positive pigs than the smaller units.

Ten out of the 11 suppliers who marketed pigs with detectable antibiotic residues stated that they had used injectable preparations (Table 4). Medicaments containing oxytetracycline seemed to be the most popular. Many of the suppliers also added growth stimulants containing antibiotics to the feed.

The different antibiotics used by the suppliers who marketed pigs with detectable antibiotics residues are shown in Table 4.

DISCUSSION

In these experiments it was shown that the adapted BR Test is a simple inexpensive test, which does not require the services of a highly skilled technician and is easy to read. Using the BR Test, results are obtainable within three hours of slaughter and contaminated carcasses can be identified on the day of slaughter. Many other microbiological tests require incubation periods in excess of 24 hours[4 14]. In South Africa, carcasses normally leave the abattoir within 24 hours, making these latter tests impractical.

The preliminary trial showed that the BR Test could be successfully adapted to detect antibiotic residues in the meat and tissues of slaughter stock.

As far as can be ascertained, a survey of the antibiotic residue status of slaughter stock has never been undertaken in South Africa.

It is essential that regular residue monitoring is carried out in slaughter stock. However, a practical test method for the detection of antibiotic residues in the meat and tissues of slaughter stock in South Africa has not been recommended by the relevant Authorities. The test described in Schedule 6 of The Standing Regulations under THE ANIMAL SLAUGHTER, MEAT AND ANIMAL PRODUCTS HYGIENE ACT, 1967 (Act No 87 of 1967), cannot be carried out in a satisfactory manner as the parameters of the test are not adequately defined. It was essential therefore that a practical screening method for the detection of antibiotic residues in the meat and tissues of food animals should

be found that could be implemented in South African abattoirs.

It is therefore recommended that the BR test is a practical screening test which could easily and advantageously be implemented and utilized for the routine monitoring of antibiotic residues in the meat and tissues of slaughter stock passing through all South African abattoirs.

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ARTICLE ARTIKEL

AN INSULINOMA CAUSING HYPOGLYCAEMIA AND SEIZURES IN A DOG: CASE REPORT AND LITERATURE REVIEW

G.N. ECKERSLEY*, A. FOCKEMA**, JUNE H. WILLIAMS*, J. VAN HEERDEN*, MARIANNE I. VERMOOTEN* and C.C. HENDERSON***

ABSTRACT: Eckersley G.N.; Fockema A.; Williams J.H.; Van Heerden J.; Vermooten M.I.; Henderson C.C. **An insulinoma causing hypoglycaemia and seizures in a dog: Case report and literature review.** *Journal of the South African Veterinary Association* (1987) 58 No. 4, 187-192 (En). Department of Companion Animal Medicine and Surgery, Faculty of Veterinary Science, Medical University of Southern Africa, P.O. Medunsa, 0204 Republic of South Africa.

A nine-year-old mixed breed dog was presented with a history of mild generalized seizures, weakness, and muscle fasciculations, following periods of excitement and exercise. Investigative procedures included haematology, chemical pathology, faecal analysis, urinalysis, cerebrospinal fluid analysis, hormone assays, computerized axial tomography and scintigraphic imaging. Results of these investigations revealed hypoglycaemia (blood glucose 1,9 mmol l^{-1}), hyperinsulinism (111 $\mu u\ ml^{-1}$) and an amended insulin-glucose ratio of 2643. The glucacon tolerance test was typical for an insulin producing pancreatic islet cell tumour and pancreas scintigraphic imaging revealed focal lesions in the pancreas and liver. Seizures were initially controlled by dietary means and by limiting exercise. Eventual control was obtained by treatment with prednisolone (1 mg kg^{-1} on alternate days) and diazoxide (10 mg kg^{-1} in divided doses daily). Post mortem examination confirmed the presence of a pancreatic islet cell adenocarcinoma with hepatic metastatis.

Key words: dog, seizures, weakness, hypoglycaemia, insulinoma, pancreas scintigraphy.

INTRODUCTION

Hyperinsulinism is a syndrome associated with inappropriate secretion of biologically active insulin by a functional pancreatic islet cell tumour (insulinoma)[25 29]. Clinical signs and complications are directly related to the resultant hypoglycaemia[25].

The first tumour of the islet cells was described in 1902, but it was not until 1924 that the concept of hyperinsulinism was introduced. In 1927 the association of hyperinsulinism and hypoglycaemia in a human patient with metastatic islet cell tumour of the pancreas was reported. The first surgical cure of insulinoma occurred in 1929. In 1935, Whipple, according to Rogers[25], described the insulinoma syndrome in humans, and the first case of hypoglycaemia associated with a pancreatic beta-cell tumour in a dog, was reported by Slye and Wells[25].

Insulinoma is the most common tumour of the endocrine pancreas in dogs[29]. It has also been reported in the cat[20]. A number of reports in the literature have dealt with the clinical signs, diagnosis, treatment and pathology of this neoplasm[1-30]. Insulinomas occur primarily in mature dogs between 8 and 12 years of age[25 29], although they may rarely occur in younger or older dogs as well[25]. There is no breed or sex predilection[4 6 13 19 25 26], but there may be a higher incidence in the Terrier breeds[3 25]. The pathology of the tumour has been described[3 4 12-14 19] and adenocarcinomas of the pancreatic islets are more common in dogs than adenomas[3]. This differs from the situation in man, where 70-80% of insulin-secreting tumours are single benign adenomas, whereas in dogs more than 75% of functional tumours are malignant[3 12 25].

* Department of Companion Animal Medicine and Surgery, Faculty of Veterinary Science, Medical University of Southern Africa, P O Medunsa 0204, Republic of South Africa
** Department of Pathology, Faculty of Veterinary Science
*** Private Practitioner, P O Box 569, Rooihuiskraal 0154

This paper presents a case of insulinoma in a dog, as well as a discussion on the literature on insulinomas.

CASE REPORT

A nine-year-old, neutered male Wire Haired Terrier cross, with a body mass of 7 kg, was presented with a history of episodic weakness, mild generalized seizures with muscle fasciculations and dilated pupils, of 2 weeks duration. These clinical signs followed periods of excitement and exercise. The episodes lasted for up to 2 hours, after which the dog recovered fully. The patient had received anti-convulsant therapy in the form of phenobarbitone, for 2 weeks prior to presentation with no improvement in the frequency of seizures.

The only remarkable findings on physical examination, which included a full neurological examination, were a thin hair coat, continuous facial muscle twitching and dilated pupils.

The diagnostic investigation included haematology, chemical pathology, protein electrophoresis, enzymology, blood glucose, urinalysis, faecal analysis, radioimmunoassays (cortisol, T3 and T4 concentrations), thoracic and abdominal radiographs, electrocardiography (ECG), cerebrospinal fluid (CSF) analysis, a computerised axial tomography scan (CAT scan), and scintigraphic imaging. No seizures were observed while the patient was in hospital.

Analysis of the results (Table 1) revealed a fasting hypoglycaemia, associated with hyperinsulinism. The following tests were performed in order to confirm that the hyperinsulinism was due to an insulin producing pancreatic islet cell tumour:

— Amended insulin glucose ratio[25] (A I G R)
— Glucose: insulin ratio[25] (G : I R)
— Insulin: glucose ratio[25] (I : G R) the results of which are depicted in Table 1.
— Glucagon Tolerance test[25]. The results of the glucagon tolerance test, are depicted graphically in Fig's 1 & 2, and compare favourably with the result

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Table 1: Pertinent laboratory findings in the insulinoma dog

Laboratory test	Day	Salient features
Serum potassium (mmol ℓ^{-1})	1⁰	3,8
	14	3,4
Serum glucose (mmol ℓ^{-1})	1	1,9
	14	1,9
	210	1,4
	217*	2,1
	240+	2,4
Cortisol (nmol ℓ^{-1})	1	299
	14	366
T4 (nmol ℓ^{-1})	1	16
	14	26
Insulin (μu mℓ^{-1})	14	111°
	210	157
A I G R	14	2643°°
G:I R (mg μu^{-1})	14	0,30'
I: G R (μu mg^{-1})	14	3,24^{x}

O Day 1 = day of first presentation
* 1 week after treatment with diazoxide was initiated
+ 1 month after treatment with diazoxide was initiated
° Control dog = 5 $\mu u\ell^{-1}$
°° normal = <30
' normal = 3,3-12,6
x normal = 0,04-0,23

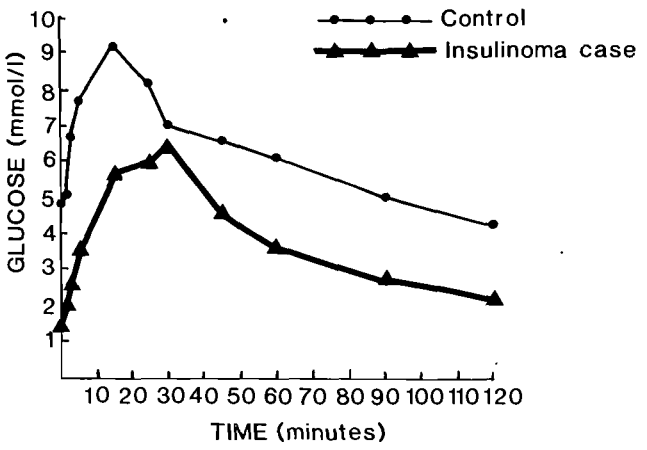

Fig. 1: Plasma glucose concentrations during the glucagon tolerance test

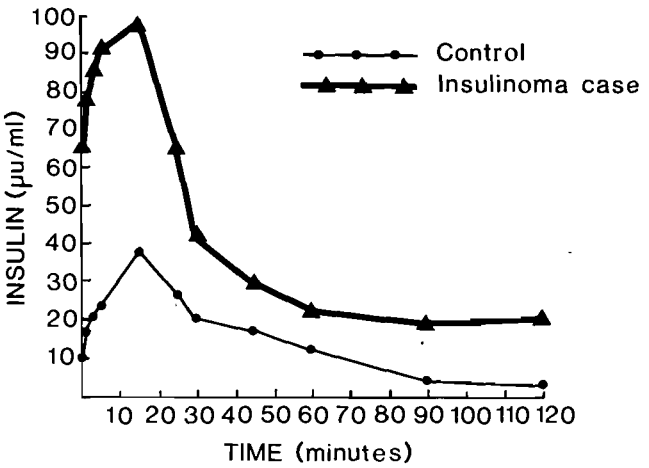

Fig. 2: Plasma insulin concentrations during the glucagon tolerance test

found by Kruth, et al[13] and others[25,26], in cases of insulinomas.

— Pancreas scintigraphy (Gamma camera GE 400 T)

A cephalic intravenous (IV) catheter was inserted and the dog was maintained on 5% dextrose-saline. The dog was tranquillised with xylazine (Rompun, Bayer). Selenium Methionine (^{75}Se methionine, Amersham, England) was used as a tracer and 100 μCi was injected IV. Static pancreas and liver images were obtained one hour later. The scintigraphy revealed a lesion in the pancreas, most likely to be a pancreatic islet cell adenocarcinoma as well as a metastatic lesion in the liver (Fig. 3 & 4).

The neurological signs were controlled for seven months by means of a strict diet. The dog was fed small meals six times a day. The diet had a high protein, moderate fat and moderate complex carbohydrate content. Simple carbohydrates were not to be fed under any circumstances. The owner was advised to keep exercise and excitement to a minimum. In the event of excessive excitement, the dog had to be observed for any prodromal signs and glucolin powder (Glucolin, Glaxo) with water had to be given orally when necessary.

When dietary control was inadequate, the dog was started on a course of diazoxide (Proglicem, Scherag) capsules (10 mg Kg^{-1} divided twice a day) and prednisolone (Prednisolone, Centaur Laboratories) tablets (1 mg Kg^{-1} every other day). The blood glucose increased soon after implementing this treatment regime (Table 1).

Five months later the dog had another seizure, which developed into status epilepticus but was not responsive to glucolin per os, 10% dextrose IV or diazepam (Valium, Roche). The dog died soon after admission into hospital.

Macroscopic autopsy findings

Obesity, pulmonary oedema and congestion, and mild brain oedema were the outstanding features. The lobulation in the pancreas was accentuated by severe interstitial haemorrhage. There was also a round, pale, raised nodule of 1,5 cm diameter in the duodenal lobe of

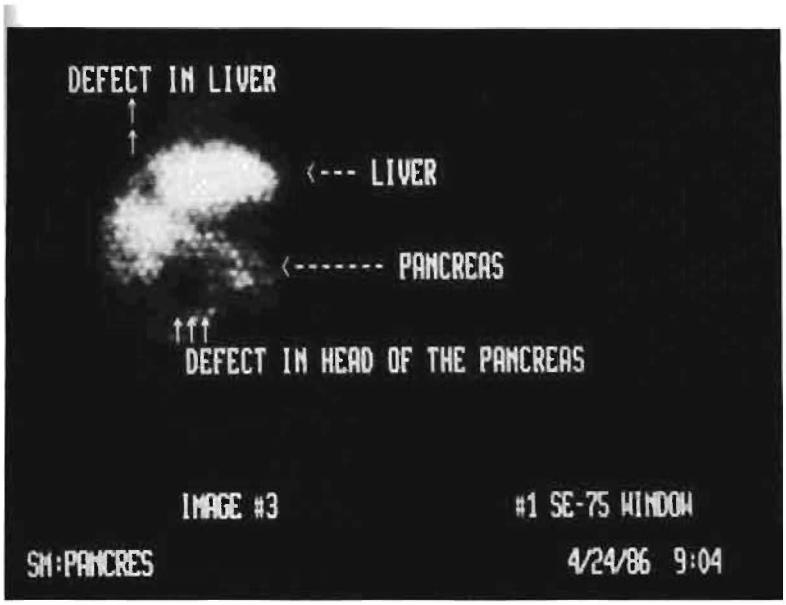

Fig. 3: Pancreas scintigraphy. Defects can be seen in the pancreas and liver

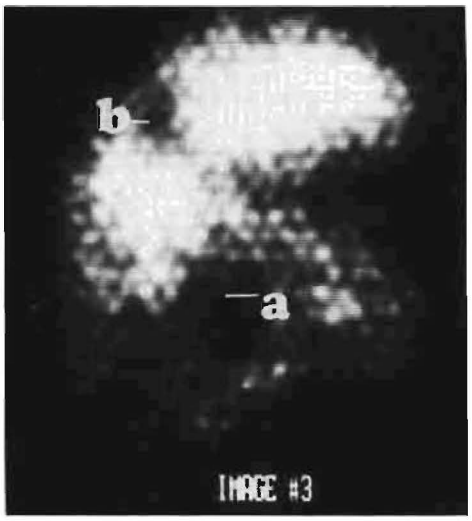

Fig. 4: Enlargement of fig. 3. "a" = pancreas defect. "b" = liver defect. "a" correspond to the tumour in the pancreas and "b" correspond to the metastatic tumour in the liver

the pancreas. A few small (2 mm diameter) pale foci were scattered throughout the right medial lobe of the liver while a single haematoma occurred in the spleen.

Histopathological findings

The nodule observed macroscopically in the pancreas, consisted of a poorly encapsulated mass of neoplastic cells that were predominently columnar in shape. Within the tumour cells the eccentric nucleus was situated in the base of the cell while abundant pink-staining cytoplasm occurred towards the apex. The nuclei were vescicular or dark-staining and varied slightly in size. The cells occurred in clusters, sometimes with a pseudoacinar arrangement, and were subdivided by thin connective tissue strands. Special stains did not assist in determining whether the tumour originated from the endo- or exocrine pancreas.

Focally disseminated haemorrhages and areas of pancreatic necrosis occurred throughout the pancreas. Haemorrhage also occurred in the tumour.

The pale foci in the liver, had a similar appearance to the neoplastic tissue observed in the pancreas. The hepatocytes also manifested cellular changes consistent with those of a steroid hepatopathy (Fig 5). Apart from the presence of a small parathyroid adenoma removed from the neck, no microscopic lesions of note were found in the other organs that were examined. Based on the changes observed histologically, a diagnosis of acute pancreatic necrosis and pancreatic carcinoma with hepatic metastases, was made. It could not be resolved histologically whether the tumour arose from the endo- or exocrine pancreas. This problem is consistent with that experienced in other documented cases[3,12,14].

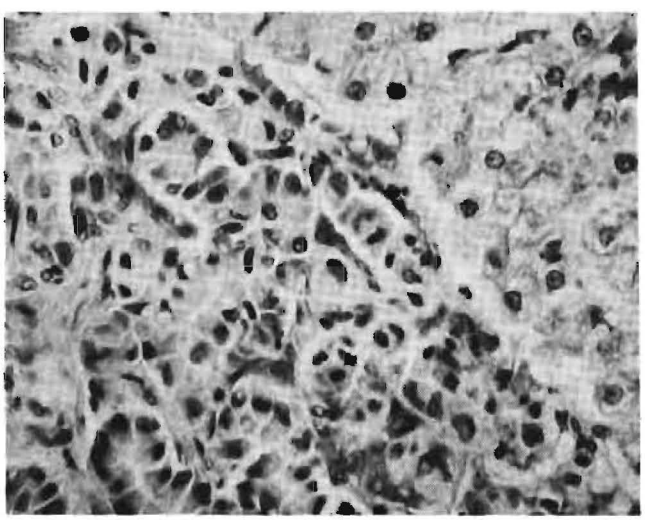

Fig. 5: Haematoxylin and eosin stained section of the liver, showing the metastatic tumour cells (x 200)

DISCUSSION

The metabolism and regulation of insulin as well as the pathophysiology of hypoglycaemia in dogs, has been discussed[25]. Significant hypoglycaemia is generally accepted in the dog as a blood glucose concentration less than 2,8 mmol l^{-1}.

The brain depends primarily on glucose as an energy source[1,27]. The ability to maintain the blood glucose concentration within normal limits, is therefore essential to life. Blood glucose homeostatis is a complex process involving carbohydrate intake and absorption, hepatic production and peripheral utilization[1].

Insulin does not enhance glucose transport into brain cells as it does in muscle and adipose tissue. Simple diffusion of glucose through the blood brain barrier occurs. The brain has a low glycogen reserve and depends on a continuous supply of glucose. As blood glucose concentrations fall, the cortex, and other highly metabolically active brain areas are affected first, (especially the occipital lobes) followed by the metabolically slower, vegetative centres below the cerebral cortex[25]. Significant, acute hypoglycaemia is resisted and counteracted by the release of counter-regulatory hormones (glucagon, epinephrine, growth hormone, ACTH and cortisol). These hormones have an anti-insulin effect and cause an increase in blood glucose by inhibiting muscle uptake of glucose, and promoting hepatic gluconeogenesis and glycolysis[1]. When these physiologic safeguards fail, hypoglycaemia develops. With progressive hypoglycaemia, the brain's extraction of oxygen from perfused blood decreases, thus many signs of neuroglycopaenia resemble those of cerebral hypoxia[1].

The pathogenesis of hypoglycaemia in dogs with islet cell tumours, is either an absolute elevation in insulin levels in the fasting state, or rarely, a failure of insulin levels to decline normally during the post-prandial period (inappropriate hyperinsulinaemia). Hyperinsulinism inhibits glycogenolysis and interferes with glyconeogenesis by direct hepatic effects and inhibition of amino acid mobilisation and lipolysis (therefore has an anti-ketotic effect). The fall in glucose production, together with ongoing glucose utilisation by the brain, results in hypoglycaemia[1]. During periods of exercise or excitement there is increased glucose utilisation by muscle tissue. This may result in a rapid decline in the blood glucose with neuroglycopaenic symptoms[16].

The severity of clinical signs is related to the rate of decline, as well as the degree and duration of hypoglycaemia. The most common clinical signs are mild generalised seizures, generalised weakness, ataxia, posterior paresis, muscle fasciculations, shaking, trembling, focal facial muscle twitching, polyphagia, behaviour abnormalities, blindness, disorientation, syncope and status epilepticus[13 18 25]. Most dogs exhibit multiple signs which tend to become more frequent and severe as the disease progresses[18 25]. Clinical signs may occur intermittently, as the blood glucose level can fluctuate significantly during the course of a day, and significant compensation is possible because of the chronic course of the syndrome. The patient therefore can have a blood glucose concentration of 1,6 to 2,2 mmol l^{-1} and be clinically normal[25]. As discussed previously, compensatory mechanisms come into play during episodes of hypoglycaemia. Catecholamine release may result in, or contribute to signs of muscle fasciculations and dilation of the pupils, due to sympathetic overactivity. All other clinical signs are a direct result of neuroglycopaenia[25]. In dogs with hyperinsulinism, lowered blood glucose levels[25] may be caused by (a) Fasting, which acts through a continued normal to elevated basal insulin secretion. (b) Exercise, excitement or stressful events, which act by increasing glucose utilisation without concurrent decrease in insulin secretion and (c) Feeding, which may cause an exaggerated insulin secretion from responsive neoplastic cells, following a post-prandial rise in blood glucose values. The tumour is generally small and does not lead to cancerous cachexia. It rarely causes significant mechanical damage to any organ but is occasionally associated with pancreatitis or glomerulonephritis[25]. The mean life expectancy after diagnosis is approximately one year[13]. Death may occur naturally because of irreversible hypoglycaemic brain damage or the effects of malignancy. Unsuccessful management of the neurologic disorder frequently results in premature euthanasia[25].

Insulinomas provide a diagnostic challenge to the clinician. The condition is often initially misdiagnosed, and treated with anticonvulsants. The tentative diagnosis of a functional pancreatic islet cell tumour is traditionally based on the demonstration of Whipples triad[25], which includes: (a) a neurological disturbance associated with hypoglycaemia (b) a fasting plasma glucose concentration of $< 2,2$ mmol l^{-1} and (c) relief of neurological disturbances by glucose administration. Whipples triad is characteristic of any cause of hypoglycaemia, which necessitates the consideration of all the differential diagnoses for this entity. The determination of fasting plasma insulin levels via radioimmunoassay may be of diagnostic significance although normal insulin levels have often been described in patients with insulinomas[25].

The amended insulin: glucose ratio (AIGR) whereby the plasma glucose and insulin concentrations are determined after a 12 hour fast, is generally considered to be the most accurate method for diagnosing insulinomas[16 25]. The following formula is used:

$$\frac{\text{Serum insulin } (\mu u\ ml^{-1}) \times 100}{\text{Plasma glucose } (mg\ dl^{-1}) - 30}$$

A resultant value of >200 is considered diagnostic for insulinoma. In obese dogs this value may be higher. The AIGR confirms an abnormally high circulating insulin concentration for the corresponding blood glucose concentration. Ratios do not necessarily correllate with the severity of the syndrome. False positives may occur in other conditions where the blood glucose is very low[18]. It has been suggested that there may be no advantage of the AIGR over the glucose: insulin ratio (G: I ratio) or the insulin: glucose ratio (I: G ratio) because they are all numerically related[10 15].

For the G: I ratio, values $< 2,5$ mg μu^{-1} are considered diagnostic for insulinomas (Rogers[25] according to Slye & Wells). False negatives may occur.

For the I: G ratio values $> 0,3\ \mu u\ mg^{-1}$ are considered diagnostic for insulinomas (Rogers[25] according to Slye & Wells). False negatives have been recorded[25].

The glucagon tolerance test is the procedure of choice[8 13 25 29]. This test evaluates insulin release through direct tumour stimulation within three min post intravenous (I V) injection, and insulinogenesis indirectly through glycogenolysis, 15 to 30 min post injection. These insulinogenic effects are usually exaggerated in the presence of an insulinoma. Diagnostic criteria are[9]: (a) A decrease in blood glucose concentration, one to two minutes after injection (caused by a rapid release of insulin from the tumour). This was not seen in this case. (b) In the absence of severe liver disease, peak glucose $< 7,5$ mmol l^{-1} (c) Hypoglycaemia (glucose $< 2,0$ Xmmol l^{-1} within 60 to 120 minutes. (d) One minute after injection plasma immunoreactive insulin values $> 50\ \mu u\ ml^{-1}$ or an average increase from fasting to one minute of $> 18\ \mu u\ ml^{-1}$. (e) an I: G ratio $> 0,75$ one min after glucagon injection. False negatives have been noted with this test. Acute hypoglycaemia may also occur during the test.

Other tests used, are the high dose IV glucose tolerance test, which may result in post injection hypoglycaemia, the oral glucose tolerance test which is affected by other factors[25], the I-leucine and tolbutamide tolerance tests, which have been used in the past, but are no longer recommended[25]. A diazoxide infusion test is sometimes used in humans but has not been described in dogs[25].

Routine haematology, chemical pathology, electrophoresis, urinalysis and faecal flotations are usually normal, unless the insulinoma is associated with another underlying disease. Hypokalaemia may occur in some patients with insulinoma as was seen in this case. This is due to insulin promoting the movement of potassium from the extracellular to the intracellular compartment. Liver enzymes may be elevated but cannot be used to evaluate metastatic liver disease. Amylase levels may be elevated if associated with pancreatitis. Abdominal and thoracic radiographs, electro-encephalography (E E G),

E C G and C S F analysis, usually do not contribute to a diagnosis. R I A for cortisol may reveal an elevated basal cortisol, which may be a compensatory mechanism[30].

Other diagnostic methods for localising pancreas tumours include intraoperative glucose and insulin monitoring, arteriography, percutaneous transhepatic portal and pancreatic venous sampling, pancreas scintigraphy, ultrasonography and CAT scans[25].

Pancreas scintigraphy is used in humans for diagnosing pancreatic tumours and for identifying liver metastasis. Scintigraphy has been mentioned as a diagnostic procedure[24] in dogs. This procedure is however, only available at specialised institutions but offers a reliable diagnostic procedure for confirming pancreatic tumours. Exploratory celiotomy may be considered as a diagnostic procedure to confirm an insulinoma[9]. The pancreatic regional lymph nodes, and liver can then be examined for visible metastatic tumours, and an attempt can be made to remove any tumours present. It remains the best diagnostic therapeutic and prognostic tool[9]. Unfortunately, metastasis often has occurred before the diagnosis has been confirmed. Nevertheless, recognising the limitations of an exploratory celiotomy, it is still the recommended first step in the management of dogs with hyperinsulinism, unless age or concurrent disease dictates otherwise[9].

The differential diagnosis list for the clinical signs seen in dogs with insulinoma, must include the various causes of seizures and generalised muscle weakness. Although functional islet cell tumours are fairly uncommon, they are a major cause of hypoglycaemia in mature animals. Other causes of hypoglycaemia in mature dogs[1 2 16 18], must be considered eg. extrapancreatic tumours[17 25 27 28], hepatic disease, hypoadrenocorticism, pregnancy toxaemia, septic shock, insulin overdose, starvation and/or malabsorption, hunting dog hypoglycaemia, alimentary tract hypoglycaemia[25] and laboratory error[9].

Therapeutic considerations include surgery, medical management and chemotherapy. Surgery offers the possibility of curing those dogs with benign adenomas and those with adenocarcinomas that have not yet metastasised[9]. Dogs with malignant, metastatic or nonresectable tumours may have a major portion of the functional tumour removed surgically, thus reducing the severity of future clinical signs, and improving the success of medical therapy[69]. However, the size of the tumour, the degree of hyperinsulinism and the severity of clinical signs are not correlated[3]. Preoperative considerations include 5% dextrose-saline infusion, taking note of other medical problems, anaesthesia, adequate preoperative surgical preparation and client contact[25]. Most islet cell tumours have been reported in the right (duodenal) lobe of the pancreas[3 19]. Intraoperatively a single nodule may be found in the left lobe, the right lobe or the body of the pancreas. Multiple nodules may be present or a disseminated form that is only visible microscopically, may be present. Grossly visible metastasis may or may not be present[25]. Blood glucose levels should be monitored intraoperatively[25]. Owners must be told that surgical removal of a neoplastic mass generally is not curative, because of the malignant nature of the tumour. Euthanasia is not recommended if metastatic or nonresectable neoplasms are found during surgery[9]. Many dogs with metastatic disease can be managed medically for longer than a year[6 13]. The surgical approach to an insulinoma case as well as postoperative complications have been reviewed in the literature[6 19 25 30]. Complications include pancreatitis which should be anticipated, and the necessary precautions should be taken. Continued hypoglycaemia may be transient for 24 to 49 hours, or may persist, which would indicate remaining functional tumour cells. The patient may be normoglycaemic, which is the ideal situation, but this may be transient due to tumour regrowth. Transient diabetes mellitus may occur when a partial pancreatectomy has been performed, due to feedback inhibition on normal pancreatic tissue caused by the high levels of insulin produced by the tumour. The patient should be loosely regulated on insulin to help stimulate natural beta cell production[25]. Medical management is an option for patients that are not surgical candidates or if relapses occur post surgically[25]. Conservative medical management should start with a controlled diet, and minimising stress and exercise. The diet should consist of feeding small meals up to six times a day, consisting of high quality protein, a moderate amount of fat and complex carbohydrates[9]. Simple sugars should be avoided, as well as easily digestible moist commercial dog food. Simple sugars are rapidly absorbed from the gastro-intestinal tract, and may stimulate excessive insulin secretion by the tumour, inducing a hypoglycaemic crisis[9]. Treatment of a hypoglycaemic crisis can be initiated by the owner at home, with an oral glucose-containing solution or syrup. When the patient is presented to the veterinary clinic in a hypoglycaemic crisis, 10% or 50% dextrose may be given intravenously. Cerebral hypoxia and oedema may occur and additional therapy may be required (eg. diazepam, corticosteroids, and diuretics) Once stable, the aims of therapy are to reduce the frequency and severity of clinical signs, and to avoid an acute hypoglycaemic crisis. Glucocorticoid therapy should be initiated when a diet cannot control the hypoglycaemic episodes. Glucocorticoids stimulate gluconeogenesis, hepatic glycogenolysis and decrease the peripheral use of glucose (in muscles) by blocking insulin receptors. Prednisolone (start with 0,5 mg kg^{-1} divided twice a day) is the corticosteroid of choice. Alternate day therapy should be initiated as soon as possible. Clinical signs of hypercortisolism will eventually develop, and the owner should be made aware of these[9]. When corticosteroids no longer control hypoglycaemia, Diazoxide therapy (5 - 10 mg Kg^{-1} [24 25] divided twice a day) should be initiated. Diazoxide should be used together with corticosteroids and a controlled diet. Diazoxide is a nondiuretic benzothiadiazide, closely related to the thiazide diuretics[9]. Its action is potentiated by the thiazide diuretics. Diazoxide inhibits the secretion of insulin by decreasing the intracellular release of ionised calcium thereby blocking the emiocytosis of insulin granules (Haemers & Rottiers according to Davis[9]). However, it does not inhibit insulin synthesis or have antineoplastic effects. Diazoxide also stimulates hepatic gluconeogenesis and glycogenolysis by stimulating the adrenergic nervous system and the secretion of adrenaline, respectively. Diazoxide also inhibits cellular uptake of glucose. The net effect is the development of hyperglycaemia. The dose may be increased up to 40 mg Kg^{-1}. The side effects include obesity and gastro in-

testinal disturbances. Other drugs, all of which have potential side effects, that have been evaluated in human patients for their hyperglycaemic effects, include phenytoin, propranolol, L-asparaginase, glucagon, somatostatin and mithramycin[9 25]. Chemotherapy is usually reserved for those patients that do not respond to surgery or medical management[25]. Chemotherapeutic agents, most of which have severe side-effects, include streptozocin[21], fluorouracil, doxorubicin and cyclophosphamide, inter alia.

The pathology of pancreatic islet cell tumours has been reviewed[3 4 12-14 20]. Tumours of the islets are usually discrete nodules (1-3 cm in diameter) readily distinguishable from the glandular pancreas by their firm consistency, their distinct gray-purple colour and their homogenous cut surface[13]. They may also be multiple[3], and in dogs, metastasis to the regional lymph-nodes and liver occurs early[3 14]. Adenomas tend to be encapsulated. Histologically they need to be differentiated from exocrine pancreatic tumours and non-insulin producing islet cell tumours, both of which are extremely rare[3]. Carcinomas of the pancreatic islets, which are more common in dogs than adenomas, can be differentiated from adenomas by their larger size, multilobular appearance, extensive infiltration into the surrounding parenchyma and the presence of metastasis to extrapancreatic sites, especially the regional lymph-nodes and liver[3]. The histological and ultrastructural features of islet cell adenomas and carcinomas are reviewed elsewhere[3 14]. It is beyond the scope of this discussion to describe the histological features. Histochemical stains and immunocytochemistry for insulin granules is sometimes necessary to confirm an islet cell tumour as a beta-cell tumour producing insulin[20].

CONCLUSION

Insulinomas provide a diagnostic challenge to the clinician. They should be considered in the differential diagnosis of seizures and hypoglycaemia in adult dogs. Early diagnosis and treatment is imperative, due to the danger of life-threatening hypoglycaemia. Various tests have been described to diagnose the condition in dogs. Scintigraphic imaging of the pancreas and liver may be helpful in localising the tumour and identifying possible metastasis.

Treatment involves dietary management, medical therapy and/or surgery. Although treatment is not curative due to the high incidence of metastasis, clinical signs can be controlled for up to a year or even longer in some cases.

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CASE REPORT **GEVALVERSLAG**

SUCCESSFUL SURGICAL REPAIR OF A VASCULAR SHUNT OF THE CORPUS CAVERNOSUM PENIS AND PENILE FIBROPAPILLOMATA IN A BULL

R.O. GILBERT*, W.A. LINDSAY** and SUSAN A. LEVINE**

ABSTRACT: Gilbert R.O.; Lindsay W.A.; Levine Susan A. **Successful surgical repair of a vascular shunt of the corpus cavernosum penis and penile fibropapillomata in a bull.** *Journal of the South African Veterinary Association* (1987) 58 No. 4,193-195 (En). Department of Medical Sciences, University of Wisconsin-Madison, Madison, Wisconsin 53706, United States of America.

A single vascular connection between the corpus cavernosum penis and the superficial penile vasculature was diagnosed in an 18-month old Holstein bull with a history of acquired failure to maintain penile erection. Previously normal serving ability had been documented. Multiple penile fibropapillomata were also found. The vascular shunt was identified by contrast cavernosography and ligated, and the fibropapillomata excised. The bull returned to normal function within three months after surgery, and no recurrence of either problem had been experienced by 12 months after surgery.

Key words: Bovine, cavernosal shunt, fibropapilloma, impotentia coeundi, penis.

HISTORY

An 18-month old Holstein bull in the process of being progeny tested as an artificial insemination sire was presented for surgical excision of bleeding fibropapillomata which interfered with semen collection. In addition, the bull had recently demonstrated ventral deviation of the penis making semen collection by artificial vagina difficult and natural service impossible. The bull had previously had the ability to obtain and maintain erection normally and he demonstrated good libido. There was no history of trauma to the penis.

CLINICAL FINDINGS

No significant abnormalities were detectable on palpation of the scrotal contents or rectal palpation of the internal genitalia. Semen collected by electroejaculation was found to be within normal limits for volume, pH, mass and individual sperm motility, and sperm morphology. The penis was examined while extended by manual tension. Multiple papillomatous lesions were found on the penis. These consisted of two large (25 mm) and one smaller (5 mm) papillomata with broad stalks attached to the glans penis adjacent to, but apparently not invading, the urethral orifice. There were also three 5-mm masses on the shaft of the penis, two of which were pedunculated and one sessile (Fig. 1).

On the basis of the history a vascular shunt from the corpus cavernosum penis was suspected. Contrast cavernosography was performed to confirm this diagnosis. General anaesthesia was induced with thiamylol sodium (Bio-tal, Bioceutic Division, Boehringer Ingelheim Animal Health, Inc) after premedication with xylazine (Rompun, Bayvet Division, Miles Laboratories Inc), and was maintained with halothane (Fluothane, Ayerst Laboratories Inc) and oxygen administered via an orotracheal tube in a semiclosed system with intermittent positive pressure ventilation (Tidal volume was 6ℓ, frequency 6/min and inspiratory pressure 26 cm H_2O). With the bull in right lateral recumbency, the penis was extended and anchored to a sterile drape by means of penetrating Backhaus towel forceps placed in the apical ligament and secured by a length of umbilical tape. An 18-gauge hypodermic needle was positioned in the corpus cavernosum penis by inserting it dorsally approximately 10 cm from the tip of the glans penis so that sterile saline solution flowed freely when injected through the needle. A survey radiograph was taken to confirm suitability of exposure factors (50 kV, 3.0 mAs, 90 cm, Lanex medium screen (Kodak), Kodak OL film (Eastman Kodak Co)). Thereafter 10 ml of contrast medium (Renovis X II, Squibb Diagnostics) was injected into the corpus cavernosum penis and a radiograph taken. The penis was rotated 90° in either direction and an additional 5 ml of contrast medium injected and two additional radiographs obtained.

The last radiograph demonstrated contrast material leaving the corpus cavernosum penis and entering a superficial vessel approximately 7 cm from the tip of the glans penis (Fig. 2). The fibropapillomata were also evident on the surface of the penis. The area of vascular communication was distant from any of the papillomata. In preparation for surgical repair of the vascular shunt and excision of the fibropapillomata the penis and prepuce were cleaned with chlorhexidene soap (Hibiclens, Stuart Pharmaceuticals, a Division of ICI Americas Inc) and rinsed with sterile saline solution.

SURGICAL PROCEDURE

A 3 cm longitudinal incision was made in the dorsal midline of the integument of the free end of the penis at the site of the vascular shunt as indicated by the contrast cavernosography. This incision was carefully deepened to expose the tunica albuginea of the corpus cavernosum penis. A thin-walled vessel was found emerging from the corpus cavernosum penis in the position of the shunt as revealed by radiography (Fig. 3). To confirm communication between this vessel and the corpus cavernosum penis, sterile 1% methylene blue solution (Elkins-Sinn Inc) was injected through a 25-gauge needle placed in the corpus cavernosum penis at a distant

* Present address: Department of Theriogenology, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, Onderstepoort, 0110 Republic of South Africa
** Department of Surgical Sciences, School of Veterinary Medicine, University of Wisconsin-Madison

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site (Fig. 4). Blue discolouration of the vessel was evident within seconds. By blunt dissection the vessel was exposed to the level of the tunica albuginea of the corpus cavernosum penis, where it was found to branch before entering the tunica albuginea at four separate sites. Each branch was individually ligated with 3-0 violet monofilament polydioxanone suture (PDS, Ethicon Inc). The tunica albuginea at the point of vascular entry was included in the ligatures. The integument was closed using No. 0 PDS in a simple interrupted pattern. Each papilloma was removed by electrocautery or sharp excision. Larger vessels were ligated and the integument closed with simple interrupted sutures of PDS. Histopathological examination of the excised tissue confirmed the diagnosis of viral papillomatosis.

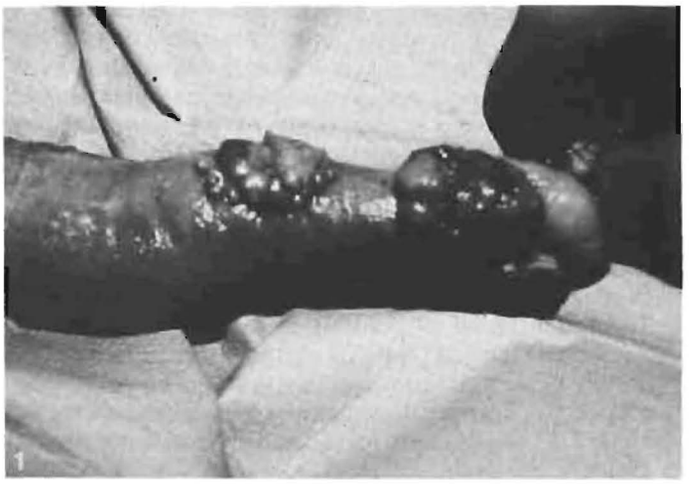
FOLLOW UP

Post surgical recovery was uncomplicated. On the day after surgery a small haematoma was present on the penile shaft at the site of introduction of the 18-gauge needle for injection of contrast medium. It resolved spontaneously within 24 hours. The prepuce was irrigated daily with a 0,2% solution of nitrofurazone (TechAmerica Group Inc). The bull was discharged on the fourth day after surgery, with instructions to the owner to allow the bull at least 4 weeks of sexual rest.

The bull was exposed to an oestrous cow six weeks after surgery, but was reluctant to make a service attempt. By 12 weeks after surgery the bull was willing to attempt service, at which time he copulated successfully. The bull entered regular natural service and semen collection by artificial vagina, and no further problems were encountered in the 12 month period following surgery.

DISCUSSION

Vascular shunts of the corpus cavernosum penis constitute a recently-recognized but well-documented cause of impotentia coeundi in bulls[1,5,6]. In this species, the normal circulation of the corpus cavernosum penis is a closed system. Blood enters the corpus cavernosum penis only via the paired deep pudendal arteries at the



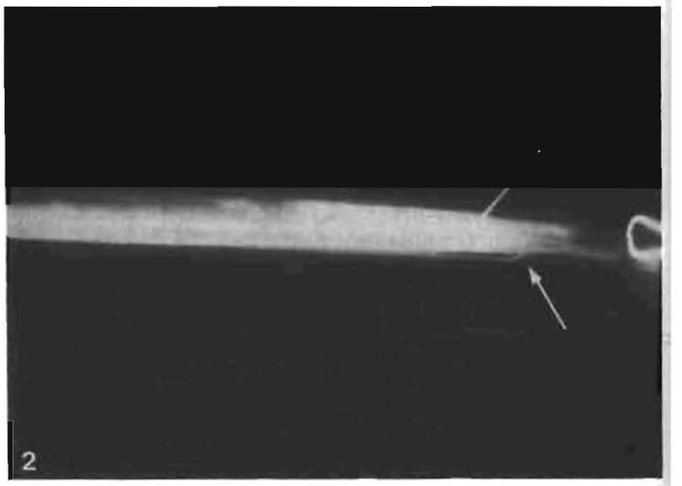


Fig. 1: Pre-operative view of the free end of the penis showing some of the papillomata

Fig. 2: Contrast cavernosogram showing vessel emerging from the corpus cavernosum penis (arrow)

Fig. 3: Communicating vessel dissected free from the fascia. A loop of umbilical tape below it helps to identify the vessel (arrow)

Fig. 4: Injection of sterile methylene blue solution into the corpus cavernosum penis to confirm communication between the dissected vessel and the corpus cavernosum penis

level of the crura penis, and leaves via the homologous veins. The corpus cavernosum penis communicates with neither the corpus spongiosum penis nor the superficial vasculature of the penis[2 6]. In the first stage of erection of the bovine penis, musculature which retains the normal resting position of the penis is relaxed. Arterial dilation allows passive filling of the cavernous spaces of the corpus cavernosum penis, and the penis is extended in response to sexual stimulation. During and immediately before mounting, contraction of the ischiocavernosus muscles and the bulbospongiosus muscles produces a short-lived but dramatic increase in blood pressure within the corpus cavernosum penis to as much as 30 000 mm Hg[3 4]. It is therefore readily understandable why even a small vascular shunt from the corpus cavernosum penis can impair maintenance of erection.

Cavernosal shunts must be differentiated from ventral deviation of the penis (so-called "rainbow penis") which is attributable to a defect of the apical ligament of the penis. In ventral deviation of the penis, the penis does not achieve its full length while straight. In the case of cavernosal shunts, the penis is usually extended normally, but erection is only maintained momentarily before the penis becomes flaccid and droops. Fastidious history taking or careful observation of a service attempt by the bull in question allows distinction between these conditions in the majority of cases. Definitive diagnosis depends on demonstration of the vascular shunt by contrast cavernosography as described in this report.

Acquired and congenital forms of cavernosal shunts have been reported[5 6]. The acquired form is suggested to result from penetrating wounds to the corpus cavernosum penis, and may be secondary to rupture of the tunica albuginea in some cases. Acquired lesions are usually single, in contrast to the congenital form in which multiple sites of leakage commonly exist, frequently located under the apical ligament of the penis. Their position and number render surgical repair in these cases more difficult[5 6]. It is not known whether the congenital form of this condition is inherited, but this possibility has to be considered before electing surgical repair. Distinction between acquired and congenital forms is complicated by the fact that congenitally affected bulls may serve normally for a variable period of time before impotence supervenes. This may be due to progressive enlargement of the sites of leakage[6].

In the case in question, there was no history of a penetrating injuiry to the penis, but the bull had been left among a herd of heifers during a period of layoff from semen collection before the impotence was noticed. At the time of presentation to the Veterinary Medical Teaching Hospital traumatic avulsion of two incisors was detected, suggesting a traumatic episode of some sort, during which the penis may conceivably have been injured. There was no indication that the shunt was related to the multiple papillomata co-existing on the penis. Although the solitary nature of the shunt suggests otherwise, congenital anomaly cannot be excluded with certainty.

In this case, and others treated by the authors, the appearance of the vessel communicating with the corpus cavernosum penis suggested that it was a normal vein and not the result of neovascularization after a penetrating wound (see Fig. 3). The multiple points of entry into the corpus cavernosum penis support this. Similar vessels have also been detected upon dissection of abattoir specimens (Gilbert RO 1986 unpublished observations), suggesting that the pathogenesis of cavernosal vascular shunts causing impotence may be more closely related to abnormal function of the vessels involved than their mere presence. This possibility deserves further investigation.

Surgical repair of solitary cavernosal shunts is a relatively uncomplicated procedure and is accompanied by a high success rate. One case of suspected postsurgical cavernositis has been reported[5]. Heightened awareness of this condition may lead to more frequent diagnosis, and to elucidation of the hereditary nature, if any, of the congenital form, as well as the pathogenesis of both forms of the condition.

Fibropapillomata constitute the commonest tumours of the bovine penis[6]. Most cases resolve spontaneously, although a protracted period of convalescence may be required[6]. Surgical excision was elected in this case in order to return the bull to service more rapidly, and because the surgery could be performed in conjunction with the repair of the cavernosal shunt.

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CASE REPORT **GEVALVERSLAG**

FATAL DISSEMINATED CRYPTOCOCCOSIS AND CONCURRENT EHRLICHIOSIS IN A DOG

M.G. COLLETT*, A.S. DOYLE**, F. REYERS***, T. KRUSE*** and BERYL FABIAN****

ABSTRACT: Collett, M.G.; Doyle, A.S.; Reyers, F.; Kruse, T.; Fabian, B. **Fatal disseminated cryptococcosis and concurrent ehrlichiosis in a dog.** *Journal of the South African Veterinary Association* (1987) 58 No. 4,197-202 (En) Department of Pathology, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, 0110 Onderstepoort, Republic of South Africa.

Laboratory findings in an adult bull terrier presented with a history of anorexia and weight loss included the following: severe anaemia, leukocytosis, neutrophilia, lymphopaenia, thrombocytopaenia, *Ehrlichia canis* morulae in monocytes, hypergammaglobulinaemia, a bleeding tendency, icterus and proteinuria. In addition, a high *Haemobartonella canis* parasitaemia, non-encapsulated yeasts on urinalysis and a localised *Demodex canis* infestation were present. Treatment for ehrlichiosis was initiated but the dog died.

Lesions found were a severe cryptococcal granulomatous pneumonia and cryptococcal colonies in the lungs, bronchial lymph nodes, kidneys, liver, spleen, heart, meninges, eyes and thoracic cavity. In addition, hyphal forms resembling *Filobasidiella neoformans,* the teleomorph of *Cryptococcus neoformans,* were seen in lung fine needle aspiration smears, impression smears and lung sections. *C. neoformans* was cultured from urine, lung and liver. Lung and kidney also yielded *Salmonella typhimureum.*

Cortical atrophy with T-cell depletion of lymph nodes as well as splenic lymphoid follicular atrophy, typical of chronic ehrlichiosis-induced cell mediated immunosuppression, could have predisposed to the fatal disseminated cryptococcis.

Key words: *Cryptococcus neoformans, Ehrlichia canis, Filobasidiella neoformans,* immunosuppression, systemic mycosis.

INTRODUCTION

Cryptococcosis is an acute, subacute or chronic noncontagious mycotic infection of man and many mammalian species caused by *Cryptococcus neoformans*[7 11 15]. *C. neoformans* is a natural saprophyte or an opportunistic pathogen[11].

In man, many cases of cryptococcosis occur in patients with acquired immune deficiency syndrome[10], Hodgkin's disease, lymphosarcoma, leukaemia[8], diabetes mellitus and in patients receiving long-term courses of corticosteroids[7]. Indeed, Spickard et al. according to Littman & Walter[15] found that coexisting disease occurs in up to 50% of human cryptococcosis cases.

In a report on 6 cases of cryptococcosis in dogs, Sutton[21] found that immunosuppressive factors did not appear to be important. Williad[23] reported cryptococcal chorioretinitis and pneumonia in a cat that was tested feline leukaemia virus positive. In another cat with lymphosarcoma and concurrent localised cryptococcosis, Madewell et al.[18] speculated that the cryptococcosis was an opportunistic infection due to the immunological abnormalities resulting from the underlying leukaemia virus infection. According to Graybill & Alford, referred to by Madewell et al.[18], cell-mediated immunity is important in resistance to cryptococcosis in man.

This report details a case of disseminated cryptococcosis in a dog with concurrent ehrlichiosis and possible ehrlichia-induced cell-mediated immunosuppression.

HISTORY, CLINICAL AND LABORATORY FINDINGS

A four-year-old male Bull Terrier, from the Pretoria North area, was admitted to the Department of Medicine with a history of poor appetite, listlessness, general weakness and weight loss of more than four weeks duration.

The usual Minimum Data Base used in the Department was instituted. This consists of a good history (if available), full clinical examination, a complete blood count, urine and faecal examination and a peripheral blood smear examination.

On clinical examination on Day 1 the following were noted: Rectal temperature 38°C, pulse 130 beats per minute, respiratory rate 30 per minute. The mucous membranes were very pale pink. An area of alopecia (50 mm x 30 mm) on the lateral right hock was affected by demodectic mange which was identified on deep skin scrapings. The dog was emaciated, showed a slight expiratory wheeze and a grade I systolic murmur. The peripheral blood smears indicated a neutrophilia with a left shift and hardly any signs of regeneration in the red cell series. *Ehrlichia canis* morulae were detected in monocytes (Fig. 1). A high parasitaemia of *Haemobartonella canis* was also present. Haematology results (Table 1) confirmed the suspected findings in the peripheral smear, with a relatively low reticulocyte count and a normocytic hypochromic anaemia.

Treatment for the ehrlichiosis and haemobartonellosis was instituted on Day 1 with doxycycline (Doxyvet, Milvet) at 10 mg/kg per os once daily. Urine was obtained on Day 3 and revealed the presence of yeast-like cells on sediment examination (Fig. 2), plus other signs of a moderate nephrosis (Table 2). A urine specimen drawn aseptically by cystocentesis was submitted for fungal culture. On Day 4 a renal biopsy was performed under general anaesthesia and at the same time bone-marrow was aspirated from the wing of the ilium. With the dog under anaesthesia small nodules could be palpated on the surface of both kidneys. Impression smears from the kidney biopsy stained with new Methylene Blue stain were also found to contain the same yeast-like cells. Urinalysis was repeated on Day 5 (Table 2).

* Department of Pathology, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, 0110 Onderstepoort, Republic of South Africa.
** Rhodes Drive, Constantia, Cape.
*** Department of Medicine.
**** Department of Infectious Diseases.

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Table 1: Haematology results

	Normal values[5]	Day 1	Day 5
Haemoglobin g/ℓ	120-180	50	40
Haematocrit	0,37-0,55	0,17	0,15
Red cell count 10^{12}/ℓ	5,5-8,5	2,2	1,5
M.C.V. fℓ	60-77	76	100
M.C.H.C. g/dℓ	32-36	30	27
White cell count 10^9/ℓ	6-17	26,8	93,2
Neutrophils (mature) 10^9/ℓ	3-11,4(60-77%)	13,4 (50%)	68,0(73%)
Neutrophils (immature) 10^9/ℓ	0-0,3(0-3%)	11,5(43%)	19,6(21%)
Lymphocytes 10^9/ℓ	1-4,8(12-30%)	0,5(2%)	- (0%)
Monocytes 10^9/ℓ	0,15-1,35(3-10%)	1,34(5%)	5,59(6%) (active)
Eosinophils 10^9/ℓ	0,1-0,75(2-10%)	0	0
Basophils 10^9/ℓ	rare	0	0
Platelets 10^9/ℓ	200-900	76	clotted
Reticulocytes	0-1,5%	4,3%	0,7%
Autoagglutination	-	+	+
Spherocytes	-	+ +	+
*Prothrombin time S	8-13	not done	25

M.C.V. = Mean corpuscular volume
M.C.H.C. = Mean corpuscular haemoglobin concentration
* Hepato Quik (Boehringer Mannheim GmbH)

Table 2: Urinalysis

	Day 3	Day 5
Appearance	Cloudy	Cloudy
S.G.	1,025	1,023
pH	6,5	6,0
Protein	2+	2+
Glucose	–	–
Ketones	–	–
Bilirubin	3+	3+
Blood	1+	1+
Hyaline casts	2+	3+
Granular casts	1+	3+
*Fungi	3+	3+

* A urine specimen (cystocentesis on Day 4) sent for culture was positive for *C. neoformans* (see microbiology)

The most striking findings on serum analysis were a markedly decreased serum albumin level, a broad-based hypergammaglobulinaemia and elevated alkaline phosphatase levels. Hyperbilirubinaemia mostly due to conjugated bilirubin was also found (Table 3).

The bone-marrow aspirate cytology revealed a slightly elevated myeloid-erythroid ratio of approximately 3:1 with signs of a slight myeloid hyperplasia. Megakaryocytes were not seen although platelets were evident. Plasma cell interstitial nephritis and cryptococcal granulomas were detected in the histopathology of the renal biopsy.

The clinical and laboratory findings were interpreted as follows: A poorly regenerative haemolytic anaemia and thrombocytopaenia as a result of the combined effect of *H. canis* and *E. canis* plus the anaemia seen in chronic inflammatory disease. Hypoalbuminaemia was considered to be due to anorexia, urine protein loss (which was not quantified, however) and possible chronic liver disease. Increased levels of alkaline phosphatase and conjugated bilirubin were suspected to be due to intrahepatic cholestasis.

Liver function would have been evaluated further if the dog's condition had not deteriorated rapidly on Day 5. Because of a mild increase in the respiratory rate on this day thoracic radiographs were taken. These revealed multiple 1 mm to 4 mm foci of increased density in the lungfield. A fine needle aspirate of lung tissue was performed and smears were made. India ink staining demonstrated round yeast cells which featured the characteristic halo of *C. neoformans.*

Because ketoconazole (Nizoral, Janssen Laboratories) therapy was being considered, a whole blood transfusion was given but the dog continued to deteriorate and died on the evening of Day 5.

Table 3: Clinical chemistry results

	Normal values*	Day 4	Direction of change
Total serum protein (TSP) g/ℓ	53-75	57,8	–
Albumin (A) g/ℓ	25-35	10,8	Lower
Globulin (G) g/ℓ	20-37	47,0	Higher
A/G ratio	1 : 1	0,23	Lower
α Globulin g/ℓ	12,8	9,5	Lower
β Globulin g/ℓ	16,8	16,1	–
γ Globulin g/ℓ	6,0	21,4	Very high
Urea mmol/ℓ	3,6-8,9	6,35	–
Alanine transaminase u/ℓ 25°C	<40	27	–
Alkaline phosphatase u/ℓ 25°C	<190	1412	Very high
Bilirubin (Total) μmol/ℓ	<6,8	36,3	Very high
Bilirubin (Direct) μmol/ℓ	<10% of Total	31,2	Very high
Creatinine μmol/ℓ	<133	56,8	–

* Normal values, Clinical Pathology Laboratory, Faculty of Veterinary Science, University of Pretoria.

NECROPSY FINDINGS

The carcase was emaciated. Mucous membranes were pale and haematomas were present adjacent to biopsy sites and venipuncture wounds.

Both lungs were severely consolidated (Fig. 3) and reddish-purple with multifocal, discrete, pale grey subpleural nodules (1 - 20 mm in diameter). Poorly demarcated, pale grey foci were scattered throughout the lung tissue. An inflammatory oedema was present, together with a moderate hydrothorax which contained a large fibrin clot. The epicardium appeared granular.

A few small (4 mm diameter), raised, subcapsular, gelatinous nodules were seen in both kidneys. The liver was enlarged, red-orange and friable. Apart from a small Gamna-Gandy nodule, the spleen as well as lymph

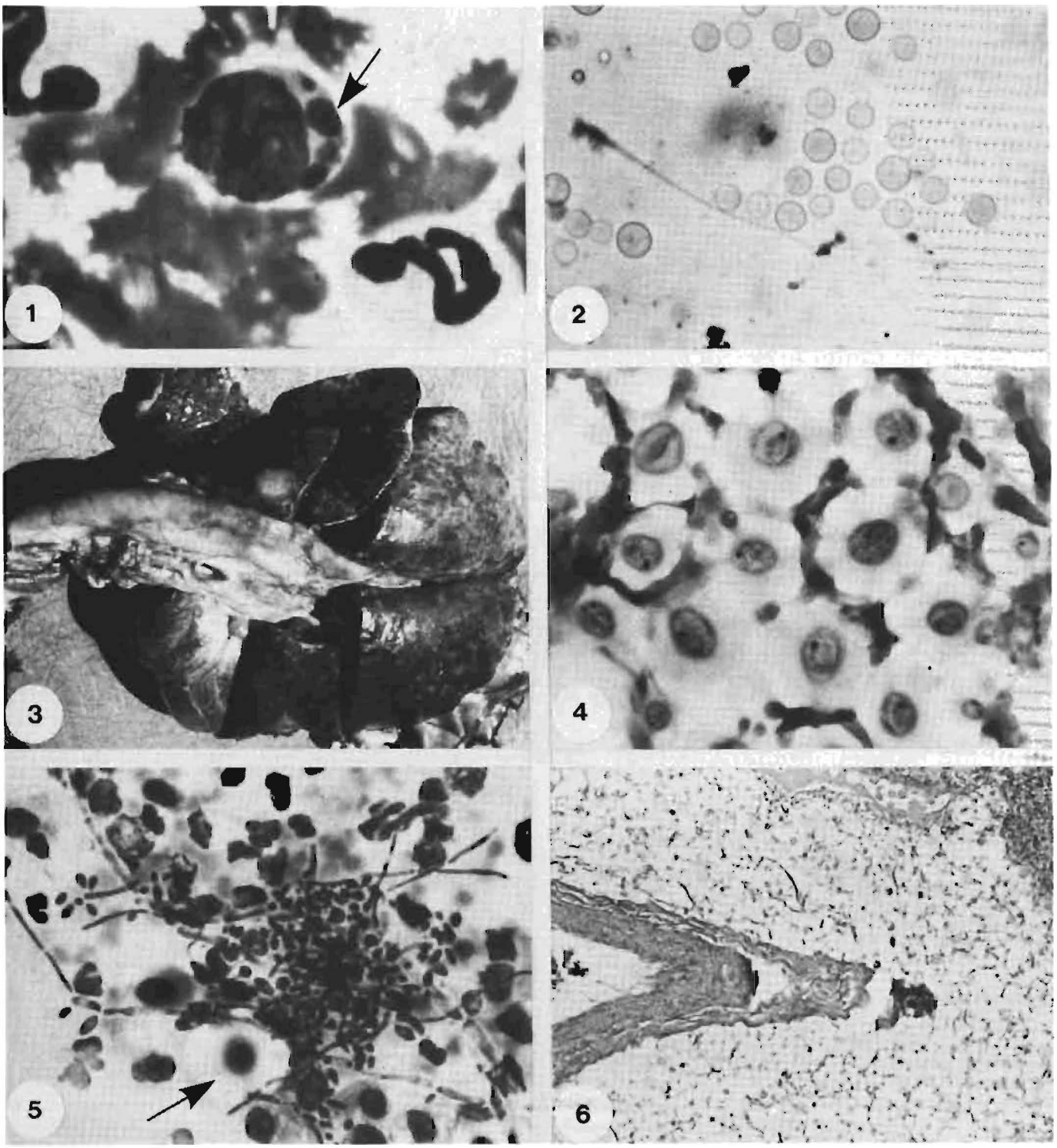

Fig. 1: *E. canis* morula (arrow) in monocyte. Blood smear stained Diff Quik X 1000
Fig. 2: Non-encapsulated yeasts in urine sediment smear. Sternheimer-Malbin X 1000
Fig. 3: Dorsal view of lungs and heart at necropsy
Fig. 4: Colony of *C. neoformans*. Impression smear thoracic clot. PAS X 1000
Fig. 5: Tangle of branching hyphal forms and typical *C. neoformans* yeast (arrow). Neutrophils and alveolar macrophages also present. Lung impression smear. Giemsa X 400
Fig. 6: Lung — large perivascular colony of *C. neoformans*. HE x 40

nodes were of normal size. The heart showed biventricular enlargement, while the left atrioventricular valve was thickened and contained a small haematoma. Red bone marrow hyperplasia was marked.

Other findings were the localised demodectic mange seen clinically and multifocal ulcerative stomatitis.

MICROSCOPIC FINDINGS

Multiple smears were made of lung impressions and the fibrinous clot from the pleural cavity. They were fixed and stained with giemsa, Gram and periodic acid-Schiff (PAS).

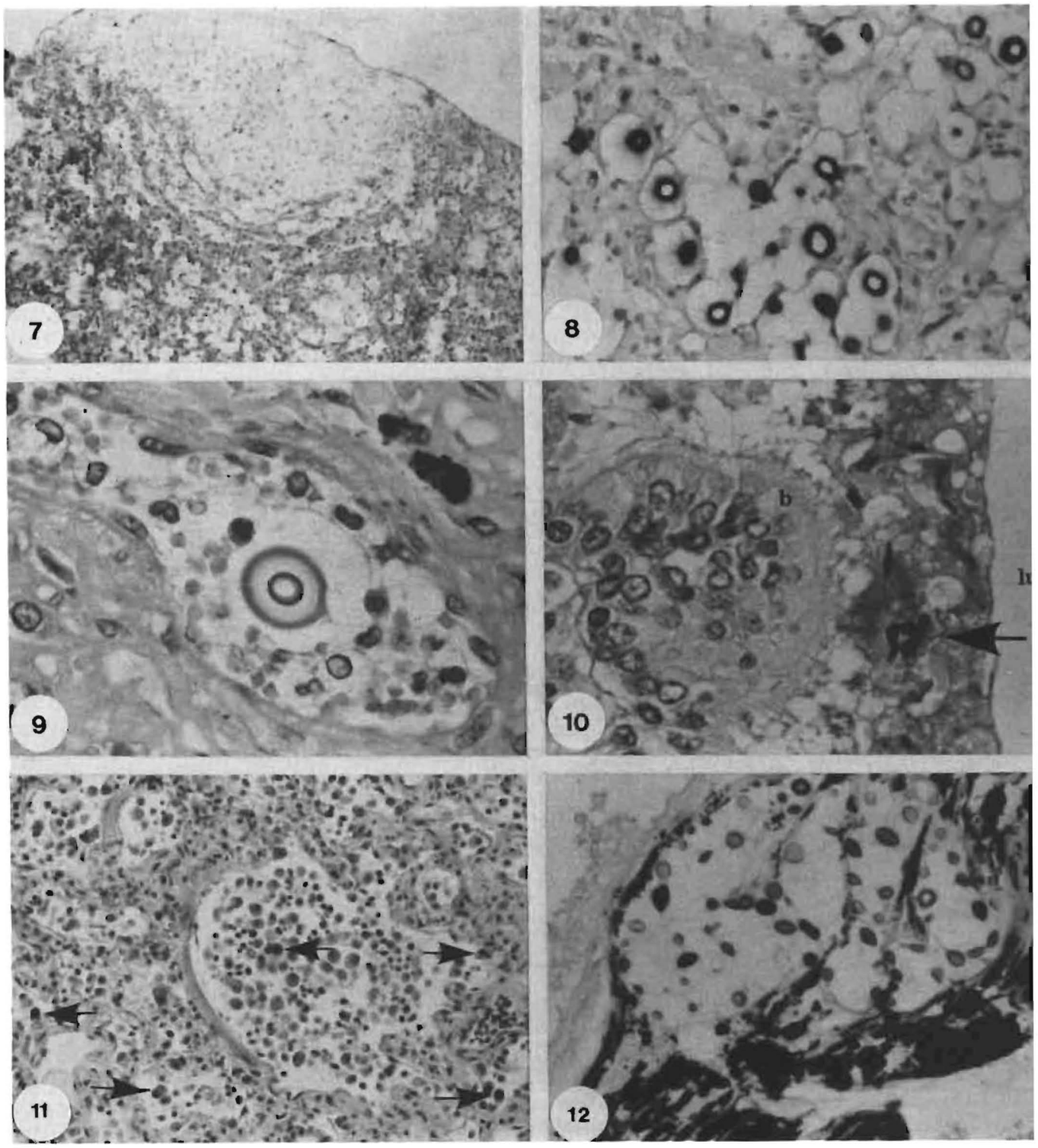

Fig. 7: Lung – subpleural *C. neoformans* colony. HE X 40
Fig. 8: Scattered cryptococci in lung. GMS X 200
Fig. 9: Large *C. neoformans* yeast in lumen of pulmonary blood vessel. PAS X 400
Fig. 10: Budding hyphal forms (arrow) in debris on surface of bronchiole epithelium (b). Lumen (lu). PAS X400
Fig. 11: Lung – massive infiltration of predominantly alveolar macrophages in absence of cryptococci. Intranuclear inclusions (arrows). HE X 100
Fig. 12: Choroid of eye with focal colony of *C. neoformans* without inflammatory reaction. GMS X 200

The impression smears revealed many colonies of yeasts having the typical morphology of *C. neoformans*. Colonies of cryptococci contained larger cells (7-12 μm not including capsule) in the centre with smaller cells (4-5 μm) towards the periphery (Fig. 4). Many cryptococci were budding. Conspicuous too, was the presence of tangles of long, septate hypae (up to 200 μm long), often with branching at right angles as well as terminal or lateral "spores" (Fig. 5). The hypae and yeast cells were PAS-positive and the yeast cells and granules within the hyphae were Gram-positive.

Neutrophils were abundant while alveolar macrophages and occasional small colonies of Gram-negative rod bacteria were also seen. No *Ehrlichia* morulae were seen in macrophages. The clot in the pleural cavity was a fibrinopurulent mass containing blood and scattered cryptococci.

Samples of most organs were collected, fixed in 10% buffered formalin and processed routinely. In addition to haematoxylin and eosin (HE) staining, selected sections were stained with Gomori's methenamine silver (GMS), PAS, Southgate's mucicarmine and alcian blue at pH 2,5.

The most severe pathology was in the lungs. A severe, subacute mycotic granulomatous pneumonia was present with abundant focal disseminate cryptococcal colonies. Cell reaction was minimal, although, in focal areas, a mild inflammatory reaction composed of predominantly lymphocytes was seen. Some cryptococcal colonies were associated with neutrophil and multinucleate giant cell infiltration. Large colonies occurred perivascularly (Fig. 6) and peribronchially in the absence of an inflammatory reaction. Occasional subpleural nodules composed of cryptococcal colonies (Fig. 7) were present. Scattered single cryptococci were dispersed throughout the interstitium, alveoli (Fig. 8) and sometimes within blood vessels (Fig. 9).

Necrotic debris, bacteria and occasional hyphal segments (Fig. 10), which resembled those seen in lung impression smears, were seen in the lumens of some bronchioles. In one area, alveolar macrophages with prominent eosinophilic PAS-positive cytoplasm were especially abundant. Cryptococci in this area were infrequent while mild fibrosis was present and some prominent, basophilic, bronchiolar and alveolar epithelial cells contained large eosinophilic intranuclear inclusions (Fig. 11), resembling those of canine herpes virus.

Alveoli, bronchioles and peribronchiolar lymphatics contained protein-rich globular oedema fluid, while congestion, focal haemorrhage and mild anthracosis were also evident. Thromboses were present in some of the smaller blood vessels.

Bronchial lymph nodes contained many subcapsular colonies of cryptococci. In all lymph nodes examined, cortical atrophy was marked and paracortical areas were severely depleted of lymphocytes. Haemosiderin-laden macrophages and plasma cells were plentiful and completely obscured the paracortical areas and medulla.

In the kidneys, focal colonies of cryptococci were scattered throughout. Some had no inflammatory reaction, while others were associated with a mild neutrophil, macrophage and plasma cell infiltration and severe necrosis of tubules. A chronic, diffuse, global membranoproliferative glomerulonephritis was present. Occasionally exudate (often mineralised) filled Bowman's space. Focal interstitial nephritis with plasma cell infiltration, degeneration of tubules, cast formation and calcification were also seen. The bladder was normal.

There was a severe hepatosis characterised by glycogen infiltration, hydropic degeneration, fatty change and centrilobular degeneration and necrosis. Fibrin was prominent in many sinusoids. Other observations were periportal plasma cell and focal Russell bodied plasma cell infiltration, portal vasculitis and haemosiderin accumulation in macrophages. Small colonies of cryptococci were scattered throughout the liver and some occurred in portal veins.

Lymphoid follicular atrophy was present in the spleen while plasma cells, Russell bodied plasma cells and haemosiderin-laden macrophages were the most abundant cells. Focal calcification was present in trabeculae as well as in the interstitium. Colonies of cryptococci in the spleen were present, but rare. Some small blood vessels were thrombosed.

A few scattered cryptococci, without inflammation, occurred in the leptomeninges of the cerebrum and cerebellum. Focal discrete colonies were present in the retina and choroid (Fig. 12) of both eyes with sequestration of neutrophils in blood vessels in the vicinity.

Focal necrosis and calcification occurred in the zona arcuata of the adrenal cortex. No cryptococci were found in the heart valves although a single colony without a reaction was found in a section of myocardium. No cryptococci were detected in the mouth ulcers.

MICROBIOLOGY

Urine specimens taken on Day 3

Approximately 5 ml of urine was centrifuged at 3 000 rpm for 5 min. A smear of the sediment, stained with India ink, showed the presence of non-encapsulated yeasts. Blood, McConkey's and Sabouraud's agar plates were inoculated and incubated at 37°C. After 3 days minute colonies appeared on the Blood and Sabouraud's agar. At 5 days the colonies reached their maximum diameter of 1 mm. Smears of the colonies stained with Gram or India ink revealed non-encapsulated yeasts. The identity of the yeast was determined as *C. neoformans* using the API 20C Auxanogramme strip (Ayerst Laboratories, P.O. Box 27202, 2011 Benrose).

Specimens taken at necropsy

Lung, spleen, kidney and liver were submitted to the Bacteriology Section at the Veterinary Research Institute, Onderstepoort. *C. neoformans* was isolated from lung and liver while *Salmonella typhimureum* (1), 4, 5, 12:i:1,2 was cultured from lung and kidney.

DISCUSSION

Cryptococcosis shows predilection for the respiratory system[15] (especially the nasal cavity) and the central nervous system[11]. Chorioretinitis[2 3 9 12 21 23] and optic neuritis[3 9 22] are common complications.

In studies of cryptococcosis in man, two main types of lesion were identified[1]. These are the early gelatinous lesions consisting of masses of organisms with minimal inflammatory response and the older granulomatous lesions. Similar lesion types were seen in the lungs and kidneys of this dog. Lung consolidation with multiple

granulomas and severe inflammation seen in this case have been described previously in dogs[2 3 9 17 21 22].

Hyphal forms in cryptococcosis cases have been reported previously[8 16]. Lurie & Shadomy[16] experimented with the hypha-forming Coward strain of *C. neoformans* in mice. Hyphae have also been demonstrated in other strains of *C. neoformans*[13 16]. The hyphae and "spores" found in lung impression smears and sections in this case closely resemble those illustrated by Lurie & Shadomy[16]. No "clamp connections", however, were seen in this case.

Kwon-Chung[13 14] has elucidated the morphogenesis of *C. neoformans*. *C. neoformans* is the asexual or anamorph state while the sexual or teleomorph state is *Filobasidiella neoformans*[13 14]. The hyphal forms in this case, therefore, are conceivably the hyphae and young basidia of the teleomorph while the "spores" are the blastospores or basidiospores. This supposition is strengthened by the absence of other fungal growth on culture.

Coetzer et al.[4] reported a case of cryptococcosis in a dog complicated by an unidentified filamentous fungus (resembling *Paecilomyces* spp.). The latter caused tuberculoid granulomas in the brain and spleen. Illustrations of this filamentous fungus have no similarity with the hyphae found in this case.

Heart and valvular lesions have been described in canine cryptococcosis[6 9 21]. The murmur detected clinically as well as the valve haematoma and endocardiosis in this case are probably unrelated to cryptococcosis, although an isolated colony was found in a section of myocardium.

Cryptococci have been detected on urinalysis[6] and also cultured from urine post mortally in 2 dogs experimentally inoculated intravenously 25 days previously [17]. The fungus has also been cultured from human urine[8]. The significance of the finding of non-encapsulated cryptococci on urinalysis and urine culture is unknown.

Ehrlichiosis was confirmed in this dog by the finding of morulae in peripheral blood smears. Other typical signs found included the severe anaemia, lymphopaenia, thrombocytopaenia, active monocytosis, hypergammaglobulinaemia, bilirubinaemia and bleeding tendency. A characteristic feature of ehrlichiosis is the plasma cell infiltration, in this case involving the spleen, lymph nodes and kidneys. Glomerulonephritis is also characteristic in ehrlichiosis cases[19].

In addition, as in other cases[19] of ehrlichiosis, splenic lymphoid follicular atrophy and cortical atrophy with T-cell depletion of lymph nodes was found. This raises the possibility of cell-mediated immunosuppression and predisposition to opportunistic *C. neoformans* infection. Nyindo et al.[20] have shown that cell-mediated immunosuppression occurs in ehrlichiosis. It is known that deficient cell-mediated immunity plays an important role in systemic mycoses.

The supposition that erlichia-induced cell-mediated immunosuppression occurred is strengthened by the additional findings of concurrent localised demodeciosis, suspected herpes virus in the lung, salmonellosis and haemobartonellosis.

ACKNOWLEDGEMENTS

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CASE REPORT **GEVALVERSLAG**

PARASITIC PNEUMONIA IN A DOG CAUSED BY A LUNG FLUKE OF THE GENUS *PARAGONIMUS*°

I.B.J. VAN RENSBURG*, ANNA VERSTER** and MARGARET A HIZA***

ABSTRACT: Van Rensburg I.B.J.; Verster Anna; Hiza Margaret A. **Parasitic pneumonia in a dog caused by a lung fluke of the genus *Paragonimus*.** *Journal of the South African Veterinary Association* (1987) 58 No. 4,203-205 (En). Department of Pathology, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, 0110 Onderstepoort, Republic of South Africa.

Post mortem examination of a dog that died after a spell of respiratory distress revealed about 20 randomly distributed cystic lesions in the lungs. Those examined each harboured 2 light-brownish conical flukes approximately 10 mm in length. Histopathological examination of such a lesion revealed the presence of *Paragonimus* (lung fluke) embedded in it. The parasite had a typical spiny integument as well as other characteristics typical for the genus. Typical pigmented operculated eggs were embedded in the surrounding tissues. The life cycle of the parasite is briefly discussed.

Key words: *Paragonimus,* pulmonary distomiasis, lung fluke, parasitic pneumonia, dog.

INTRODUCTION

Paragonimiasis or pulmonary distomiasis may be caused by a number of *Paragonimus* spp. *P. westermanii* is widely distributed in China, Japan, Korea, Taiwan and the Philipines. It is also found in parts of India, Malaysia and Indonesia. Other species are occasionally encountered in parts of Africa (Liberia, Zaire, Cameroun and Nigeria) and Central and South America. Domesticated animals such as the dog, cat, pig, goat and bovine as well as wild carnivores such as mink, fox, marten, musk rat, mongoose serve as definitive hosts while man can be accidentally infected by this zoonotic parasite[3,6].

In Southern Africa *P. kellicoti* was reported in the cat by Mönnig in 1934. Many years later a further case was found in a cat from Zululand (Verster, unpublished data 1972) while Proctor & Gregory (1974) recorded the presence in Natal of *Paragonimus* eggs in the faeces of cats and a child[5].

CASE HISTORY

In Mandini, northern Natal during March 1985 a four-month-old Doberman Pincher bitch in a fair condition was presented to one of us (MAH) for clinical examination. The history was one of recent listlessness. A rectal temperature of 39°C, pulse rate of 160 per min and pronounced hyperpnoea were recorded. Slight salivation was present. No remarkable lung sounds were evident on auscultation of the thoracic cavity. Examination of a peripheral blood smear revealed a moderate neutrophilia. Treatment consisted of the subcutaneous administration of atropine sulphate and of etamiphylline camsylate (Millophyline, Centaur) at the appropriate dosage rates and a seven day course of cotrimoxazole (Purbac, Lennon).

Sixteen days later she was again presented for examination, this time with severe respiratory distress, characterised by dyspnoea, cyanosis, excessive frothing at the mouth and an extremely fast, laboured abdominal-type breathing with the head held in an extended position. The rectal temperature was 37,8°C and the pulse rate 116 per minute. Blood smear examination revealed signs of anaemia and pronounced neutrophilia. The animal died before any treatment could be instituted and a necropsy was performed.

Macroscopic findings

Lesions were confined to the thoracic cavity. A pneumothorax was present. The lungs showed areas of consolidation and of emphysema with large airfilled bullae and approximately 20 randomly distributed cystic granulomatous lesions in the parenchyma. The latter were brownish-white, well circumscribed and about 15 mm in diameter. Each incised lesion consisted of a cyst-like cavity harbouring two light brown conical flukes approximately 10 mm long, surrounded by a thin wall. Some cavities contained a brown purulent exudate. It was surmised that the pneumothorax resulted from the rupture of an emphysematous bulla.

One of the parasitic granulomas was fixed in 10% formalin for histopathological examination.

Microscopic findings

The central portion of the granuloma consists of a cavity harbouring two parasites with morphological characteristics typical of those of the class Trematoda (Fig. 1). Each has a spiny integument — the spines being bifid at the tips (Fig. 5) A ventral sucker near the mid-portion is present (Fig. 2). Other features noticed are testes in the posterior half of the body (Fig. 6), a pretesticular ovary, a subcuticular layer of smooth muscle and a digestive tract (Fig. 3).

The parasites are immediately surrounded by an exudate containing many neutrophils, large amounts of granular black-brown pigment (which does not stain positively for iron with the Prussian blue reaction) red blood cells and a globular proteinaceous deposit of uncertain origin (Fig. 4). Surrounding this there is a dense layer of granulomatous reaction in which numerous eggs of the parasites are embedded (Fig. 7). These are large, measuring approximately 100 x 50 μm, operculated and have a yellow coloured shell in H & E stained sections. The shell is birefringent in polarised light. Many have an indented collapsed appearance but lie in a tissue cavity which outlines the original shape of

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Departments of Pathology* and Parasitology**, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, 0110 Onderstepoort.

*** Practitioner, Mandini.

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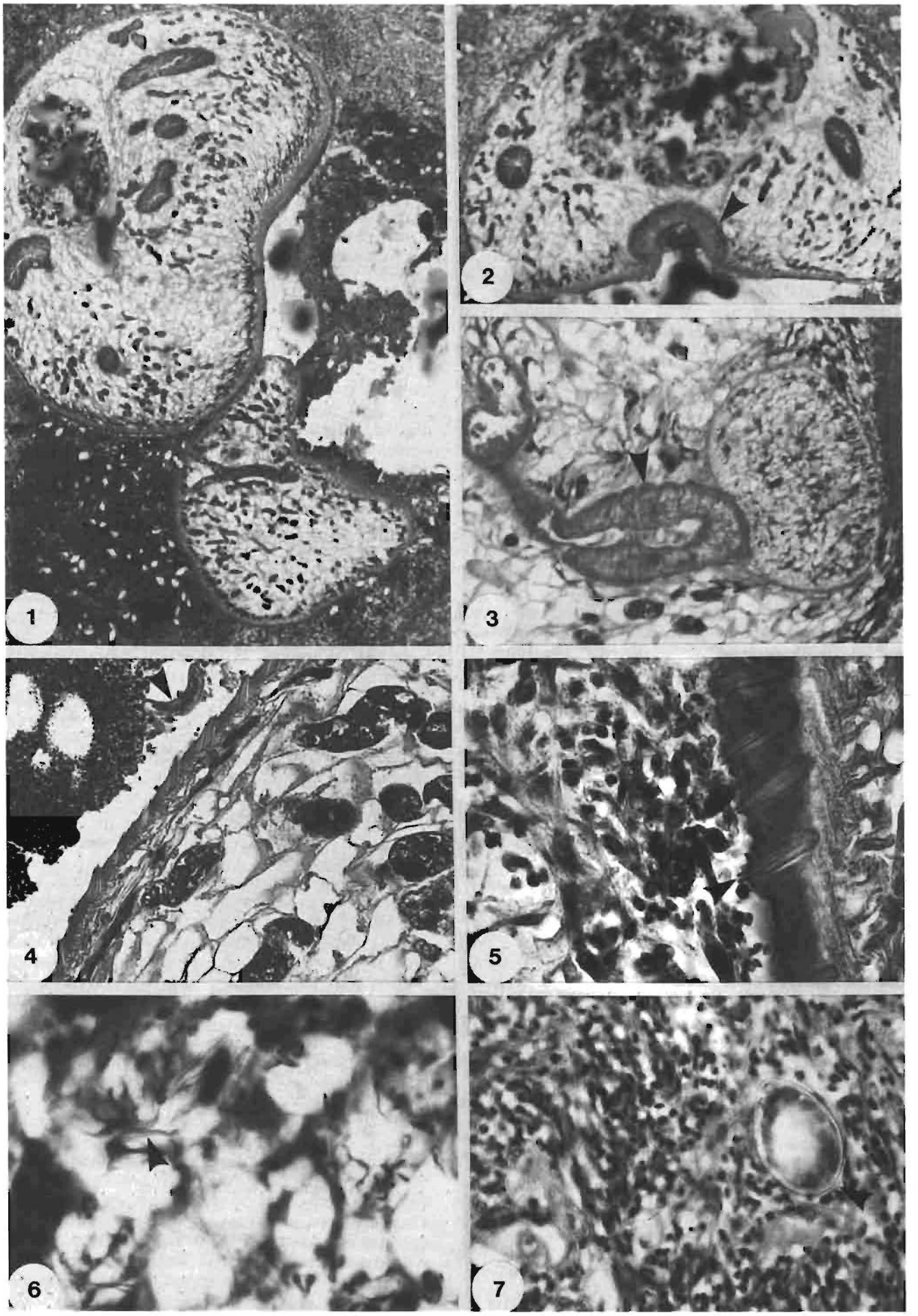

Fig. 1: Two adult *Paragonimus* parasites surrounded by exudate, pigment and numerous eggs

Fig. 2: Section through one of the parasites showing the ventral sucker

Fig. 3: Portion of the digestive tract of the parasite

Fig. 4: The spiny integument, unidentified blackish pigment and a collapsed egg (arrow) are demonstrated

Fig. 5: The bifid tips of the cuticular spines can clearly be seen (arrow)

Fig. 6: Spermatozoa in the testes are demonstrated (arrow)

Fig. 7: A well preserved egg with a single operculum (arrow) is illustrated. Note the surrounding granulomatous reaction

the contained egg. The leukocytic response in these areas consists mainly of large numbers of plasma cells, eosinophils and macrophages laden with haemosiderin and/or lipofuchsin.

The surrounding lung tissue is atelectatic and pneumonic with exudative debris filling the small bronchi.

DISCUSSION

According to Jubb, Kennedy & Palmer[1] *Paragonimus* is the only genus of the *Trematodes* which has its final habitat in the lungs. This fact, as well as the morphological characteristics of the parasite and its eggs which are in accordance with that of *Paragonimus*[1 3 6], strongly supports the identification of the parasite as being a member of this genus. Further support is offered by the finding of two flukes in each of the "inflammatory cysts".

This is the first case known to be diagnosed in a dog in Southern Africa. The incidence of the condition in all animal species is probably low in this part of the world, but cases may pass unrecognised because of unfamiliarity with the parasite.

The life cycle of *Paragonimus* spp. is briefly as follows[1 3 6]: Adults lay eggs in fibrous cystic cavities in the lungs. These escape in mucus from the lung and may be present in the sputum — which often has a characteristic rusty colour[6]. The egg is then swallowed and may be found in the faeces. The miracidia escape and penetrate aquatic or amphibious snails in which sporocysts, rediae and cercariae develop. The latter escape from the snail — particularly in the late summer and autumn[6] and penetrate a suitable freshwater crab or fish in which it encysts. The final host is infested by eating uncooked infested crustaceans or by drinking water in which dead infested intermediate hosts have disintegrated. Metacercaria can survive for 3 weeks in water when released from degenerating crustaceans[6].

In the final host the young fluke penetrates the intestinal wall and migrates through the abdominal cavity, diaphragm, pleural cavity and penetrates the lungs. Other organs such as the abdominal organs, brain, spinal cord and subcutis are rarely infested if abberant migration takes place[3]. The parasite forms a cystic cavity in the pulmonary parenchyma and communication with bronchioles is established in about 5 weeks. The prepatent period is 30-36 days[6]. The interior of the cyst may become partially epithelialized by cells from the bronchioles[3 4 6].

During the migratory phase the parasite may cause an eosinophilic peritonitis, diaphragmatic myositis or pleuritis while multifocal pleural haemorrhages are also common[3 6]. A multifocal chronic eosinophilic granulomatous pneumonia associated with degenerating eggs is often present. These resemble the pseudotubercles caused by schistosome eggs[6]. Infested animals commonly are lethargic and show intermittent cough, dyspnoea and pneumothorax. The diagnosis is made by the demonstration of eggs in the sputum or faeces[3 5 6] or by radiological examination of the lungs[4]. *Paragonimus* ova are fragile and may be distorted or ruptured by hypertonic salt solutions used in faecal flotation procedures[4]. The collapsed appearance of so many eggs encountered in the sections of the case concerned in this report is probably due to tissue shrinkage which normally takes place during formalin fixation.

In the single lesion available for histopathological examination in this case there is no distinct capsule surrounding the two parasites but rather a wide zone of granulomatous reaction in which numerous eggs are embedded. The cell reaction, as to be expected in a lesion of parasitic origin, consists of eosinophils, plasma cells, fibroblasts and macrophages, many of the latter being filled with haemosiderin and/or lipofuchsin. The blackish-brown pigment partially surrounding the parasites could not be positively identified but resembled parasitic haematin.

In the first case of paragonimiasis found in this area the fluke was identified as *P. kellicotti*[2]. This parasite has spines on the integument each of which has a number of points, whereas the parasite described in this paper has spines with bifid tips which more resemble those of *P. westermanii*. Unfortunately adult parasites were not collected at the time of necropsy for a positive identification of the species involved in this case. According to Pechman[4] human infection by *P. kellicotti* has not been reported while the species present in Natal does infect humans[5].

It appears that paragonimiasis could be an endemic disease in Northern Natal/KwaZulu in man and animals and practitioners in the area should be on the look-out for it either as a fulminating disease, as in the bitch described here, or as a subclinical or debilitating condition.

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CONGRESS PAPER KONGRES-REFERAAT

ARTHROPOD PARASITES OF SOME WILD ANIMALS IN SOUTH AFRICA AND NAMIBIA**

I.G. HORAK*

ABSTRACT: Horak I.G. **Arthropod parasites of some wild animals in South Africa and Namibia.** *Journal of the South African Veterinary Association* (1987) 58 No. 4,207-211 (En). Department of Parasitology, Faculty of Veterinary Science, University of Pretoria, 0110 Onderstepoort, Republic of South Africa.

Earlier research on the parasites of wild animals in South Africa is reviewed and the findings of more recent research discussed. The life cycles of various gasterophilid and oestrid fly species are described and the seasonal abundance of their larvae in their zebra and antelope hosts is considered. The seasonal abundance of fleas, lice and ixodid ticks on their hosts is given and the role played by both small and large mammals and some birds in the maintenance of tick populations is described.

Factors contributing to severe parasitism of wild animals are listed and the chemical and biological control of ectoparasites of wild animals are discussed.

Key words: Arthropod parasites, wild animals, South Africa, Namibia.

INTRODUCTION

Early research on the parasites of wild animals in South Africa was devoted mainly to the description of new species. As data accumulated host-parasite checklists were compiled[6 15 17 52 63 69 71] and the geographic distributions, particularly of ticks and fleas, were described[15 63]. Publications of particular note in this regard are those of De Meillon *et al.*[15], Haeselbarth *et al.*[17], Ledger[52], Theiler[63] and Zumpt[69 71]. Later research included life cycles, seasonal abundance, pathology[1 3 5] and control[4 23 31 60 62].

The most important ectoparasites of wild animals are biting flies[17]; the larvae of calliphorid, gasterophilid and oestrid flies[71]; fleas[15 17]; lice[52]; ixodid and argasid ticks[63]; and mites[69]. The present paper reviews work published on the larvae of calliphorid, gasterophilid, hypodermid and oestrid flies, and on fleas, lice and ixodid ticks. It also discusses factors which could lead to severe parasitism of wild animals, and where applicable, deals briefly with control.

CALLIPHORID FLIES

Descriptions of the larvae of the calliphorid flies can be found in Zumpt[71]. Few cases of myiasis caused by the larvae of the metallic blowflies have been recorded in wild animals. Infestations with the larvae of the non-metallic fly *Cordylobia anthropophaga* have been found in black-backed jackal pups *(Canis mesomelas)* (De Vos, 1980 unpublished data) and recorded in the African wild cat *(Felis libyca)* and the leopard *(Panthera pardus)*[71]. The larvae of *Auchmeromyia bequaerti* inhabit warthog burrows where they feed upon the inmates, when these are at home[71].

GASTEROPHILID FLIES

Descriptions of these flies and their larvae may be found in Zumpt[71], who also lists references to earlier descriptions. *Gasterophilus* spp. are found in the gastro-intestinal tracts of zebras while those of *Gyrostigma pavesii* and *Platycobboldia loxodontis* are found in the black and in the white rhinoceros *(Diceros bicornis* and *Diceros simus)* and the African elephant *(Loxodonta africana)* respectively[71].

Burchell's zebras *(Equus burchelli)* can be infested simultaneously with the larvae of 5 or 6 *Gasterophilus* spp.[32 46], and the larvae of 6 *Gasterophilus* spp. have also been recorded in Hartmann's mountain zebras *(Equus zebra hartmannae)*[33 48]. Cape mountain zebras *(Equus zebra zebra)* in the Mountain Zebra National park are apparently infested with the larvae of only 3 species[36 59 67].

The flies attach their eggs to the hair of the limbs of the zebras or to the grazing, where they have to be licked to hatch, or close to the mouth, where they hatch spontaneously. First stage larvae may be found in the peridontal spaces and in the tongue, while second and third stage larvae, depending on their species preferences, are found attached at various sites in the gastro-intestinal tract[32 33 71]. Where the third stage larvae of 2 species use the same site for attachment, competition for space may be avoided by the larvae of 1 species maturing and detaching before those of the other[32]. The mature larvae pass out through the anus and pupate in the soil.

The eggs of *G. pavesii* are firmly attached to the rhinoceros' skin, mainly on the head. It is unknown how the larvae reach the stomach, where they attach and mature, before passing out through the anus[71]. The eggs of *P. loxodontis* are attached to the base of the elephant's tusks and the 3 developmental stages are found in the stomach where they are not attached to the wall. The mature larvae crawl up to the mouth from which they probably drop out when the elephant is feeding[70 71].

HYPODERMID FLIES

The only representatives of this group found in South Africa belong to the genus *Strobiloestrus*[71]. The larvae and adults of these flies have been illustrated by Zumpt[71], while Howard[44] has suggested a possible life cycle and Horak *et al.*[37] have determined the seasonal abundance of the larvae of *Strobiloestrus clarkii* in the gray rhebok *(Pelea capreolus)*. The eggs are attached to the hosts' hair and once the larvae hatch they probably follow a subdermal migration to the site of warble formation[44].

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* Department of Parasitology, Faculty of Veterinary Science, University of Pretoria, 0110 Onderstepoort, Republic of South Africa

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In South Africa the preferred hosts of the larvae are klipspringer *(Oreotragus oreotragus)*, grey rhebok, steenbok *(Rhapicerus campestris)*, mountain reedbuck *(Redunca fulvorufula)*, common reedbuck *(Redunca arundinum)* and kudu *(Tragelaphus strepsiceros)*[49 71]. Cattle, sheep, goats and bontebok *(Damaliscus dorcas dorcas)* that are sympatric with the natural hosts may also be infested[9 24 37 71]. In the south-western Cape Province the life cycle in grey rhebok takes a year to complete[37]. First stage larvae are present in the subcutaneous tissue during December, second stage larvae from December to June and third stage larvae during June[37].

OESTRID FLIES

The flies belonging to this group and their larvae have been described by various authors[1 2 22 45 57 66 68 71], and host-parasite checklists have been compiled by Bedford[6] and Zumpt[71]. Flies of the genera *Kirkioestrus, Oestrus* and *Rhinoestrus* lay first stage larvae in and around the nostrils of their definitive hosts[22 30 71]. The remainder of their parasitic life cycles is completed in the nasal passages, paranasal sinus cavities or peripharyngeal regions. In the case of *Oestrus* spp. in the blesbok *(Damaliscus dorcas phillipsi)*, first stage larvae may migrate to the lungs via the trachea, or they could be aspirated into the lungs, before migrating back up the trachea to the nasal passages[21]. The mature third stage larvae leave their hosts via the nostrils to pupate in the soil. *Oestrus macdonaldi* in the blesbok, *Rhinoestrus antidorcitis* and *Rhinoestrus vanzyli* in the springbok *(Antidorcas marsupialis)*, and *Rhinoestrus steyni* and *Rhinoestrus usbekistanicus* in Burchell's zebras all appear to complete only 1 life cycle annually[16 21 32]. In contrast *Oestrus variolosus* in the blesbok and blue wildebeest *(Connochaetes taurinus)*, and *Kirkioestrus minutus* and *Oestrus aureoargentatus* in the blue wildebeest appear to complete more than 1 life cycle annually[21 22 30].

Flies of the genus *Gedoelstia* deposit first stage larvae on the corneae of the eyes of their definitive alcelaphine antelope hosts[1 3 21]. Two migratory routes to the nasal passages and sinus cavities may then be followed. The one, which is encountered in blesbok and bontebok, involves migration of the first stage larvae in the blood stream from the eyes to the heart and thence to the lungs. Here the larvae break through into the alveoli and then migrate up the bronchi and trachea to the pharynx and soft palate and thence to the nasal passages where they moult to the second and then third stages[3 21 37]. The other, which is followed in the blue and the black wildebeest *(Connochaetes gnou)*, involves migration of the first stage larvae along the optic nerve from the eyes to the subdural space and thence via the nerve tracts and foramina in the cribriform plate to the nasal passages where they moult to the second and then the third stages[3 30]. The remainder of the life cycle is similar to that described for the other oestrid flies.

Gedoelstia spp. larvae may aberrantly infest other antelope species as well as sheep and cattle causing specific oculo-vascular myiasis. The pathology of these infestations in antelope as well as in domestic stock has been described by Basson[1 3 5].

Basson[4] was also able to reduce the incidence of blindness as well as mortality in naturally infested sheep by the oral administration of 3 organophosphate antiparasitic compounds. Snijders & Horak[62] successfully treated natural infestations of *Gedoelstia hässleri* in blesbok with rafoxanide (Ranide, MSD), while measures aimed at preventing the introduction of antelope infested with *Gedoelstia* larvae into small nature reserves have been suggested[23].

FLEAS

All animal species which spend part of their lives in nests or lairs are infested with fleas. Most fleas are host-specific, but some may be found on a variety of animals[15 17]. Fleas of prey animals may be encountered on the animals predatory upon them[17].

Many fleas and their distributions have been illustrated by De Meillon *et al.*[15], while both De Meillon *et al.*[15] and Haeselbarth *et al.*[17] have produced host-parasite checklists.

Surveys have been conducted in which the total numbers of fleas infesting rock dassies *(Procavia capensis)* and warthogs *(Phacochoerus aethiopicus)* have been determined[28 35 43]. No pattern of seasonal abundance could be determined for the fleas *Procaviopsylla creusae* on rock dassies or of *Echidnophaga larina* on warthogs in one of the warthog surveys[28 35]. In the other the sticktight fleas *Echidnophaga inexpectata* and *E. larina* reached peak levels of abundance on warthogs during May and September, while no pattern of seasonal abundance could be determined for the jumping flea *Moeopsylla sjoestedti* on the same animals[43].

LICE

Many of the lice infesting wild animals in South Africa have been described, but many still remain undescribed. Ledger[52] has produced a host-parasite checklist of the lice infesting mammals and birds in South Africa and also lists the references applicable to the descriptions of the lice. Comparatively small animals such as rock dassies and helmeted guinea fowls *(Numida meleagris)* may be infested with large numbers of lice (Horak, 1986 unpublished data) of numerous species[52]. Larger animals again frequently harbour fairly small burdens comprising only a few species[26 30 37].

Although the actual lice burdens of several mammal species have been determined[8 25 26 27 30 31 37], their seasonal abundance has been ascertained on only a few species. Peak burdens of *Damalinia theileri* are encountered on blue wildebeest during April and September of the first year of life, while *Linognathus gorgonus* reaches peak numbers on the same animals from February to April and during August and September[30]. Peak numbers of *Damalinia peleae* are encountered on grey rhebok in the south-western Cape Province from April to August[37]. On common duiker *(Sylvicapra grimmia)*, in the Transvaal, *Damalinia lerouxi* reaches peak numbers during May and *Linognathus breviceps* during October[8]. *Haematopinus phacochoeri* is present in peak numbers on warthogs in Namibia during June, and during August on warthogs in the Kruger National Park[28 43].

The control, with ivermectin (Ivomec, MSD), of natural populations of the sucking lice *Linognathus aepycerus* and *Linognathus nevilli* on captive impala *(Aepyceros melampus)* has been described[31]. This antiparasiticide had no effect on the biting lice *Damalinia aepycerus* and *Damalinia elongata* infesting the impala at the time of treatment[31].

IXODID TICKS

Descriptions of the ixodid ticks infesting wild animals in South Africa have been published by numerous authors.

Theiler[63] lists the references applicable to these descriptions and she has also produced a host-parasite checklist for these ticks as well as describing their geographic distributions. Subsequent to Theiler's work a number of new tick species from wild animals have been described[10-13 18-20 50 64]. Howell *et. al.*[47] have illustrated the geographic distribution of several of the ticks and described their seasonal abundance on domestic stock. The geographic distribution of *Rhipicephalus glabroscutatum* has been illustrated by MacIvor[53], while Clifford *et al.*[14] and Norval *et al.*[58] have illustrated that of *Rhipicephlus kochi* and *Rhipicephalus zambeziensis* respectively. Morel[56] has described the distribution of *Rhipicephalus nitens,* and those of *Amblyomma hebraeum* and *Amblyomma marmoreum* have been illustrated by Walker & Olwage[65].

The development of techniques to accurately quantify the tick burdens of slaughtered animals has made a valuable contribution to our knowledge on this subject[25 29 34 55]. Consequently it has been possible to determine fairly closely the actual numbers of various tick species infesting a variety of animals from mice to giraffes in size[29 37 40].

Using these techniques it has been shown that the helmeted guinea fowl is an important host of the immature stages of *A. hebraeum, A. marmoreum* and *Haemaphysalis silacea*[34]. Small ground-frequenting birds may be infested with the immature stages of both sub-species of *Hyalomma marginatum* and small rodents with those of *Hyalomma truncatum*[40]. The scrub hare *(Lepus saxatilis)* is the preferred host of the immature stages of *H. truncatum* and of both subspecies of *H. marginatum*[40]. The rock dassie harbours virtually only its own host-specific ticks as well as the immature stages of *Rhipicephalus arnoldi,* which infests red rock rabbits *(Pronolagus ruprestris)*[35].

The larger the host species the more likely it is to harbour large numbers of adult ticks[29 41]. Thus eland *(Taurotragus oryx)* and buffalo *(Syncerus caffer)* are excellent hosts of adult ticks of several species[29]. Some animals such as the springbok and the blue and black wildebeest appear to be resistant to ticks and are generally only lightly infested[25 30].

During the past decade the seasonal abundance of several tick species on a variety of wild hosts has been determined. Listed in alphabetical order these ticks are:

(i) *A. hebraeum* on helmeted guinea fowl, warthogs, common duiker, kudu and Burchell's zebras[8 32 34 43 51 54]. The larvae generally are present in autumn, the nymphae in spring and the adults from spring to summer.

(ii) the immature stages of *A. marmoreum* on helmeted guinea fowl[34]. The larvae are most abundant from autumn to early winter and the nymphae from spring to summer.

(iii) *Boophilus decoloratus* on blue wildebeest and Burchell's zebras on which all stages occur in the greatest numbers during spring[30 32].

(iv) *H. silacea* on helmeted guinea fowl and on kudu[34 51]. Larvae occur mainly from autumn to early winter, nymphae during winter and spring and the adults in early summer.

(v) *Haemaphysalis hyracophila* on rock dassies on which the adults are present from April to December[35].

(vi) the immature stages of *Hyalomma marginatum rufipes, Hyalomma marginatum turanicum* and *H. truncatum* are most abundant on scrub hares from autumn to winter[40 61] and the adults of the latter 2 species on Cape mountain zebras and eland during summer[40].

(vii) *Ixodes pilosus* on scrub hares, caracals *(Felis caracal)*, grey rhebok and bontebok on which the larvae are generally most abundant during autumn, the nymphae during winter and spring and the adults during early or late summer[37 42].

(viii) *Ixodes rubicundus* of which the larvae and nymphae are most prevalent on red rock rabbits and caracals from autumn to winter and during spring respectively and the adults on caracals, mountain reedbuck and eland from autumn to spring of the following year[39].

(ix) *Margaropus winthemi* on Cape mountain zebras on which massive numbers of all stages of development occur during July[36].

(x) *Rhipicephalus appendiculatus* on common duiker, impala and kudu[8 26 51]. Larvae are generally present from autumn to winter, nymphae from winter to spring and the adults during the late summer.

(xi) the immature stages of *R. arnoldi* exhibit no clear pattern of seasonal abundance on rock dassies[35].

(xii) *Rhipicephalus distinctus* on rock dassies[35]. Larvae are present in peak numbers from December to March, nymphae during January and March and adults from August to January.

(xiii) *Rhipicephalus evertsi evertsi* on impala, Burchell's zebras and Cape mountain zebras[26 32 36] and *Rhipicephalus evertsi mimeticus* on Hartmann's mountain zebras[33]. Except for the impala on which the immature stages of *R. evertsi evertsi* are present in the largest numbers from May to July[26], no clear pattern of seasonal abundance can be established for either of the ticks.

(xiv) *R. glabroscutatum* on common duiker, grey rhebok, mountain reedbuck, bontebok, kudu, eland and Cape mountain zebra[36 37 54]. This is a 2-host tick and the larvae and nymphae are generally most abundant in autumn and the adults from August to February.

(xv) the larvae of *Rhipicephalus nitens* are present on scrub hares, grey rhebok and bontebok in the greatest numbers during autumn, the nymphae during spring and the adults during summer[37].

(xvi) the adults of *Rhipicephalus simus* are most abundant on Burchell's zebras and warthogs during summer[32 43].

(xvii) and the nymphae of *R. zambeziensis* are most numerous on warthogs during August[43].

Stressful conditions such as a broken leg may lead to increased tick burdens even on animals that are normally tick resistant[30]. Drought, with the resulting reduced resistance of animals and the possible accompanying reduced time spent on grooming, can also lead to a marked increase in tick burdens[41]. These may also be high on animals kept in a habitat in which they did not originally occur[29], or where overstocking of a nature reserve of limited size occurs[23 27].

The role of the red-billed oxpecker *(Buphagus erythrorhynchus)* in the control of ixodid ticks on wild animals has recently been investigated[7]. Although these birds daily consume large numbers of ticks[7], they are unlikely to achieve complete tick control as their own survival depends on the presence of sufficient ticks as prey.

It has been demonstrated that the effective control of ticks on domestic stock on a farm, by means of acaricidal treatment, can result in a marked reduction in the numbers of particularly *A. hebraeum* on wild animals on the farm[38 60]. No effective means of controlling all ectoparasites on free-ranging wild animals has yet been devised.

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CONGRESS PAPER **KONGRES-REFERAAT**

THE VETERINARIAN AS BIOLOGIST*

J. DU P. BOTHMA**

Conflict is the essence of human existence. Whether the conflict is generated by political, academic, personal or other ideals, man always strives to overcome some real or imagined obstacle or to surpass norms set by himself or others. The turmoil associated with conflict leads to the evaluation and acceptance of new ideas. The development of the veterinary profession locally and world-wide has not escaped this force.

Yet much of what we regard as new today, may well be ancient. Thus holism, a concept central to much of modern ecology has been a pivotal aspect of many old religions[24]. The concept of holism has also been central in the development of the veterinary profession, but apparently it has been neglected to the detriment of that profession in recent years. This phenomenon and related aspects will be examined in more detail below.

THE ORIGIN OF THE VETERINARY PROFESSION

Adaptation of living organisms to their environment, and problems of survival are central to ecology and the well-being of animals. This concept gradually became clearer as the medical and veterinary sciences developed mainly from 1700, and the health of man and animal began to be studied in an organised way. As early as 1787, lectures on rural ecology were an integral part of the training programme at the Charenton centre for veterinary education just outside Paris[15].

South Africa: A Brief Review

The following review is based mainly on Gutsche (1979)[15]. In 1781-1784 the zoologist Francois le Vaillant spent three years at the Cape of Good Hope and pointedly remarked on the ecological basis of animal diseases. This was emphazised again 10 years later by Martin Hinrich Karl Lichtenstein when he visited the Cape as a doctor of medicine.

The first incumbent veterinarian in South Africa appears to have been Samuel Wiltshire who was appointed as Colonial Veterinary Surgeon in the Cape Colony in 1874. Veterinary science in South Africa from the onset leant heavily on related biological sciences. Thus in 1887, Mac Owan produced a brochure "Plants that furnish Stock Food at the Cape" which also dealt with toxic plants. As early as 1884 Duncan Hutcheon was ridiculed by his superiors for the conclusion that both botulism and stiffsickness were due to defective nutrition (phosphate deficiency).

In 1886 Jotello Festiri Soga, son of a Scottish clergyman in the Transkei, became the first South African to qualify as a veterinarian, winning a gold medal for botany in the process at the Royal Veterinary College at Edinburgh. In 1891 Arnold Theiler, a young Swiss, arrived in Pretoria to set up practice as a veterinary surgeon.

In 1892 nature had begun sending shock after shock through southern Africa. Locusts, smallpox, bluetongue, rinderpest, horsesickness and other problems were rife. Yet the Transvaal had no Agricultural Department, much less organised veterinary services. By this time, however, the Cape employed several veterinarians and in 1893 their agricultural services were elevated to a full Ministry of Agriculture.

Meanwhile the need for a holistic approach to veterinary problems developed slowly. In the Cape, Samuel Wiltshire still refused to believe the theory that redwater was spread by ticks, rather believing the germ to be in the soil. This led to the appointment of C.P. Lounsbury, an entomologist, in the Cape Colony in 1895 to add to the existing expertise. On 12 December, 1895, Surgeon-Major David Bruce produced his "Preliminary Report on the Tsetse Fly Disease or Nagana in Zululand", a classic in parasitology. In it the tsetse fly was established as a carrier and not the cause of nagana (trypanosomiasis).

Rinderpest now struck in all violence. Yet while Arnold Theiler was asked in early 1896 by President Kruger to help advise his and other governments on combating the disease, the Transvaal still had no formal veterinary service. However, the creation of Rinderpest Committees in the Transvaal in April 1896 led to Theiler's finally being appointed as Government Veterinarian in the Transvaal in May 1896. Also in 1896 Natal re-organized their Agricultural Department, appointing Herbert Watkins-Pitchford as Principal Veterinary Surgeon but without staff or facilities.

By 1897 the rinderpest situation had worsened and economic and social catastrophe reigned as transport and work-oxen died. Yet, despite the arrival of Robert Koch and the efforts of others such as Theiler, Watkins-Pitchford, Danysz and Bordet, the ravages of rinderpest were never fully assessed *ecologically*. This is strange, for not only were the origins of the disease, but its whole course and subsequent effects a vast storehouse of ecological interaction. The mere implications of the statement by the Vryburg magistrate that in 1897 it was possible around Vryburg to ride for an hour through the veld and not to see the spoor of a living animal, are staggering in an ecological sense.

In the Soutpansberg not one ox and hardly a horse (due to horsesickness) was left and the natives reverted to planting cereal crops which were in turn destroyed by locusts and drought. The wild animals were likewise destroyed, and with them also the tsetse fly (see later). This was followed by footrot in donkeys, anthrax in mules and malaria in the people of the Lowveld; indeed a picture of social, economic and ecological turmoil while politically, war loomed on the horizon. Yet the very severity of the rinderpest epidemic propelled veterinary science into the centre stage of the public eye and led to the initially reluctant acceptance of the veterinary profession by farmers at large in 1897.

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** Eugene Marais Chair of Wildlife Management, University of Pretoria, Pretoria, Republic of South Africa

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A seemingly trivial event then occurred which was to have fundamental economic and ecological effects for the world. In August 1897, D. Buchanan had a letter written in Dutch for his signature asking Landdrost Schutte of Pretoria to hire the now long-derelict Daspoort Disinfection Station for three years. Theiler, now Vice-President and an Executive Committee Member of the Pretoria Agricultural Society, was consulted about this letter and he replied that as part of his position as Government Veterinary Surgeon he wanted to convert the Daspoort facilities into a Vaccine Institute.

Environmental chaos again struck in 1897; Pretoria was plagued by a lethal epidemic of typhoid followed by smallpox in the winter. In March 1898 the Transvaal Volksraad approved the proposed institute at Daspoort. On 28 June 1898 the Daspoort Laboratory was occupied. The first laboratory devoted to health in the Transvaal, it initially produced smallpox vaccines mainly, but it gradually expanded into other vaccines.

War then temporarily impaired the development of veterinary science in South Africa. The years immediately following the war led to widespread reorganization of the old and new English colonies, first individually and later as a formal Union. In 1902 F.B. Smith, became director of a new Department of Agriculture for the Transvaal, with a Veterinary Division as one part of it. In 1903 the new Veterinary Division was divided into two sections, with Stockman heading the Contagious Diseases Section and Theiler heading the Bacteriological Laboratory (Daspoort) and the Experiment Station (Potchefstroom).

At the end of September 1917 Theiler announced his impending retirement from Onderstepoort, negotiating at the same time with the Transvaal University College (now the University of Pretoria) and the University of Stellenbosch for an academic appointment. The news that he had decided to accept a position as Professor in Animal Health in the Faculty of Agriculture of the University of Stellenbosch prompted immediate reaction to retain him in Pretoria. Theiler ultimately went to the Armoedsvlakte Experiment Station instead of the University of Stellenbosch. On 27 August 1919 Louis Botha died, to be succeeded by J.C. Smuts as Prime Minister. Smuts soon offered Theiler the joint appointment of Dean of the Faculty of Veterinary Science at the Transvaal University College and Director of the Institute of Veterinary Research at Onderstepoort. Theiler accepted and assumed office on 1 April 1920. The groundwork for significant advances of the veterinary profession in South Africa was now complete.

THE THEILER ERA

There can be no doubt that although other events and personalities also prevailed, Arnold Theiler was a prominent driving force in the early development of veterinary science in South Africa. He was a man full of confidence and drive, and his great opportunism and adaptability to social, economic and political change allowed him to keep the development of veterinary science on course despite many setbacks. The following brief review of his approach is based on Gutsche (1979)[15].

In the years following 1895, Theiler increasingly became involved in government-related investigations and research on various major animal diseases, with rinderpest, botulism and horsesickness prominent on the list. He travelled extensively and shrewdly chose every opportunity to advance the cause of a full Veterinary Division for South Africa with the two-fold approach of research and training.

As he grew in stature Theiler unashamedly used his reputation and any honours bestowed on him to force action in any direction which he saw fit. Recognition, initially slow, was inevitable for Theiler worked hard and diligently shared his knowledge with the world.

Arnold Theiler was a holist believing in the ecological basis of animal diseases and in an interdisciplinary approach to solving the problems associated with them. He emphasized research and continually sought advice and even formal training in fields new to him but where instinct and/or scientific progress indicated that the answers to many veterinary problems lay.

Theiler at last realized his interdisciplinary approach when a Cornell-educated entomologist, C.B. Simpson, arrived in Pretoria on 26 August 1903 for duty at Daspoort. In the following years Theiler's interdisciplinary team expanded. While C.B. Simpson continued working on ticks, flies, mosquitoes and other probable disease vectors, J. Burtt-Davy (botanist) worked on plants toxic to cattle and H. Ingle (chemist) studied soils and bones for chemical deficiencies causing osteoporosis. In a paper read on Theiler's behalf in Johannesburg at the 75th meeting of the British Association for the Advancement of Science on 29 August 1905, Theiler confidently asserted that the time was near when South Africa would not be devastated by stock diseases anymore. Significantly, however, he added that *that point would be won not only by the advance of veterinary science, but by science in general*, i.e. by a multidisciplinary approach.

The interdisciplinary approach flourished under Theiler, yet Theiler himself still felt inadequate and a great need to learn more about zoology, botany and chemistry to aid him in his quest for veterinary solutions. Thus in June 1909 Theiler left to study zoology, physics and chemistry for a whole semester at the University of Zurich, aiming to obtain a doctorate in philosophy, specializing in helminthology. In 1911 Theiler again left for more interdisciplinary study at Basle in Switzerland. He repeated the process in 1920 at the University of Berne.

Thus Theiler prepared the early table well for the development and advancement of veterinary science. Sadly his brilliance ultimately was so much in demand that his research productivity suffered in the process. Yet throughout his life Theiler firmly believed in a multidisciplinary approach with frequent interchange between veterinarians and other scientists, especially biologists. He also clearly separated the task of veterinarians into curers (veterinary surgeons) and researchers. Overall his personal approach was one of a practical research scientist employing exhaustive methods of all related disciplines to establish reliable conclusions. Below it will be attempted to measure the more recent veterinary successes and approaches to Theiler's example.

THE POST-THEILER ERA

The current status of our knowledge of animal diseases is beyond the scope of this paper. However, the overall

approach adopted to study and combat some well-known diseases will give an insight into the current veterinary approach.

Trypanosomiasis: The Tsetse Story

Many species of African game and other vertebrates act as reservoir hosts of pathogenic trypanosomes, but show few if any signs of these infections, and records of trypanosomiasis in game are limited. Carnivores, especially lions, have an unexpectedly high infection rate but they probably get trypanosomiasis mainly by feeding on infected meat, the parasite entering the system through small wounds in the buccal mucosa, nose or muzzle. Some carnivores could also possibly infect each other through social grooming of wounds and sores[22].

Dark-skinned species of game are preferentially bitten by the tsetse fly while the paler antelope and zebra are rarely attacked. In addition the habits and habitat preference of both the tsetse fly and the game species present in an area largely determine the chances of infection. Yet, most studies of trypanosome infections to date took little or no note of the ecology of the trypanosomes' host or vector, including the fact that the tsetse fly is severely hampered in its movements by treeless open areas of only a few hundred metres wide[10]. Dräger and Mehlitz (1978[8] were two of the first authors to relate trypanosome infection to the population dynamics of the buffalo, and to compare infection data for high and low-density tsetse fly areas[22].

The rinderpest panzootic, which severely reduced especially those game most susceptible to trypanosomiasis in many parts of Africa, revealed that tsetse fly belts also disappeared or largely retracted in such areas, allowing livestock into formerly restricted areas. This initially led to many campaigns of large-scale destruction of game to curb the tsetse fly in the early part of this century[22]. However, subsequent studies have clearly shown that most of these campaigns were expensive, inefficient and wasteful[5 10 22]. Game and stock fences and tsetse fly control posts have also been used in Botswana, Zambia and Zimbabwe in conjunction with game removals in corridors between game and livestock areas. Again the efficiency and economics of such an operation is questionable[22].

Currently insecticides are used widely, but often indiscriminately, in the control of tsetse fly populations. Insecticides are effective if used properly and according to a strict code of conduct but their injudicious use can cause serious health and environmental problems[12 22 23 36]. Even hand-spraying of dieldrin into tsetse resting sites is undesirable[36].

Yet, the effects of agricultural mismanagement in and use of areas cleared of tsetse (and game) often eclipses all the detrimental effects of the initial control methods. Severe habitat degradation due to livestock overpopulation following the introduction of livestock into naturally marginal grazing areas poses serious health and environmental threats[12]. Furthermore, members of the *Glossina palpalis* group can exist easily in small, isolated areas of forest in peridomestic habitats in the more humid areas and in areas of intensive cultivation. Where pockets of tsetse flies remain, overgrazing and the resultant bush formation also soon create ideal habitat for the resurgence of the tsetse fly.

Although residue analyses of pesticide levels in animal tissues after tsetse control have been done, reliable, controlled studies involving base-line analyses seldom exist. Tsetse control is still too frequently regarded as an end in itself, but the whole spectrum of ecological implications should be evaluated before any action is taken. Bush-clearing as a control method in large areas of Africa has also substantially increased the pressure on the remaining natural forests as sources of firewood, where wood-use volumes far outstrip the rate of wood regeneration[22].

Alternative methods of control can be found. In parts of Africa the use of odour-scented baits attracting tsetse flies primarily by smell to toxic traps are proving increasingly effective. The aromas currently used as bait are chemicals of three breath components of an ox, i.e. carbon dioxide, acetone and octenol[12].

The complexity of the tsetse fly problem requires intensive interdisciplinary research, much of which is still lacking, and detailed planning which includes environmental and conservation considerations. The clear web of interrelationships between trypanosomes and environmental factors, and the regulating factors and population ecology of the tsetse fly, the wildlife hosts of trypanosomes and the combined implications for conservation and sound livestock production in Africa provides a rich field for future interdisciplinary study.

Rinderpest

Rinderpest is known to have occurred in the Nile Valley of Egypt from 1841-1842 where it was contained. However, when it was reintroduced into Somalia or Abyssinia in 1889, probably following the importation of Zebu cattle from India for the Italian armies in north Africa, it caused a virgin-ground panzootic in Africa from 1889-1898. It spread rapidly along the trade routes, reaching the Cape some 5 000 km from its source by December 1896. Its effects were disastrous and it killed some 5,3 million cattle in southern Africa alone[29].

In the case of rinderpest, wildlife was less of the culprit than the victim as game is considered as less of a menace as a source of infection of rinderpest for cattle than some other major diseases[16]. Rinderpest was eliminated in southern Africa but remained established in the Sub-Saharan regions of Africa where it caused periodic waves of infection. In the early 1930's a focus of permanent, mild infection was established in the Serengeti. Clinically apparent rinderpest last occurred in Serengeti buffalo and blue wildebeest populations in 1960-61, while subclinical and restricted infections last positively occurred in 1965-66. Although some unsubstantiated later reports of rinderpest exist for Tanzania[30], no serological evidence of the virus has been found since 1966[29].

Rinderpest had a profound effect on several major wildlife species in Africa, and is considered by some authors to have been the major regulating factor of buffaloes in the Serengeti from 1889-1960's[29]. These effects have obvious conservation and wildlife management implications and the contribution of veterinarians to help the recovery of the great Serengeti herds by the elimination of rinderpest must be acknowledged.

Some conflicting attitudes still persist among some conservationists who claim that without cattle the threat of a new rinderpest panzootic would not exist, while some veterinarians believe that somewhere there may be a lurking pocket of rinderpest infection in the game

herds of Africa[29 37]. The truth, as often is the case, is probably somewhere in between.

The present situation is still dangerous as there are now few rinderpest-immune herds of wildlife left, while in the Serengeti populations of herbivores of high susceptibility such as the buffalo, blue wildebeest, eland and warthog are present in greater numbers than at any time since the panzootic. Should these and other herds be reinfected by the illicit movement of cattle from the north it would be futile to question the regulatory tendency of rinderpest as has been done by some ecologists[10]. Vigorous and rapid interdisciplinary action will be necessary, with qualified staff of all disciplines involved formed into research teams to study and combat the disease[29].

Rabies

Rabies is another viral disease of great concern to man and wildlife. Rabies is spread throughout the world, except Australia and many islands, but the epidemiological pattern varies from region to region due to the dominant role of the various main vector species acting through their density, which in turn is determined largely by their habitat quality[31].

Rabies was first recorded in South Africa in 1892[14] but canine rabies was known as early as 2300 B.C. in Babylon[31]. In Africa the principal hosts of rabies are the jackals[31] but in recent years in South Africa the genus *Cynictis* was involved in 69 per cent of all rabies cases reported for wild animals[14], with a rather specialized form of horizontal spread of rabies in the kudu in South West Africa[2]. The complex and dynamic nature of rabies appears to have contributed to various changes in the epidemiological pattern of rabies in South Africa[14].

Rabies does not easily switch from one principal host to another, even under ecological conditions which would favour this event. Domestic and wild ungulates, although susceptible to rabies, are dead-end hosts and do not transmit the disease[31]. Host species predominance in a given area is a factor of habitat quality, and host species with a high degree of habitat specificity are important as sources of rabies only over a small part of their distribution range while hosts with wide habitat tolerances play a more significant role as sources of rabies over a larger area[14].

The main factors responsible for control over the spread and incidence of rabies are largely linked with host density, movements and social behaviour[31]. Control methods of hosts should therefore involve ecological and behavioural studies of both healthy and infected animals. Thus Zumpt[38] and Gummow & Turner[14] have illustrated that the stress of eviction of subadult yellow mongooses, winter food shortages and other stresses may trigger off clinical rabies in that host. Barnard and Hassel[2] in turn demonstrated how comparisons between the movements, feeding habits and the degree of physical contact between normal and rabies-infected kudus helped explain the pattern of that form of rabies outbreak. Barnard[1] on the other hand, briefly discussed the behaviour (literature-based) of healthy wild animals to yield reasons why the disease is contracted or spread.

It is still popular today to control rabies merely by reducing the number of actual or potential hosts in a given area, despite evidence that most of the hosts involved have social density regulatory factors (e.g. territoriality) and that a reduction in population pressure will rapidly be remedied[31] *inter alia* through a stimulus in reproduction, increased survival rates and even the re-invasion of areas where the host was totally exterminated[38].

In the process of host population reduction both the ecology of the host and the ecological consequences of the technique of control are often overlooked, while ethical aspects are often totally ignored[7 25 27 33]. Current attitudes and techniques need constant re-evaluation. Alternative control methods of the host involved[6] and oral immunization of some hosts[31] offer distinct improvements over existing methods of control in South Africa[27] while still taking the *bona fide* needs of the farming community[9] into consideration.

Above all the whole problem of rabies in South Africa is a prime candidate for a multidisciplinary research approach to solve the many pertinent questions surrounding the epidemiology of this important disease[4 31 38].

CONCLUSIONS

The above three disease examples clearly show how correct Theiler's multidisciplinary approach to veterinary problems was, and accentuate the role of environmental conditions in these and other animal diseases. With the increasing development of game ranching, sometimes coupled with livestock production, with increased pressure on existing game reserves and the fencing of most natural areas the potential for new ecological relationships increases. In this regard the veterinarian will increasingly have to rely on an interdisciplinary approach to help him in his task[13 17 18 19 26 28 32].

Increasingly also, poor agricultural practices and the injudicious use of pesticides and other agricultural chemicals can have wide-ranging effects on the health of man, his livestock and on wild animals and the ecosystem as a whole[3 19 20 35]. Even the social behaviour[11] and taxonomy[21] of vectors and hosts may be important in the study of animal diseases, while diseases and disorders of the digestive system can have a profound effect on the health of an animal[34]. Anthropological, sociological and other historic events are also at times linked directly or indirectly with the diseases of man and his animals (U. de V. Pienaar 1987 Chief Director, National Parks Board, P.O. Box 787, 0001 Pretoria, personal communication).

A PHILOSOPHY RENEWED

In the last twenty years or so the growing conservation and economic importance of wild animals is increasingly necessitating that they receive as much attention as domestic livestock in the past. Since the Theiler era in South Africa the treatment and prevention of diseases in livestock have been developed to a level of which the veterinary profession can be justifiably proud. Yet, based on the facts examined above it is clear that relatively little progress has been made in understanding the full nature of all animal diseases, particularly their ecological causes and consequences. Even basic descriptions of the anatomy, morphology, physiology and pathology, and base-line values of the clinical pathology of many wildlife species are still lacking.

At a time when the veterinarian requires increasingly wider scopes of knowledge, the basic sciences comple-

ment in his training also has been reduced to a single year of physics, chemistry and biology recently. Long gone is the teaching of ecology as a full veterinary subject as was done by Mogg in the early days. It would appear that the veterinary profession recently has leaned too heavily in the direction of curing diseases while neglecting to study their causes and effects. In the process many veterinarians have also sadly neglected their role as researchers, and have thus retarded the development of a vital part of their profession.

There are signs though that an increasing number of veterinarians is becoming aware of the need to practise interdisciplinary research. Now is the time that veterinarians should sit down and rethink their entire role and training, since as veterinarians they are no longer only dealing with cattle, but increasingly with a whole array of biological organisms.

The choice is in the hands of the veterinary profession. Judgement is in the hands of posterity. I believe that there can be but one answer: the veterinarian is a biologist, and he must accept this fact and return to the biological fold to make full use of the knowledge and expertise lodged there to find meaningful solutions to the problems with which he deals in animal diseases, and the effects of his own actions. In many ways veterinarians can also assist other biologists to do more meaningful research. In doing so the veterinarian will not embark on a new course but will simply renew an old philosophy which originally helped to place South African veterinary science at the forefront of the world of science.

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POISONING OF WILDLIFE IN SOUTHERN AFRICA*

P.A. BASSON**

ABSTRACT: Basson P.A. **Poisoning of wildlife in Southern Africa.** *Journal of the South African Veterinary Association* (1987) **58** No. 4, 219-228 (En). P.O. Box 81, Grootfontein 9000, South West Africa/Namibia.

Wildlife can be poisoned by both plants and chemicals. The co-evolution of wildlife and toxic plants has resulted in an increased resistance to these substances as compared with domestic animals. Both groups of animals are however susceptible to chemical poisons. The results of experimental poisonings with *Dichapetalum cymosum, Urginea sanguinea, Senecio retrorsus, Nicotiana glauca* and prussic acid are discussed. The effect of poisoning of wildlife with *Crotalaria* spp. *Geigeria spp, Lantana camara,* chlorinated hydrocarbons, organophosphates, carbamates, strychnine, heavy metals and other plants and chemicals is reviewed.

Key words: wildlife, poisoning

INTRODUCTION

Over the past two decades game farming has become increasingly popular as a means of meat production, for the provision of animals either for sale or trophy hunting, and for recreation purposes. The reintroduction of animals and their translocation to strange habitats has become a frequent operation, sometimes with unnecessary or unforeseen problems and losses. These developments occurred in an age of ever increasing environmental pollution which is threatening life in various forms. All these factors highlighted the effects of environmental pollution on wildlife and in particular have focussed our attention on wildlife poisoning.

As with their domestic counterparts, wild animals can be poisoned either by natural toxicants in plants or by unnatural substances such as pesticides, pollutants (including heavy metals) and other chemicals. Also, certain animals are known to be more susceptible to the side-effects of some drugs. However, for the purpose of this communication, pharmaceutically induced poisoning will be excluded.

POISONING BY PLANTS

The co-evolution of wildlife with their natural enemies, be they other animals, micro-organisms, parasites or toxic plants, usually leads to the improvement of specific defence mechanisms up to a point where co-existence is possible. In such a well-balanced ecosystem, local populations are being kept hardy, virile and well-adapted to their surroundings by natural selection. Those that are most susceptible to diseases, parasites and poisonous plants are regularly removed by predators or die before they can reproduce. The relative lack of information in the literature on subjects such as plant poisoning in wildlife, especially in Southern Africa, is therefore not surprising. Cases of such poisoning which have been reported include the following (Table 1):

Steyn described stiffness among some free-living antelopes during years of abundance of *Crotalaria* spp[32]. The numbers and species concerned were not specified. Lewis, Wilson and Hill (1973)[22] recorded three cases of chronic laminitis in the common duiker *(Sylvicapra grimmia)* in Zimbabwe due to *C. barkae.* Grosskopf reported cases of *Geigeria* poisoning in springbok *(Antidorcas marsupialis)*[14]. In one instance they were seemingly so weak that they could be caught by man on foot. Confirmation at autopsy or by histopathological examination was not mentioned. Grosskopf also described cases of bloat in waterbuck *(Kobus ellipsiprymnus)* that died in lucerne fields on the Limpopo river[13]. Various other abundant species such as kudu *(Tragelaphus strepsiceros)*, impala *(Aepyceros melampus)*, steenbok *(Raphicerus campestris)* and bushbuck *(Tragelaphus scriptus)* which grazed in the same fields were never seen to be affected. Myocardial fibrosis was observed in springbok near Potchefstroom that dropped dead while running (Prozesky L, Veterinary Research Institute (VRI), Onderstepoort, unpublished data). These antelopes were confined to overgrazed camps with abundant gousiekte bossie *(Pachystigma pygmaeum).* It seemed evident that the springbok were dying of "gousiekte" under these circumstances. Suspected cases of gifblaar *(Dichapetalum cymosum)* poisoning in kudu occurred under similar circumstances of confinement and overutilization of camps (Naude TW, Faculty of Veterinary Science (FVS), University of Pretoria, unpublished data). Sudden deaths occurred but no carcasses were presented for autopsy. *Lantana camara,* a well-known declared exogenous weed, was suspected of being responsible for photosensitization in gemsbok *(Oryx gazella)* (Coetzer JAW, VRI, Onderstepoort, unpublished data), but again no carcasses were obtained for confirmation.

In a series of experiments in SWA/Namibia since 1974, some of the most toxic plants of Southern Africa were used as models to determine the intake and susceptibility of some indigenous wild ungulates[4 5 26]. Eland *(Taurotragus oryx),* kudu, springbok, gemsbok and one giraffe *(Giraffa camelopardalis)* were used in a comparative study with domestic ruminants. In order to minimise or exclude the effect of stress which could lead to capture myopathy, shock and injuries, the majority of animals were caught and subjected to a period of adaptation which lasted for several months or years. In fact, many of the experimental animals, especially eland, were born in captivity. The antelopes were kept in small camps on natural bushveld and their diets supplemented with horse cubes or lucerne hay. Usually, before each dosing trial, both antelopes and domestic

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** P.O. Box 81, 9000 Grootfontein, South West Africa/Namibia

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Table 1: Wildlife poisoning by plants

Animal	Poison	Area	Malnutrition	Mortality	Clinical Signs	Pathology	Confirmed	Reference
Springbok Eland (R + +) Kudu (R + +) Gemsbok	Gifblaar *(Dichapetalum cymosum)*	South West Africa/Namibia		Some	Interrupted recumbency, inappetence, depression, sudden death	Cardiomyopathy Hepatosis Nephrosis Gall bladder oedema	Experimental	5
Kudu	Gifblaar	Farm (C) Transvaal	Yes	Few	Sudden death		No	Naudé T.W. pers. comm. 1987
Springbok (R + +) Eland(R +) Kudu (R +) Gemsbok(?)	Slangkop *(Urginea sanguinea)*	South West Africa/Namibia		Some	Transient diarrhoea, inappetence, listlessness, sudden death	Cardiomyopathy Hepatosis Nephrosis Enteritis	Experimental	5
Springbok Eland(R +) Kudu	*Senecio retrorsus*	South West Africa/Namibia		Some	Wobbly, pushing syndrome, depression	Necrosis and haemorrhage of liver Pulmonary oedema	Experimental	5
Giraffe Springbok Eland (R + +) Kudu (R + +)	Prussic acid	South West Africa/Namibia			Eland: ataxia, imbalance, drowsiness, sedation, sternal recumbancy. Mild spasms rare.		Experimental	5
Gemsbok	*Nicotiana glauca*	South West Africa/Namibia	Yes	Yes	Hypersensitivity Spasms Paresis	 Myocardial, hepatic degeneration	No Experimental	Ebedes H. 1974 unpublished data Basson *et al* 1974 *unpublished* data
Springbok	*Pachystigma pygmaeum*	Farm (C) Potchefstroom	Yes	Numbers unknown	Dropped dead while running	Myocardial fibrosis (sub-endocardial region)		Prozesky L. pers. comm. 1987
Various antelopes	*Crotalaria* spp				"Langklou", stiffness			32
Common duiker	*Crotalaria barkae*	Zimbabwe		3 cases	Elongated hoofs			22,
Springbok	*Geigeria* sp			Not recorded	Weakness Easily caught		No	14
Blou duiker	*Cestrum laevigatum*							37
Gemsbok	Lantana?				Photosensitization		No	Coetzer J.A.W. pers comm. 1987
Waterbuck	Lucerne	Transvaal, RSA		Few	Bloat		Yes	13
Various antelopes (Kudu)	Tannins	Confinement	Yes				Experimental	35, 36

R +, R + + = Increasing degrees of resistance (R)
? = Questionable
C = Camps

animals were starved and withheld from water for approximately 18 - 24 hours. Each animal's mass was accurately determined and, with the exception of a few antelopes, restraint in a crush was preferred to chemical immobilisation in order to exclude possible interactions between immobilising agents and the toxic principles concerned. Dosing was done by stomach tube with the aid of a suction pump. The experiments were conducted in the Mangetti area approximately 150km north of Grootfontein and continued for six years mainly because of the limited number of available antelopes at a specific time. A few antelopes were experimented on in the Etosha National Park and a few sheep at Onderstepoort. The following plants were used in these experiments:

Gifblaar (D. cymosum): This plant contains monofluoroacetate (MFA)[32] which is converted in the body to fluorocitrate, a potent cardiac poison which

blocks the Krebs cycle[27]. Voluntary intake of the plant was determined by offering sublethal doses of specific quantities of counted leaves to starved antelopes. This procedure was followed periodically for more than one year. Initially a remarkable avoidance was noticeable in eland but later, prolonged experiments indicated that in captivity these antelopes will ingest varying quantities of gifblaar leaves. Some individuals were more cautious than others. Kudu, however, were much more wary and their intake was both exceptional and limited. It is noteworthy that the later experiments were conducted after the animals had been in captivity for a few years. By this time some could have lacked parental education and others could have lost some of their avoidance behaviour.

In the dosing trials, because of the prolonged experimental period and the consequent variation of MFA content in the various batches — of which only some 50 per cent were able to be assayed — a comparison between animals was only valid within the same period or whenever the MFA content could be calculated. The total numbers of animals used for the dosing trials were 32 goats, ten eland, four kudu, six springbok and two gemsbok (Table 2). The *c.*LD100 for domestic goats was found to be equivalent to 1,01 - 1,6 mg/kg. Springbok and gemsbok were as susceptible as goats but confirmation is needed for the latter in order to eliminate a possible interaction between MFA and the immobilising drugs which had to be used to make this species more tractable.

Table 2: Mortalities caused by *D. cymosum*

Dosage c.MFA mg/kg	No. of deaths/No. dosed				
	Goats	Springbok	Gemsbok	Kudu	Eland
0,4-0,8	0/4	1/1	–	–	–
1,0-1,1	3/4	1/1	–	–	0/1
1,6	1/1	–	–	–	0/1
2,2-3,5	9/9	4/4	2/2	0/2	0/4
4,5-5,9	5/5	–	–	0/1	0/1
6-8	1/1	–	–	1/1	1/2

The most important lesions were degeneration of the myocardium and liver. Myocardial lesions were usually more frequently encountered in the papillary muscles and subendocardially. Replacement fibrosis occurred in animals that survived for two or more days[3].

Slangkop (Urginea sanguinea): This plant, which contains cardiac glycosides[32], is widespread throughout the country and causes serious losses in domestic stock during early spring. Either the inflorescence or bulbs during various growth stages were used as a blend in tap water. A total of 39 goats, 14 eland, nine kudu, six springbok and four gemsbok were used in the trials (Table 3). The plant material did not always provoke identical reactions and the impression was gained that the early inflorescence and bulbs of the seed-bearing stages were more toxic than the other stages. The *c.*LD50 and *c.*LD100 for goats were 3 - 4 g/kg and 4 - 5 g/kg respectively. A transient diarrhoea and inappetance were noticed in eland and kudu at 5 g/kg but they only started dying at 6 g/kg. One eland died at 7 g/kg without showing any diarrhoea. Springbok showed no symptoms even at their maximum dose of 6 g/kg. The gemsbok that died (at 2,5 - 4 g/kg) showed complications of shock and capture myopathy. One of these gemsbok (4 g/kg) had a diarrhoea. However, another gemsbok which received a higher dosage of 5 g/kg did not develop any symptoms.

It was evident that the resistant antelopes have either a superior ruminal degradation or superior systemic detoxification or both.

The most important lesions caused by slangkop were confined to the heart and could not be distinguished from those caused by gifblaar[3]. Subacute to chronic lesions of replacement fibrosis were encountered in animals that survived for more than six days[3,5].

Senecio retrorsus: Finely ground dried material obtained from the Eastern Cape was dosed to eight sheep, six goats and six eland (both males and females), and to one kudu heifer and one springbok ram (Table 4). All five sheep given 5 g/kg died but none of the goats even at 8 g/kg succumbed. One eland heifer and one young eland bull were given 8 g/kg. The heifer became wobbly five days after treatment, showed the pushing syndrome and died on the same day. Both kudu (8,7 g/kg) and springbok (7 g/kg) died on day three post dosing. Lesions typical of *Senecio* poisoning were found both macro- and microscopically[17]. It was therefore established that whereas the *c.*LD50 for sheep was between 2,5 - 5 g/kg (Table 4), the MLD for goats was more than 8 g/kg and that of eland about 8 g/kg.

Senecio spp. are well-known for their production of pyrrolizidine alkaloids. In general some ruminal degradation of alkaloids can occur in animals. Pyrrolizidine alkaloids, which as such are nontoxic, are, after absorption, converted in the liver by microsomal enzymes to pyrroles which are hepatotoxic[12]. Rapid pyrrole production is therefore also correlated with increased susceptibility. It is of interest to note that Dean and Winward (1974) (quoted by Fowler[12]) have also reported that black-tailed deer *(Odocoileus hemionus columbianus)* can consume large quantities of *Senecio* without ill-effects.

Prussic acid (HCN): Potassium cyanide powder was used as a source of HCN because of the practical problems in procuring fresh plant material with adequate cyanogenetic glycosides. Thirteen cattle, two sheep, 28 goats, 20 eland, two kudu, five springbok and one giraffe were given dosages of calculated HCN ranging from 2 - 7 mg/kg. For fear of losing some of the limited number of animals, an antidote consisting of an aqueous solution of 25% sodium thiosulphate and 2,5% sodium nitrite was invariably given by intravenous route (40 ml/200 kg) during severe convulsive stages or sometimes also in animals with milder clinical signs (Table 5). The onset of clinical signs was slower in those animals such as the eland which were less susceptible. It varied from 1 - 6 minutes (*ca.*2,6) in cattle at 2 - 4 mg/kg, 2 - 15 minutes (*ca.* 4,2) in goats at 3,7 - 7 mg/kg and 4 - 18 minutes (*ca.* 8,4) at 4 - 7 mg/kg in eland. The clinical signs noticed included lip-licking, chewing, salivation, foaming at the mouth, drowsiness, ataxia, imbalance, respiratory distress, recumbency, severe convulsions or intermittent spasms. Severe spasms were commonly seen in cattle at 3 mg/kg and in goats at 5 - 7 mg/kg but only once in one eland at 6 mg/kg. Eland were more inclined to show ataxia, imbalance, drowsiness, sedation and sternal recumbency. Recovery

Table 3: Mortalities caused by *U. sanguinea*

Growth stage	Parts dosed	Dosage g/kg	No. of deaths/No. dosed				
			Goats	Eland	Kudu	Springbok	Gemsbok
Early flowering	Bulbs	2	0/5	–	–	–	–
Early flowering	Bulbs	2,5	–	0/1	0/1	–	1/1?
Late flowering	Bulbs	3	3/8	–	–	0/3	–
Early flowering	Inflorescence	3	0/3	–	–	–	–
Early flowering	Inflorescence	4	7/8	0/3	0/3	0/1	2/2?
50% Flowering	Bulbs	4	3/6	0/1	0/1	–	–
50% Flowering	Bulbs	5	5/5	0/5	0/1	–	–
Early flowering	Inflorescence	5	–	–	0/1	–	0/1
Seed-bearing	Bulbs	6	4/4	2/3	2/2	0/2	–
Seed-bearing	Bulbs	7	–	1/1	–	–	–

? Complications : Shock, capture myopathy.

Table 4: Mortalities caused by experimental *S. retrorsus* poisoning

Dosage mg/kg	No. of deaths/No. dosed				
	Sheep	Goats	Eland	Kudu	Springbok
2,5	0/1	–	–	–	–
5	5/5	0/2	–	–	–
6	2/2	0/1	–	–	–
7	–	0/1	0/4	–	1/1
8	–	0/2	1/2	–	–
8,7	–	–	–	1/1	–

with high dosages, however, sometimes took an hour or more. One eland died two hours after a dosage of 7 mg/kg. Three eland (at 6 - 7 mg/kg), which received the antidote 19-245 minutes after dosing, took longer than the other animals to recover completely (45 - 50 minutes). Results from the other animals used are rather inconclusive because of their limited numbers, but indications were obtained that kudu and springbok are less susceptible than cattle. Antidotes were probably administered too soon in both springbok and giraffe because losses were feared in their very limited numbers. The HCN levels in the blood showed some correlation with the duration of clinical signs but none with the dosage or severity of clinical signs.

Plants which may cause HCN poisoning are known to contain cyanogenetic glycosides which, by a process of hydrolysis under the action of ruminal organisms, enzymes liberated from macerated plant material and suitable pH, liberate cyanide[42]. Free HCN, however, can also occur in plants that are stunted, wilted or damaged by frost, hail or trampling[32]. It is also of interest to note that some herbicides could increase toxicants such as cyanogenetic glycosides in plants[42].

Resistance to cyanogenetic glycosides could imply either a very rapid systemic detoxification by conversion to thiocyanate or a beneficial ruminal process whereby microbial hydrolysis is retarded or by both. As potassium cyanide instead of plant material with cyanogenetic glycosides was used, the results need not be a true reflection of the susceptibility of antelopes to the natural compounds. It can merely be concluded that some antelopes, such as eland, have a more efficient systemic detoxification process than domestic ruminants.

Wild tobacco (Nicotiana glauca): This commonly occurring introduced poisonous plant[32] was suspected of causing mortalities in gemsbok in the western regions of SWA/Namibia (Ebedes H, National Zoological Gardens, Pretoria, 1974, unpublished data). Consequently plant material was collected and a blend prepared with tap water and dosed to two gemsbok (Basson P A, Ebedes H and Norval A G, 1974, unpublished data). Muscular twitching, hypersensitivity and spasms were noticed approximately 5 - 10 minutes after dosing. Respiratory and cardiac arrest occurred within 30 minutes in an antelope that received 5 g/kg of the plant material. The other gemsbok at 2 g/kg developed posterior paresis and torticollis with sporadic spasms. It died approximately 17 hours after dosing.

The most important pathological findings were degenerative changes in the myocardium and liver, glial swelling and mild status spongiosus of the brain, haemorrhages and oedema of the urinary bladder, abomasal oedema, and kariorrhexis in the spleen and lymph nodes. Disseminated intravascular coagulopathy in the myocardium associated with necrotic fibres was a striking feature in the gemsbok that had survived for 17 hours.

Other secondary plant compounds: Stahl (1888) (quoted by Fowler[12]) introduced the concept that plants could produce compounds, other than direct toxicants, which serve as defence mechanisms against herbivores. Essential oils as well as other bacteriostatic compounds which are highly unpalatable and which interfere with digestion were given as examples[12]. In South Africa, Van Hoven[35 36] proved that bushes can increase their production of unpalatable tannins in response to overutilisation. The tannins react with digestive enzymes and proteins rendering them either inactive or indigestable and in this manner cause indigestion and star-

Table 5: Number of animals experimentally dosed with potassium cyanide powder which showed spasms and number which were given antidotes (AD)

Dosage mg/kg	No. with spasms/No. dosed (No. given AD)						
	Cattle	Sheep	Goats	Springbok	Giraffe	Kudu	Eland
2-2,5	0/1	0/1					
2,8	1/2(1)						
3-3,7	6/9(6)		0/7(2)	0/2	0/1(1)	0/1	0/3
4	1/1(1)	1/1(1)	1/7(4)	1/2(2)		0/1	0/2
4,8			0/1(1)				
5			5/6(5)	0/1(1)			0/6
6			3/6(3)				1/7(2)
7			2/2(2)				0/2(1)

vation. Confinement and overstocking of browsers and browser-grazers could therefore lead to starvation and death in spite of an adequate food supply. Although this need not necessarily be regarded as direct poisoning *per se*, such chronic effects and indirect ways of affecting the well-being of herbivores need more emphasis.

POISONING BY MYCOTOXINS

Only one record was obtained where two cases of suspected ergotism were diagnosed in impala in the Midmar Game Camp, Natal (Lewis AR, Natal Parks Board, 1976, unpublished data). However, only *Claviceps paspali* and not *C. purpurea* was identified from very heavily parasitised *Paspalum dilatatum*, a very common species in the Park.

POISONING BY BACTERIAL TOXINS

Under anaerobic conditions *Clostridium botulinum* type C can produce toxins in carcass and other organic material in water, which can poison waterbirds such as wild ducks and wild geese[16,34]. The toxins, which are probably contained within the bacteria, cause paralysis and polydipsia in birds. They usually lie with outstretched necks and legs and drooping wings. Paralysis of the nictitating membranes is apparently a very characteristic feature. The toxin can also cause malfunctioning of the supraorbital gland because of its blocking action on the facial nerve. The gland has an extrarenal excretory capacity which, with a small dose of toxin, can easily be overloaded. In this way death can be precipitated by excessive salt. Intravenous treatment with antiserum seems to be very effective[16].

POISONING BY PESTICIDES, POLLUTANTS AND OTHER CHEMICALS

Poisoning of wild animals by unnatural substances (Table 6) is more important than poisoning by natural toxicants contained in plants because animals usually lack resistance against these substances. Knowing that some animals have an innate resistance to MFA, the commercial product 1080 is one of the exceptions in this respect.

Chlorinated hydrocarbons (CHC) are well-known, persistent, poorly degradable pesticides whose residues accumulate in an ecosystem[6]. Their effects are particularly noticeable in birds of prey that are at the top of a food chain. The worldwide decline in raptor populations due to direct mortality or indirect effects such as increased fragility of eggs, terminal death of embryos and a delay in onset of breeding due especially to DDE, the principal metabolic product of DDT, is well-documented[25,29].

In South Africa, Wiese and Basson[39], Wiese *et al*[41] and Basson[2] studied the effects of certain levels of dieldrin and photodieldrin on birds such as guinea-fowl *(Numida meleagris)* and established that they are more susceptible to these compounds than domestic fowl. Wiese *et al*[40] also described very high mortality rates in blesbok *(Damaliscus dorcas)* and springbok on fields sprayed with dieldrin for termites. Typical acute clinical signs were described as well as a dumb syndrome characterized by stupor, lack of fear and apparent blindness. Myocardial and muscular lesions as well as glial swelling and brain oedema were reported. It was concluded that these antelopes are more susceptible to these compounds than any vertebrates reported on previously (Table 8). Rams were also more susceptible than ewes. Clinical signs described in birds with CHC poisoning were inappetance, tremors, incoordination, spreading of the rectrices, spasms, salivation and muscular dystrophy[25,39]. Dieldrin proved to be more powerful than DDT in delaying the onset of breeding in birds[29]. Such a delay consequently reduces the chances of reproductive success. Records of CHC poisoning, especially in raptors, occur worldwide, including many in Southern Africa[6,16,30,33,38].

The polychlorinated biphenyls (PCB), which are widely used in industry as plasticizers and in paints and lubricants, resemble DDT in molecular structure and produce similar physiological actions in animals[29]. They can also cause thinning of egg shells but, as with dieldrin, are more effective in delaying the onset of breeding. The PCBs are also released when plastic materials are burned and are consequently widely distributed over the earth.

Organophosphors (OP), although more easily degradable than the CHCs, can cause acute deaths and high mortalities in birds and mammals[20]. In sheep-farming areas, some farmers use concentrated insecticides such as diazinon on carcasses in order to control blowflies. By doing so many scavenging birds and mammals can be killed. Periodic mortalities of grain-eating wild birds by different OP pesticides in various parts of Southern Africa have been encountered from time to

Table 6: Wildlife poisoning by pesticides

Animal	Poison	Area	Mortality	Clinical Signs	Pathology	Confirmed	Reference
Blesbok Springbok	Chlorinated hydrocarbons (Dieldrin Photo-dieldrin)	Republic of South Africa	High (100%)	Violent form: excitement, spasms Dumb form: Fearlessness blindness	Muscular and myocardial degeneration Brain swelling Degeneration of liver and kidney	Yes	40
Birds	Chlorinated hydrocarbons	Zimbabwe				Yes	38
Guinea-fowl	Dieldrin	Transvaal		Inappetence, incoordination, spreading of rectrices, seizures, salivation. Eggs: low chick survival		Experimental	39, 41
Herons, raptors	Chlorinated hydrocarbons	Transvaal					30
Birds	Dieldrin, Photo-dieldrin					Experimental	2
Guinea-fowl	Organophosphors	South West Africa	Many			Yes	Grant R, Basson P.A. Unpublished data
Spurwing	Organophosphors ("Monocrotophos")	Natal	31			Yes	Bath G 1987 Pers. comm.
Egyptian goose, guinea-fowl, dove, crowned crane, francolin, redbilled teal, sacred ibis	Organophosphors ("Monocrotophos")	Natal	2 7 +			Yes	Bath G 1987 Pers. comm.
Coot	Organophosphors ("Triazophos")	Natal	1			Yes	Bath G 1987 Pers. comm.
Vultures	Organophosphors ("Parathion")	Natal	2			Yes	Bath G 1987 Pers. comm.
Geese, Guinea-fowl	Organophosphors ("Diazinon")	Natal	+			Yes	Bath G 1987 Pers. comm.
Vultures	Organophosphors ("Dioxathion")	Kruger National Park	c.50				Kellerman T.S. 1987 Pers. comm.
Birds (Blue Crane)	Organophosphors ("Diazinon")	Cape	19				Schneider D.J. 1987 Pers. comm.
Vultures	Organophosphors	Zimbabwe	>60			Yes	21
Birds Elephant Zebra Giraffe Jackal Fish Frogs etc.	Carbamate (Aldicarb) ("Temik")	Transvaal (River)	5 1 2 1 6 Many Many	Found dead near or in water.		Yes	Loock T.J. 1987 Pers. comm.
Vultures	Carbamate (Carbofuran) ("Curaterr")	Caprivi	250			Yes	20
Raptors, scavenging birds, brown hyaena, honey badgers, bat-eared foxes	Strychnine					Yes	20
Vultures	Strychnine	RSA, SWA	>44			Yes	8, 9, 31
Vultures	Strychnine	Botswana	>100			Yes	20

Vultures	Strychnine	Transvaal	37			Yes	20
Wahlbergs eagle	Strychnine	Transvaal	1			Yes	10
White-backed Vulture	Cyanide	Kruger National Park	c.50			Yes	Kellerman T.S. 1987 Unpublished data
Cape vulture Tawny eagle Bateleur eagle Spotted Hyaena Black-backed Jackal	Cyanide	Kruger National Park	1 2 1 1 Several			Yes	Kellerman T.S. 1987 Unpublished data
Vultures, crows (Feeding on primary victims)	Monofluoroacetate (1080, "Blougif")						21
Eland	Arsenic					Yes	Basson P.A. 1972 Unpublished data
Black rhino	Creosote			Weals on skin	Hepatic necrosis Gastric ulcers		43
Grants gazelle Dorcas gazelle	Corrosive weedkiller used to eradicate kikuyu grass	Zoo	Few	Necrobacillosis	Necrosis: Buccal cavity, rumen, liver	Yes	Basson P.A. and Smit J. (1970) Unpublished data
Rock Lobster	Organic pollution (fish factories)	Western Cape	Thousands			Yes	24
Fish	Organic pollution (fish factories)	Western Cape	Many			Yes	24
Birds	*Clostridium botulinum* type C toxin	Transvaal Orange Free State Western Cape		Paralysis, polydipsia, paralysis of nictitating membrane		Yes	16, 34 Schneider D.J. (1987) Pers. comm.
Waterfowl	*Oil*			*Incoordination* Tremors	*Lipid pneumonia*, fatty liver, *nephrosis*, gastro-enteritis	Experimental	15
Impala	Ergot?	Natal	2	Limping	Necrosis lower extremities	No	Lewis A.R. 1976 unpublished data

Table 7: Susceptibility of animals to various poisons

	MFA (mg/kg)	Urginea (g/kg)	HCN (mg/kg)	Senecio (g/kg)
Sheep	0,25-0,5[4]	–	2-2,3[4]	5[6]
Goat	0,3 -0,7[4] 1,0 -1,6[6]	3-4[5]	3-6[3]	8[1]
Cattle	0,25-0,6[4]	–	3[3]	–
Eland	6-8[2]	6[2]	6-7[3]	8[2]
Kudu	6-8[2]	6[2]	4[1]	±8?[2]
Giraffe	–	–	±3?[1]	–
Springbok	±0,84[2]	6[1]	±4-5[3]	±7?[2]
Gemsbok	±0,7[2]	5?[1]	–	–

? Only one animal dosed or complications encountered.
1 = Nil deaths 3 = anticipated MLD 5 = c.LD 50
2 = Onset of death (MLD) 4 = LD 50 6 = c.LD 100

time over many years. Poisoning is carried out by contaminating grain with various OP pesticides, especially parathion, and then putting it out for the birds to eat. This intoxication has been repeatedly confirmed in guinea-fowl, Egyptian geese *(Alopochen aegyptiacus)*, blue cranes *(Anthropoides paradisea)* and other wild birds (Kellerman T S, VRI, Onderstepoort, 1987, pers comm). In a recent incident in the northern Kruger

Table 8: Arbitrary orders of susceptibility to some toxicants (high to low)

Poison	5	4	3	2	1
Dieldrin	Pheasant	Guinea-fowl	Domestic fowl		
Dieldrin and photodieldrin	Blesbok	Springbok	Domestic ruminants		
Gifblaar	Dog	Domestic Ruminants, Springbok			Eland, Kudu
Slangkop	Goats, sheep			Eland, Kudu Gemsbok	Springbok
Senecio	Horses	Cattle, Sheep	Springbok	Eland, Kudu Goats?	Goats
Prussic Acid	Cattle, Sheep	Giraffe	Springbok Goat	Eland, Kudu	

National Park, about 50 vultures were found dead in a deliberate "muti"-killing and the OP dioxathion was found in the possession of the person suspected of causing the incident (Kellerman T S, VRI, Onderstepoort, 1987 pers comm). When the control of redbilled quelea finch *(Quelea quelea)* swarms is deemed necessary, the Department of Agriculture uses OPs by extensive aerial application to the swarms from aircraft. This is done at night when the swarms are sleeping. Initially parathion was used but now fenthion methyl ("Queletox") is used instead. This particular OP has the advantage over parathion that it is more toxic to birds than mammals and is furthermore more easily adsorbed onto organic matter, which inactivates it (Bot J, Plant Protection Research Institute, Pretoria, 1987, pers comm). Fenthion methyl is extensively used as, amongst others, the agricultural remedy "Lebaycid" and as the veterinary ectoparasiticides "Tiguvon" and "Bayopet Spotton".

Carbamates are generally not as poisonous as OPs although some extremely toxic compounds are available and have caused severe mortalities. Carcasses are also treated with carbamates such as carbofuran ("Curaterr") in order to control predators (Naude T W, FVS, Onderstepoort, 1987, pers comm). Ledger[20] quoted one example in the Caprivi where 250 vultures were killed.

The injudicious use of strychnine to control predators is suspected to be a major cause of the near extinction of the Cape vulture *(Gyps coprotheres)* in SWA/Namibia[6, 9]. In one incident in Botswana 100 vultures were killed by strychnine stuffed into a carcass[7]. Clinical signs reported were regurgitation, extension of the necks and aggressive behaviour. Deaths of other scavenging birds and mammals and even birds not known as carrion-eaters, such as the Wahlberg's eagle *(Aquila wahlbergi)*, have also been recorded[10].

"Blougif" (1080) (commercial MFA) is odourless and tasteless, extremely stable, and notorious for poisoning scavengers when placed in carcasses. Vultures or crows feed on animals that have died of such poisoning and may be secondarily poisoned[21].

Heavy metals such as lead, mercury and cadmium may be so concentrated in industrial wastes that they present a hazard to both wildlife and man[25]. Alkyl-mercury compounds previously used as fungicides in seed dressings are more poisonous because of their solubility in both water and fat. In Europe, widespread mortalities occurred mainly in granivorous birds but also in raptors due to secondary poisoning[25]. It was also established that mercury could reduce the production and hatchability of eggs and the viability of chicks. Cases of mercurial poisoning in wildlife in Southern Africa could not be traced, but one case of arsenical poisoning was diagnosed in an eland (Basson P A, 1972, unpublished data).

A serious outbreak of cyanide poisoning in the Kruger National Park has been confirmed recently where cyanide was put in a carcass in order to kill vultures (Kellerman T S, VRI, 1987, pers comm). Approximately 50 vultures were killed as well as some raptors and predators.

An unspecified corrosive weedkiller, used to eradicate kikuyu grass in the Pretoria Zoo, caused buccal and pharyngeal lesions with secondary necrobacillosis in Grant's gazelle *(Gazella granti)* and dorcas gazelle *(Gazella dorcas)* (Basson P A and Smit J, 1970, unpublished data).

A suspected fatal case of creosote poisoning in a black rhino *(Diceros bicornis)* has been reported in the Transvaal[43].

Organic pollution from fish factories evidently caused the death of thousands of rock lobsters *(Jasus lalandii)* in the Western Cape[24]. This was seemingly due to a drastic depletion of oxygen caused by the degradation of accumulated organic matter. Toxicity of oil to waterbirds involves external oiling as well as ingestion of oil during preening[15]. It was established experimentally that lipid pneumonia, gastro-intestinal irritation, fatty hepatosis and toxic nephrosis were some of the most common lesions in such cases.

DISCUSSION

In order to establish the susceptibility of wild animals to poisonous plants of Southern Africa, Basson *et al*[5] have dosed 221 individual animals over a period of six years under difficult circumstances in the field. Although the numbers in some of their trials were still inadequate, valuable information was obtained (Table 7). It became apparent that, wherever co-evolution with toxic plants existed, antelopes usually showed some superior detoxifying mechanisms or innate resistance. It is of interest to note that gifblaar areas generally fall within the natural habitat of both eland and kudu which are mainly browsers or browser-grazers and which proved to have a relatively high resistance against MFA. On the contrary springbok, which are as susceptible as goats, occur in habitats where gifblaar is absent. They are, however, very resistant to slangkop which is found in semi-arid as well as subtropical areas. Various farmers have also reported that the common duiker, steenbok, springhare

(Pedetes capensis) and porcupine *(Hystrix africaeaustralis)* feed on the bulbs of slangkop and other bulbous plants without being poisoned.

In Australia, very interesting work has been done on MFA by Oliver[27, 28], King[18, 19], Mead[23] and Aplin[1]. They studied the metabolism and detoxification of MFA and established, amongst others, that the substance is only detoxified systemically and not by ruminal degradation. Susceptibility of various indigenous animals was determined by administering MFA systemically and subsequently measuring the plasma citrate levels[27]. This was a superior and more reliable method than the one used by Basson *et al.* By these means the co-evolutionary role of MFA in plant-animal interactions was studied and resistance to the compound used as a genetic marker to trace the evolutionary history in their country. Several animals such as the brush-tailed possum *(Trichosurus vulpecula)* from Western Australia where MFA-producing plants occur, were 150 times more resistant than the same species in Southern Australia.

In an excellent review of plant poisoning in free-living wild animals, Fowler (1983)[12] described various factors influencing the susceptibility of wild animals to toxicants. They can cope with these toxicants by avoidance, by eating many other plants and thus diluting the toxicants, by ruminal degradation and by systemic detoxification. Genetically controlled food identification, parental education, interaction with con-specifics, own experience and availability of adequate food supply are all factors that play a role. Starved animals are more likely to consume toxic plants than those with adequate food supply. Also well-nourished animals have a healthier and more balanced gastro-intestinal microflora to assist them in the degradation of toxicants than under-nourished animals or those on maintenance rations. Ingestion of bacteriostatic compounds will consequently also have an inhibitory effect on micro-organisms and thus indirectly increase the susceptibility of animals where ruminal degradation of toxicants plays an important role.

Fowler further stated that effective detoxification is dependant on adequate periodical microsomal stimulation[12]. The translocation of wildlife or plants from their natural habitats to strange surroundings and the confinement of animals to smaller areas than they were used to, are therefore fraught with many dangers. Not only may wild animals ingest strange toxicants and die as a result of a lack of proper defence mechanisms, but inadequate stimulation or exercise of the systems or processes involved in detoxification can lead to poisoning should these animals subsequently be exposed to more poisonous plants. This means that either ruminal degradation or systemic detoxification can become less effective with inadequate stimulation. In this respect it is of special interest to mention the successful ruminal degradation by specific bacteria of the toxicant mimosine, contained in *Leucaena leucocephala* which is an indigenous plant in South America where poisoning of domestic stock by this plant is unknown (Jones R, CSIRO, Townsville, Australia, pers comm). However, poisoning was experienced where this high protein-yielding shrub has been cultivated in Australia[11].

Young animals with poorly developed microsomal systems are more susceptible than adults, and stress factors which are invariably imposed on captured animals can also lead to inadequate detoxification[12].

Research both overseas and in Southern Africa has indicated that, apart from behavioural safeguards, wild animals usually have either an innate resistance to or very efficient ruminal degradation of natural toxicants occurring in their habitats. These features, together with other beneficial factors such as hardiness and more efficient and diverse use of plants (especially by browsers), make them ideal animals in their own domain. This is so because of strong long-term natural selection.

Poisoning of wildlife by pesticides, pollutants and other chemicals is currently of more importance than poisoning by natural toxicants because wild animals lack resistance against these substances. Over the past decades it has therefore become increasingly clear that, in the production of chemicals for the control of parasites and diseases, preference should be given to those that disturb the eco-systems the least. These compounds should be more parasite specific and preferably easily degradable. Education of the public in their use should continue but this is no safeguard. Naturally more emphasis is needed on biological conrol. At a recent symposium on the "Use/Misuse of Poison in our Farming Community" in SWA/Namibia, the following question was posed: "Do we want a monoculture of humans and sheep or a variety of life-forms?" (Van Heerden, 1986, quoted by Ledger[21]). It may well be asked whether we want life at all if we refuse to consider pollution in general as a serious worldwide hazard.

It is evident that further research is required on toxicological aspects of wildlife. By doing so and applying our knowledge to restocking and translocation of animals, mortalities will be minimized.

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PRACTICAL SMALL ANIMAL DERMATOLOGY I: STRUCTURE AND FUNCTION OF THE SKIN

O.M. BRIGGS*

ABSTRACT: Biggs O.M. **Practical small animal dermatology I: Structure and function of the skin.** *Journal of the South African Veterinary Association* (1987) 58 No. 4, 229-231 St. Francis Veterinary Hospital, 157 Main Road, Bergvliet, 8001 Cape Town, Republic of South Africa.

The structure and function of the canine and feline skin is reviewed. Comparisons in tubular form have been made of certain aspects where confusion may arise.

INTRODUCTION

The clinician must have a basic knowledge of the structure and function of skin before the skills of observation, interpretation, and correlation of history, clinical findings, and diagnostic tests can be used to arrive at a correct diagnosis.

The integument forms the anatomic boundary between the animal and its external environment and is the largest of the body organs. The most important function of skin is to act as a barrier. It encloses the body organs, maintaining an internal barrier and also protects against injurious external agents. The two principal layers are the epidermis and the dermis (Table 1).

EPIDERMIS

The chief epidermal cell is the keratinocyte (Table 2) which begins as the germinal cell in the basal layer. As the cell migrates outward with time, it differentiates to form the histologically recognised layers (prickle cell, granular, clear and horny). The prickle cell layer is named from the intercellular bridges which appear after artifactual shrinkage of the cells in fixed preparation. The granular layer consists of nucleated cells whose cytoplasm contains blue-black granules of keratohyalin. The clear layer occurs only in the footpad skin. These layers compose what is known as the living epidermis. The non-living epidermis (horny layer) is composed of many layers of dead, fully keratinised, flattened cells. Together with the granular layer it forms the barrier zone that prevents outward loss of moisture and essential elements, and inward penetration of chemicals, bacteria and other harmful agents.

Other cells are present in the epidermis and their functions are listed in Table 2. The melanocyte is particularly important in animals as pigmentation plays a role in recognition and conversely, camouflage.

Table 1: Structure of canine and feline skin

SKIN (CUTIS)	
Epidermis	
horny layer	(stratum corneum)
clear layer	(stratum lucidum)
granular layer	(stratum granulosum)
prickle cell layer	(stratum spinosum)
basal cell layer	(stratum basale/germinativum)
Epidermal appendages (adnexa)	
glands of the skin	
sebaceous	
apocrine	
eccrine	
nails (ungues)	
hair (pili)	
Dermis	
fibres	
collagen	
elastic	
reticular	
ground substance	
blood vessels, nerves, lymphatics	
dermal cells	
HYPODERMIS (SUBCUTIS)	
stratum adiposum	
stratum fibrosum	

DERMIS

The dermis is composed of collagen, reticulin (immature collagen), and elastin fibres embedded in a glycosaminoglycan-rich ground substance. It is divided into superficial (containing finer fibres of collagen) and deep (coarser) layers. The dermis is constructed to resist tearing, and prevent bruising but is also flexible enough to allow movement. It has homeostatic functions of water storage and the maintenance of blood cations and mineral storage. The cellular elements of the dermis are compared in Table 2.

PILARY APPARATUS

The hair follicle is the basic unit of hair production. It is an epidermal invagination which together with its sebaceous and apocrine glands and erector pili muscle forms the apopilosebaceous unit (Figure 1). At birth the follicles have only one hair emerging from the follicular opening and are known as simple or primary. As the animal grows, accessory hairs develop in accessory or secondary follicles which arise as buds from the original single follicle, forming a compound follicle. There is a single follicular orifice with one primary or guard hair and a number of secondary hairs. Hair follicles undergo cyclic growth, divided into three phases: anagen (active growth), catagen (transitional) and telogen (resting). The hair shaft consists of a central medulla surrounded by a cortex and cuticle.

Hair is the first line of defence and is important in recognition of stimuli. Absence of hair where it would

* St. Francis Veterinary Hospital, 157 Main Road, Bergvliet, 8001 Cape Town, Republic of South Africa

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Table 2: Skin cells

	Function	Biological process	Description
Epidermal			
keratinocyte	protection permeability pigmentation	desquamation	columner-cuboidal to dead flattened keratinocytes in a lamellar pattern
melanocyte	synthesis of melanin pigmentation	melanogenesis and transfer of melanin granules	dendritic cells lying on the basal lamina: appear as clear cells
Langerhans cells	cell-mediated immune reactions	antigen recognition and processing	dendritic cells in upper stratus spinosum appear as clear cells
Merkel cell	slow-adapting mechanoreceptors	specialised neurosecretory cells located in the stratum basale	clear cells confined to the stratum basale especially of tylotrich pads (a sensory organ)
Dermal			
fibroblasts	forms collagen, elastin, and ground sugstance	collagenogenesis	flat, elongated cell with cytoplasmic processes at each end and an elongated, oval nucleus
mast cells	release of vasoactive substances	tissue injury and inflammation cause release by degranulation	round to oval centrally located nucleus prominant nucleolus, cytoplasmic granules
histiocyte/ macrophage	produce reticular fibres and phagocytose material	phagocytosis and immune defence	eccentrically located indented nucleus
plasma cell	antibody production		eccentrically located round nucleus with nuclear material in a clock face pattern; clear area in cytoplasm adjacent to nucleus, remainder basophilic

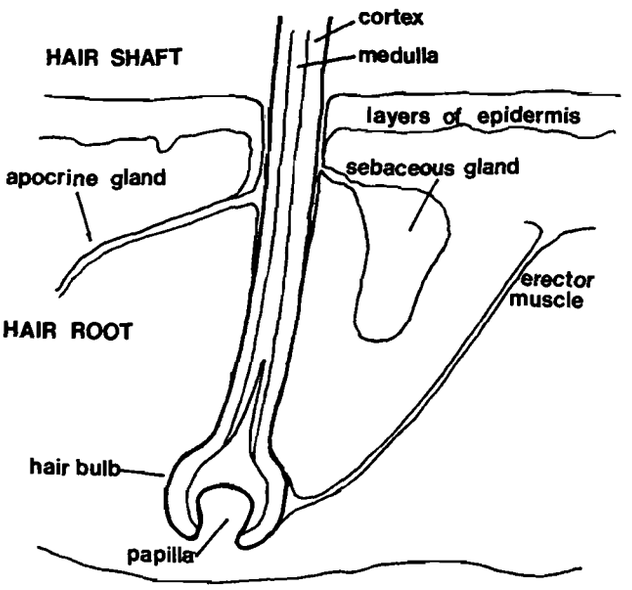

Fig. 1: The hair follicle unit consists of the hair follicle, its erector pili muscle, and apocrine and sebaceous glands. It is known as an "apopilosebaceous complex".

normally be present (alopecia) is an important indicator of disease. Thermoregulation is enhanced by the coat, and specialised (sinus) hairs perform a sensory function.

GLANDS

The sebaceous glands (Table 3) open into the upper part of the hair follicle and are closely associated with it. The apocrine glands are large, loosely coiled glands which lie deep in the dermis and subcutis. The eccrine glands are found only in the footpads in the dog and cat and are located at the junction of the dermis and epidermis. The skin glands play a role in temperature regulation, secretion and excretion besides the obvious function of softening and lubrication.

The barrier function of the skin can be divided into physical, chemical and microbial components. The hair coat, stratum corneum and supportive underlying layers provide the physical barrier. The chemical barrier includes the lipid film from the sebaceous glands and the water soluble surface proteins (immunoglobulins, antiviral glycoproteins, interferon and complement) from the apocrine glands. The pH at the skin surface is kept optimum for protection and a microclimate is maintained to support resident bacteria (the microbial barrier) which inhibit invasion by pathogenic organisms.

The circumanal glands (Table 4) are complexes found near the anal orifice and consist of a superficial sebaceous component opening onto the skin surface and a deeper non-sebaceous part known as the perianal glands. The tail gland area is located on the dorsal surface of the tail just distal to the tail root. The hair coat over this area is often sparce forming an oval, roughened unsightly patch. This can be prominent in male cats ("stud tail") and seborrhoeic animals and is often mistaken as an area of self-trauma in the Bull Terrier.

Table 3: Glands of the skin

	Sebaceous	Apocrine	Eccrine
Location in the skin	evaginations of hair follicles in the dermis	deep in the dermis	junction of dermis and hypodermis
Location on the body	everywhere except planum nasale and glabrose skin	everywhere except planum nasale	only in the footpads
Lining cell	basal cells surround the "foamy" cells (holocrine secretion)	single cuboidal-columnar layer beneath which are fusiform myo-epithelial cells	single layer of cuboidal epithelial cells and a single layer of myoepithelial cells
Secretion	cholesterol, squaline, waxes, esterified fatty acids and protective proteins	proteinaceous, white and "milky" containing protective proteins	watery
Function	softening, physical and chemical barrier	mixes with sebum	unknown in the dog and cat produce true "sweat" in man

Table 4: Glandular components of specialised skin organs

Specialised organ	Glandular component
external auditory meatus	sebaceous and apocrine
anal sacs	sebaceous in duct lining, apocrine in fundus
circumanal glands	sebaceous
tail gland	sebaceous and apocrine

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DIE ANTERIOR ENTERITIS SINDROOM IN DIE PERD

P. STADLER*

ABSTRACT: Stadler P. **The anterior enteritis syndrome in the horse.** *Journal of the South African Veterinary Association* (1987) **58** No. 4, 233-235 (Afrik) Department of Medicine, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, 0110 Onderstepoort, Republic of South Africa.

The anterior enteritis syndrome in the horse is reviewed with reference to the aetiology, pathogenesis, clinical findings, laboratory findings, diagnosis, treatment, prognosis and post mortem findings.

Key words: Anterior enteritis, horse.

INLEIDING

Anterior enteritis, ook bekend as proksimale enteritis, duodenojejunitis, duodenitis-proksimale jejunitis en hemoragiese enteritis[5 9], is vir die eerste keer in 1977 in Georgia beskryf. Hierdie geval is aanvanklik verkeerdelik as 'n ongewone geval van trombo-emboliese iskemie van die proksimale dunderm diagnoseer[11]. Terselfdertyd is enkele, sporadiese gevalle ook in ander dele van die VSA gesien[10 11]. In 1980 is die toestand vir die eerste keer in Duitsland rapporteer, terwyl meer gevalle in Amerika mettertyd beskryf is[4 10].

Die toestand is sover nog slegs in volwasse perde beskryf[5] en daar blyk geen voorkeur te wees vir enige ras of geslag nie[11]. Dit kom enige tyd van die jaar voor, maar blyk nie aansteeklik te wees nie[11].

ETIOLOGIE

Die presiese oorsaak van hierdie toestand is onbekend[10 11], maar daar word vermoed dat bakterië en hul toksiene 'n rol speel[5]. Kulture van die derminhoud van gevalle het nie 'n konstante voorkoms van bakterië aangetoon nie, maar clostridia[5 11] of *Salmonella* spesies[7 13] mag betrokke wees in die ontstaan van hierdie toestand. Verskeie spesies van die genus *Clostridium* is reeds geïsoleer[9 11]. Die verspreiding en voorkoms van nadoodse letsels is soortgelyk aan die wat deur *Clostridium perfringens* in mense en neonatale varkies veroorsaak word[9 11]. *Salmonella* spesies wat soms isoleer is uit gastriese vloeistof of mis, het organismes uit groepe C, D en E ingesluit[3 7].

PATOGENESE

Soos die naam aandui, is dit primêr die duodenum en proksimale jejunum wat in hierdie toestand aangetas word[5]. 'n Moontlike verklaring[3] vir hierdie proksimale voorkoms is dat primêre ileus of 'n nie-strangulerende obstruksie van die dunderm aanvanklik voorkom wat lei tot verminderde uitvloei van suur maaginhoud uit die maag en 'n terugvloei van alkaliese duodenale inhoud. Die gevolglike verhoging van die intestinale pH en verminderde wegvoer van bakterië mag toestande skep wat die oorgroei van sekere organismes bevoordeel. Hierdie toestand ontstaan wel na gastriese chirurgie by die mens[2].

Weens 'n tydelike verhoging in die serum amilase en lipase aktiwiteit van 'n betekenisvolle getal gevalle het die vraag ontstaan of primêre of sekondêre pankreatitis betrokke is in die patogenese[6]. Voorlopige ondersoek dui egter daarop dat die verhogings eerder 'n aanduiding van nierskade is, aangesien daar in die meerderheid van gevalle 'n gepaardgaande verhoging is in die verhouding van die uriene gamma-glutamiel transpeptidase aktiwiteit tot die uriene kreatinienvlak sowel as die teenwoordigheid van proteïen en gietsels in sowel as die teenwoordigheid van proteïen en gietsels in die uriene. Ander moontlike verklarings sluit in toksiengeïnduseerde pankreatitis, stygende infeksie van die pankreasbuis en blokkering van die pankreasbuisopening sekondêr tot erge intestinale inflammasie en nekrose[3]. Geen aanduiding van makroskopiese of mikroskopiese pankreas-skade kon ook gevind word nie[6].

Na aanleiding van bevindinge uit die ondersoek van dermbiopsies en nadoodse dermmonsters van dieselfde perde word die volgende hipotese vir die ontwikkeling van die dermwandletsels daargestel[9]: Aanvanklik is daar uitgesproke kongestie van alle vate in alle lae van die aangetaste derm wat gevolg word deur uitgesproke edeem en bloeding veral in die submukosa. Nekrotiese veranderinge in die mukosa volg op die vaskulêre veranderinge en tas uiteindelik alle intestinale lae aan.

By gevorderde gevalle mag lekkasie, perforasie of ruptuur van die nekrotiese dermwand aanleiding gee tot sekondêre peritonitis.

KLINIESE BEVINDINGE

Die perd mag 'n akute aanvang van anoreksie en depressie toon, maar word meestal gevind met tekens van abdominale pyn[5]. Die graad van abdominale pyn varieer van matig tot uitgesproke[1 5 8]. Die temperatuur kan normaal[5] of verhoog[1 3 10] wees, terwyl sowel die harttempo[1] as die respirasietempo[1] verhoog is. Die slymvliese is kongestief, toksies of in erge gevalle selfs sianoties[5 10 11], terwyl die kapillêre hervultyd verleng is[5]. Kliniese dehidrasie ontwikkel[5 10 11]. Die teenwoordigheid van ileus is 'n belangrike bevinding[5] en diarree kom nie voor nie, hoewel die konsistens van die mis soms kan verminder[5]. 'n Rektale ondersoek is gewoonlik negatief[5]. Ten spyte van die feit dat die dunderm gevul is met

* Departement Geneeskunde, Fakulteit Veeartsenykunde, Universiteit van Pretoria, Privaatsak X04, Onderstepoort 0110, Republiek van Suid-Afrika

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vloeistof, wissel die deursnee daarvan van normaal[5 10 11] tot effens groter as normaal[1]. Gastriese uitsetting kom voor[5] en groot hoeveelhede vloeistof kan herhaaldelik met 'n maagbuis afgetap word[10 11]. Hierdie vloeistof, waarvan die kleur varieer van groen tot rooibruin[8], mag onwelriekend wees[8]. Dit is positief vir okkulte bloed[5] en die pH daarvan is verhoog tot so hoog as sewe tot agt[3 12].

Depressie vervang gewoonlik die koliek na 12-24 uur, hoewel die perd voortgaan om onderbroke pyn te wys met uitsetting van die maag[1 10 11]. Uiteindelik ontwikkel toksiese skok[5 10 11].

Moontlike komplikasies wat sekondêr mag ontstaan, sluit in laminitis[3 5], gedissemineerde intravaskulêre stolling[6], multifokale absessasie van die perifere limfknope[5], nefritis, hepatitis, pneumonie en pleuritis[6].

LABORATORIUM BEVINDINGE

Weens die dehidrasie verhoog beide die hematokrit en die totale hoeveelheid serum proteïene[5]. Daar is dikwels 'n betekenisvolle verhoging in die bloedglukose[5] sowel as die serum laktaat (ongepubliseerde data). Die serum elektroliete is gewoonlik normaal[10 11], hoewel 'n matige hipochloremie in vyftig persent van gevalle voorkom[5]. Die serum kalium is gewoonlik normaal, hoewel die totale liggaamskalium verlaag kan wees in die teenwoordigheid van asidose[5]. Die suur-basis balans hang af van die stadium, die hoeveelheid gastriese terugvloei en die graad van skok[10 11]. Milde gevalle toon gewoonlik minimale veranderinge[10 11] en hoewel 'n metaboliese alkalose aanvanklik mag voorkom[3], toon meeste gevorderde gevalle 'n metaboliese asidose[5].

Daar is geen konstante veranderinge ten opsigte van die witbloedselle nie en beide leukopenie en leukositose met 'n degeneratiewe of 'n regeneratiewe linksverskuiwing word gevind[3 5].

Analise van peritoneale vloeistof lewer nie-spesifieke resultate[8]. Aanvanklik is gedink dat 'n verhoogde hoeveelheid proteïen sonder ander veranderinge spesifiek vir hierdie toestand is[1 8 11], maar latere bevindinge weerspreek dit en dui ook aan dat die totale hoeveelheid gekernde selle in die vloeistof kan verhoog[8]. In die gevalle waar die amilase en lipase aktiwiteit in die vloeistof bepaal is, was dit ook verhoog[5].

DIAGNOSE

'n Definitiewe diagnose kan slegs met behulp van 'n laparotomie of nadoods gemaak word[10 11]. Die kliniese tekens van hierdie toestand is egter spesifiek genoeg om in meeste gevalle 'n kliniese diagnose te kan maak[10 11] en sluit in abdominale pyn, algemene depressie, tekens van toksemie, ileus, redelik negatiewe rektale bevindinge en terminale toksiese skok. 'n Ander belangrike bevinding is dat groot hoeveelhede (verskeie liters) vloeistof met 'n verhoogde pH herhaaldelik met behulp van 'n nasogastriese buis uit die maag onttrek kan word.

Die belangrikste differensiaal diagnose is 'n obstruksie van die dunderm en dit is klinies soms nie onderskeibaar van hierdie toestand nie[10 11].

BEHANDELING

Die behandeling is ondersteunend van aard eerder as om 'n spesifieke etiologiese agent uit te skakel[5] en sluit die volgende in:

- Herhaalde aftap van die maaginhoud[5 11]. Dit mag nodig wees om die maagbuis te manipuleer en te suig om vloei te laat begin[5]. Die toediening van middels deur die maagbuis is nutteloos en teenaangewese, aangesien normale dermvloei afwesig is en meeste van die middels teruggekry word in die vloeistof[5].
- Groot volumes (> 40ℓ) intraveneuse vloeistof[5] om die hipovolemie en skok teen te werk.
- Natriumbikarbonaat vir die behandeling van die metaboliese asidose[5 11].
- Nie-steroïedale anti-inflammatoriese middels soos flunixin meglumien (Finadyne, Centaur) teen 'n dosis van 1,1 mg/kg teen die pyn, inflammasie van die derm en die effekte van endotoksiene[1 5 11]. Die dosis kan herhaal word, met inagneming van die potensiële newe-effekte, indien die pyn weer vererger. Ander pynstillers kan ook oorweeg word, maar hul tekortkoming is dat hul nie die effekte van endotoksiene effektief teenwerk nie.
- Breë-spektrum antibiotika sistemies om septisemie en die gevolge daarvan teen te werk[5]. 'n Kombinasie van natrium bensiel penisillien (Novopen, Novo Industries Pharmaceuticals (Pty) Ltd) intraveneus en gentamisien sulfaat (Genta 50, Phenix) intramuskulêr kan gebruik word[10 11].
- Baie klein dosisse heparien, naamlik 25 IE/kg tweekeer per dag, om te probeer om laminitis te voorkom[10 11].
- *Clostridium* antitoksien is al gebruik, maar die effek daarvan is nie bewys nie[1].
- Plasma terapie mag nodig wees om edeem te voorkom in gevalle waar enteriese proteïen verlies gekombineer met rehidrasie tot erge hipoproteïenemie lei[3].
- Sagte beddegoed behoort voorsien te word en die hoewe behoort gereeld ondersoek te word vir die moontlike ontstaan van laminitis[5].
- Anti-lipopolisakkaried hiperimmuum perde plasma (Atoxin, Atox Pharmaceutical Co.) word gebruik om endotoksemie teen te werk (ongepubliseerde data), maar die effek daarvan is nog nie bewys nie.
- Chirurgiese omleiding deur middel van 'n duodenocecostomie of 'n duodenojejunostomie om terugvloei na die maag te verminder en die spoed van dehidrasie te verlaag[10 11]. Chirurgie moet egter liefs vermy word aangesien baie gevalle op mediese behandeling sal reageer en aangesien die chirurgie selfs die mortaliteit kan verhoog[1]. Chirurgie behoort slegs oorweeg te word in daardie gevalle waar obstruksie vermoed word of waar die intraveneuse vloeistof terapie nie kan vergoed vir die verlies weens gastriese terugvloei nie[11]. 'n Staande laparotomie deur die regter flank is verkieslik[1 5], aangesien algemene narkose baie gevalle met sirkulatoriese skok en/of laminitis tot gevolg gehad het[1].

PROGNOSE

Die mortaliteit is minstens vyftig persent selfs met intensiewe behandeling[5]. Minder ernstige gevalle mag binne 'n paar dae verbeter[11], terwyl meer ernstige gevalle vir tot tien dae terugvloei van vloeistof na die maag mag ondervind[1 11].

MAKROSKOPIESE PATOLOGIE

Letsels kom hoofsaaklik voor in die duodenum en proksimale jejunum, maar af en toe word die laer dunderm en selfs die kolon aangetas[5]. In Duitsland is daar ook 'n hemoragiese gastritis in sekere gevalle gevind[11]. Die graad van die letsels varieer van geval tot geval[10].

Die duodenum en jejunum is uitgeset tot 'n deursnee van 5-7 cm[9 11]. Die serosa-oppervlak vertoon helder- tot donkerrooi met wit of geel strepe en bloedings wat varieer van puntbloedings tot eggimoses[9 11]. Die intestinale lumen bevat 'n rooibruin, waterige vloeistof[9], terwyl die wand effens verdik is weens edeem[11]. Die mukosa is donkerrooi met bloedings en af en toe nekroties met ulserasies[9].

Die makroskopiese patologie kom dus neer op 'n transmurale enteritis van primêr die duodenum en proksimale jejunum bestaande uit hiperemie, edeem, bloeding en nekrose wat strek van die mukosa tot die serosa[5 9]. Hierdie voorkoms en verspreiding van letsels is soortgelyk aan die wat deur *Clostridium perfringens* in mense en neonatale varkies veroorsaak word.

HISTOPATOLOGIE

Mikroskopiese letsels kan van die maag tot die kolon teenwoordig wees, maar is meestal beperk tot die duodenum en proksimale jejunum[9 11]. Die graad van die letsels varieer van geval tot geval as volg[9]: (a) Milde letsels bestaande uit hiperemie en edeem van die mukosa en submukosa met infiltrasie van neutrofiele (b) matige letsels toon areas van degenerasie en verlies van villusepiteel met klein bloedings in die mukosa en submukosa (c) erger letsels toon verlies van epiteel en atrofie van die villi met erger edeem, bloeding en neutrofiel infiltrasie van die mukosa, submukosa, tunica muscularis en serosa (d) die ergste letsels bestaan uit nekrose en bloeding van die mukosa, ekstensiewe fibrienryke edeem en bloeding in die submukosa en bloeding en edeem in die tunica muscularis en serosa. Die wit of geel strepe is foki van nekrose en verettering.

Elektronmikroskopiese ondersoek het die teenwoordigheid van asteroïede plakke op die epiteeloppervlak by die punte van die dundermvilli aangetoon, maar die belang daarvan is onbekend[9].

Konstante letsels is nie in enige ander organe gevind nie[9].

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WINNER OF THE BOSWELL AWARD FOR 1987

DR C M VEARY

It is fitting that this award which is given for eminent service rendered to the Profession through the South African Veterinary Association, is this year awarded to Dr C M Veary, or for some or other reason, better known as 'Tubby'. Few people in recent years have served the Profession with more hard work, enthusiasm or loyalty.

Tubby Veary is currently Associate Professor and acting head of the Department of Veterinary Public Health in the Faculty of Veterinary Science of the University of Pretoria at Onderstepoort.

After matriculating at St John's College, Johannesburg, he obtained the BVSc degree in 1963 and the Diploma in Veterinary Public Health in 1978, respectively, both at the University of Pretoria. Since graduation he initially assisted in a number of private practices in Durban and Johannesburg. Since 1969 he specialised in Veterinary Public Health and held various positions both in municipal and in the State Veterinary Services. After being transferred to the Transvaal Regional Office of the Veterinary Services in 1982 as Assistant director, a position that he held until 1985, he was appointed Associate Professor at the University of Pretoria.

Even though he is only newly appointed in the academic field, he has lectured in the field of Veterinary Public Health on a number of occasions and has also acted as external examiner in the subject at the Veterinary Faculty, University of Pretoria. At present he also has other lecturing responsibilities at the University of Pretoria, Medical University of Southern Africa and in the Department of Health.

Despite all these various activities, he has been one of the most active and involved members of the Association during the last number of years. After becoming a member of the Association in 1964, he was secretary of the Natal Clinicians Group from 1968 to 1969; secretary of the Natal Branch from 1970 to 1964; Vice-Chairman of the same branch from 1974 to 1976 and Chairman of the branch from 1976 to 1978; thereafter he was *ipso facto* member of the Natal branch from 1978 to 1979 and again Vice-chairman from 1979 to 1980. From 1980 to 1981 he was again member of the committee of the Natal Branch whereafter he joined the Northern Transvaal Branch in 1982. In 1982 his contributions were recognised by the Natal Branch who elected him as an Honorary Life Member; he was also elected Honorary Member of the Witwatersrand Branch in 1986.

During this time he was the Natal representative on the Council of the South African Veterinary Association from 1970 to 1978. From 1978 to 1982 he served on the Federal Council of the Association as a nationally elected member whereafter he was the Vice-President of the Association 1982 to 1984 and President from 1984 to 1986.

Currently he is Chairman of the Public Health Group, Chairman of the Finance Committee of Council and member of the following Council committees: Executive Committee, Editorial Committee and the Awards Committee. Previously he served on the Education Comittee and the Advisory Committee on Ethical Matters. He has been a trustee of the Veterinary Foundation since 1974 and has served as vice-chairman of this Foundation.

Despite all these activities he still found the time to become involved in community service and has served actively on town councils, clubs and investigating committees. He is an avid hockey player having been captain of the first hockey team of the University of Pretoria in 1962. Currently he is a team member of the Harlequins Old Croconians.

Particularly during the latter years of his terms of office in the Association, he guided the Association through troubled waters with distinction and it is in particular his dedicated attention to his office as Vice-President and President, and currently as Chairman of the Finance Committee, that will be remembered best. This alone, qualifies him without reservation as a most deserving recipient of the Boswell Award.

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CLINICAL AWARD OF THE SOUTH AFRICAN VETERINARY ASSOCIATION FOR 1987

DR O M BRIGGS

The clinical award recognises excellence in clinical practice and is awarded to any veterinarian who is a member of the Association and registered in the RSA who through his/her study, research and implementation of new ideas has been an example to collegues and has improved the status of the profession in the eyes of the public. This award for 1987 is awarded to Dr Martin Briggs who is a longstanding member of the SAVA mother body and the Cape West Branch.

Martin Briggs matriculated in 1967 at the St Andrews College in Grahamstown and obtained the BVSc degree at the Faculty of Veterinary Science, University of Pretoria in 1976 after first obtaining a BSc degree majoring in mathematics, mathematical statistics and numerical analysis and computation at the University of Cape Town in 1972. In 1977 he became a member of the Royal College of Veterinary Surgeons (by registration) and obtained the MSc (Med) degree in chemical pathology at the Medical School of the University of Cape Town. The title of his thesis for the latter degree is: 'Uric acid metabolism in the Dalmation Coach Hound'. He became a Fellow of the Royal College of Veterinary Surgeons in 1986 by examination in the subject 'Canine Dermatology'.

During this time he first worked as locum tenens in South Africa and in the UK. Thereafter he practiced as an assistant in the Cape Province whereafter he became a partner in a five-person small animal practice.

He expanded his knowledge in dermatology by working with Professor Danny Scott at Cornell University while he also attended various congresses on the same topic.

Despite facing the rigors of general practice, Dr Briggs has published nine papers in local and overseas international scientific journals since 1980. His

readiness to share his knowledge with his colleagues, particularly in the western Cape, has led to the implementation of new ideas specifically in the field of canine dermatology. His close association with the Medical School of the University of Cape Town has also enhanced the status of the profession in these quarters.

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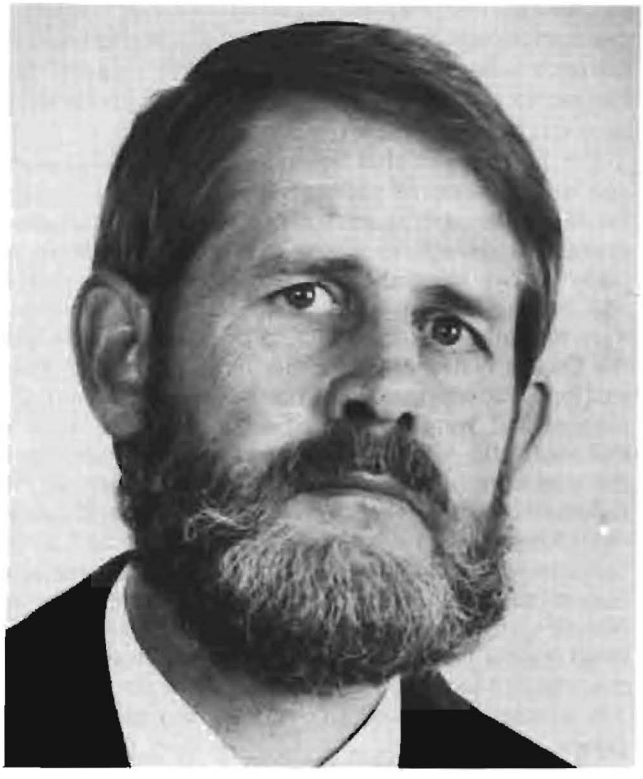
ONTVANGER VAN DIE NAVORSINGSTOEKENNING VAN DIE SAVV VIR 1987

DR J D BEZUIDENHOUT



Vanjaar is die tweede keer dat die navorsingstoekenning aan Dr J D Bezuidenhout, of Dürr, soos hy in die wandel bekend staan, toegeken word. Hierdie toekenning word gemaak aan enige lid van die Suid-Afrikaanse Veterinêre Vereniging vir die beste artikel of reeks artikels wat onlangs in enige wetenskaplike tydskrif verskyn het. Die doel van hierdie toekenning is om navorsing van hoogstaande kwaliteit aan te moedig.

Dr Bezuidenhout kwalifiseer in 1966 as veearts aan die Fakulteit Veeartsenykunde van die Universiteit van Pretoria waarna hy tot 1974 as staatsveearts in Suidwes-Afrika werk. Vanaf 1974 is hy werksaam in die seksie Entomologie by die Navorsingsinstituut vir Veeartsenykunde; eers as veeartsenykundige navorser; vanaf 1979 tot 1981 as hoof van die seksie en sedert 1981 as Assistent Direkteur en Hoof van die seksie Entomologie. Sy hoof navorsingsaktiwiteite is gerig op die biologiese beheer van bosluise, bosluistoksikoses, bosluisweefselkulture, bosluisweerstandbiedendheid teen dipstowwe en hartwater.

Dit is veral weens die vordering wat daar gemaak is in die ontrafeling van die geheime van hartwater, dat hierdie toekenning vir 'n tweede keer Dr Bezuidenhout te beurt val.

Hartwater is een van die belangrikste bosluisoordraagbare siektes van herkouers in Suider Afrika. Aanvanklike goeie vordering in navorsing op die siekte het gedurende die jare 1950 amper tot stilstand gekom. Dit kan met reg gesê word dat dit in hoofsaak vanweë die entoesiasme en werkywer van Dürr Bezuidenhout is dat 'n span spesialiste geaktiveer is om verskeie aspekte van hartwater na te vors. Hierdie aktiwiteite het ook gelei tot die totstandkoming van 'n koördinerende komitee vir hartwaternavorsing.

Die tyd wat Dürr by die Rocky Mountain Laboratory in die VSA deurgebring het, het hom sonder twyfel meer in staat gestel om nuwe benaderings tot die oplos van probleme te definieer. Dit was veral vanweë die onvermoeë om *Cowdria ruminantium* in vitro te kweek, dat baie min vordering gemaak is op verskeie aspekte van hartwater. Veral in Nederland en die VSA het navorsers sonder sukses gepoog om *C. ruminantium* in weefselkultuur te kweek maar was dit onder die leiding van Dr Bezuidenhout dat dié wêrelddeurbraak gemaak is. Hierdie deurbraak stel 'n meganisme beskikbaar waardeur 'n lewendige patogeen in weefselkultuur gekweek kan word; meer gesofistikeerde toedienings kan gemaak word en dit is ook 'n bron van suiwer antigeen vir die bestudering van biochemiese en biologiese aspekte. Verder vergemaklik dit die bestudering van die immunologie en immuniserings potensiaal van hierdie organisme. Reeds is 'n bosluisentstof ontwikkel wat, indien die probleme met toksisiteit uitgeskakel word, heelwat goedkoper en makliker standaardiseerbaar is as die bestaande entstof.

Hy en sy span bestudeer verder die oordraagbaarheid van die organisme en stel vas dat dit ook in ander gashere voorkom — skilpaaie en tarentale dien dan ook as reservoir.

Voortspruitend uit die werk reël Dr Bezuidenhout die internasionale hartwaterkongres wat in 1986 in die Nasionale Krugerwildtuin plaasgevind het. Al die referate sal in die September 1987 uitgawe van die Onderstepoort Tydskrif verskyn en word dit verwag dat dit tussen 500 en 600 bladsye sal beslaan.

Die resultate voortspruitend uit hierdie navorsing verskyn vanaf 1985 as 'n reeks van 12 artikels in verskeie wetenskaplike tydskrifte; 9 artikels wat oor dieselfde onderwerp handel is tans in druk. Dr Bezuidenhout is of die senior of mede-outeur in al die betrokke publikasies.

Dit is met trots dat die vereniging erkenning aan hierdie besondere prestasie kan verleen deur die toekenning van die Navorsingstoekenning.

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SILVER MEDAL OF THE SOUTH AFRICAN VETERINARY ASSOCIATION POSTHUMOUSLY AWARDED TO

THE LATE JOHN MURRAY (IAN) HOFMEYR

Ian Hofmeyr who was an eminent wildlife researcher as well as an outstanding wildlife clinician, died tragically in 1984. During his life he gained worldwide recognition not only for himself but also for the South African veterinary profession. He matriculated in 1958 and obtained the BSc (Agric) degree majoring in pasture science at the University of Pretoria in 1962 and qualified as a veterinarian in 1967. As a student already he showed an early interest in wildlife work and engaged himself in several projects as a member of the Fieldwork section of the Wildlife Society of South Africa. As a student he also worked in the Kruger National Park, the Umfolosi, Hluhluwe and the Mkuzi Game Reserves in Natal, the Moremi Game Reserve in Botswana, Henderson Game Rance in Zimbabwe and the Timbavati Game Reserve in the Transvaal.

After working as a locum in 1968 to gain experience, he joined the Entomology Section of the Veterinary Research Institute at Onderstepoort, where he worked on the biological control of the sand tampan. In 1970 he joined the South West African division of Nature Conservation and Tourism and was appointed as the first veterinarian in charge of the game capture team. This unit became highly mechanised and operated in some of the most remote and rugged areas of the country. Ian assisted in developing this team into one of the most successful in southern Africa.

The first project that he undertook was the capture and translocation of endangered wild animals. A long list of spectacularly successful capture and translocation operations, which involved hundreds of animals of many species attested to his skill, complete dedication and professionalism. The most important operations were the following: introduction of 52 black rhino into the Etosha National Park, capture and airlift of roan antelope, the capture of 121 black-faced impala and the capture and translocation of sable antelope, tsessebe and reedbuck. All these operations were conducted with the minimum of losses. Apart from the capture of endangered species, the capture of problem animals, restocking of farms with game, the capture of game for ecological and veterinary investigations were also successfully undertaken. During his 14 year career in Nature Conservation, using different methods, he captured several thousand animals, totalling 29 species. He immobilised some 2000 animals of 24 species, including 200 immobilisations on free-ranging and captive black rhinos.

Ian Hofmeyr was totally dedicated to the well-being of wildlife and for him the operations did not end when the animals were released in their new homes. He insisted on adequate long-term monitoring of the animals for many years. The fruit of this work is that several endangered species were saved from extinction, and that Etosha and the Waterberg Plateau Park, and even some farmers, can now boast of several new species. In 1975 after five very fruitful years with the game capture team, he was appointed as Wildlife Veterinarian in Okaukuejo, Etosha, where he was responsible for the monitoring of animal diseases and mortalities.

Ian Hofmeyr's work was based on sound conservation requirements and carried out with a flair for improvisation and the development of new and the improvement of old techniques. In fact one of his most important contributions was the development of new capture techniques now used all over Africa, which dramatically reduced the mortalities associated with the capture and translocation of wild animals. In addition to his pioneering work on game capture, he also dedicated much of his time to testing and assessing various drugs. As in the other spheres of his work, he did this with total commitment and attention to detail. Several of these drugs are now in standard use.

Ian Hofmeyr's professional competence and his pioneering research work undertaken in the field of game capture, as well as his expertise in minimising losses during such operations, resulted in world-wide recognition and the frequent invitations to deliver presentations at local and overseas congresses. He was so well-known the world over that the Government of Sri Lanka requested his services in 1979 to assist in the translocation of a pocketed herd of Asian elephants to

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the safety of the Walpattu National Park. Shortly before his death he was invited by Indonesia to capture and translocate elephants and rhino in Sumatra. Also during this time he was listed on the Conservation for Development Register of the IUCN as a specialist in large animal immobilisation.

In spite of the difficult circumstances in the field, he has 22 publications to his credit. At the time of his death he was also registered at the Faculty of Veterinary Science as a candidate for the DVSc degree; the title of his thesis was: 'The role of management and drugs in the prophylaxis and treatment of stress in restrained wild animals'

For his dedicated work he was posthumously awarded one of the first gold medals of the World Wildlife Fund on their 25th anniversary in 1986.

It is against this background that it is an honour for the South African Veterinary Association to award its Silver Medal posthumously to this respected member of our profession.

SILVER MEDALJE VAN DIE SAVV

DR L COETZEE

Die silwer medalje word toegeken aan enige veearts in die Republiek van Suid-Afrika in erkenning van langtermyn diens aan en uitbouing van die Veterinêre beroep.

Louis Coetzee kwalifiseer as veearts in 1961 waarna hy by die Navorsingsinstituut vir Veeartsenykunde in diens tree. Gedurende hierdie tydperk was sy hoofbydrae die identifikasie van en die bepaling van die relatiewe belangrikheid van virussiektes in die pluimveebedryf. As gevolg van sy werk is die tegnologie vir die identifisering van die virusse bemeester en het dit ook as grondslag gedien vir die moderne benadering tot pluimveegesondheid.

In 1965 word hy aangestel as hoof van die Pluimveeafdeling van die Navorsingsinstituut vir Veeartsenykunde te Onderstepoort; 'n pos wat hy tot 1977 beklee. Hierdie fase van sy loopbaan was gekenmerk deur die leidende rol wat hy gespeel het in die bekamping van Newcastlesiekte. Onder sy leiding is ook die eerste oliebasisentstof vir die siekte in die RSA ontwikkel. Hy het ook die leiding geneem in die algemene inenting van hoenders teen virussiektes en het 'n bepalende rol gespeel in die neerlegging van standaarde waaronder pluimvee-entstowwe ingevoer kon word.

Hy tree ook op as tegniese adviseur by die registrasie van pluimveemiddels en speel verder 'n leidende rol deur middel van 'n voorligtingsdiens aan die boer asook die ontwikkeling van 'n bedryf wat besig was om te intensifiseer. Verder is hy lid van die kommissie wat ondersoek instel na die onwettige invoer van pluimvee asook die beleidsbepaling van die regering ten opsigte van die invoer van pluimveeteelmateriaal.

In 1977 word hy hoof van veeartsenydienste by die destydse Stein Broers Plase, of Golden Lay Plase soos dit vandag bekendstaan. Sy aktiwiteite hier lei tot 'n omvattende gesondheidsdiens in die lêbedryf wat gerugsteun word deur die toepassing van die mees moderne tegnologie.

In 1985 word hy aangestel as direkteur van biologiese dienste in die firma waar benewens sy veeartsenykundige insette ook die volgende uit sy werk voortvloei: kwaliteitskontrole programme in die lêbedryf en die vestiging van gevorderde mikrobiologiese tegnieke in voedselprosesseringsmaatskappye.

Hy beklee verder 'n leidende rol in die georganiseerde pluimveebedryf en is voorsitter van die tegniese komitee van die Suid-Afrikaanse Pluimveevereniging (SAPV), ondervoorsitter van die telersorganisasie van die SAPV, lid van die sentrale raad van die SAVP, lid van die statutêre advieskomitee van die minister van landbou aangaande die pluimveebedryf, gekoöpteerde lid op die komitee vir die bepaling van spesifikasies van ontsmettingsmiddels, en as SAVV lid was hy 'n stigterlid van die Noord-Transvaal tak en van die Pluimveegroep.

Gedurende 1980/1 het hy die eerste kommersiële pluimvee-entstof buite Onderstepoort vervaardig; hiervoor kry hy nasionale en internasionale erkenning en ontvang veral hiervoor die volgende toekennings: landboukundige van die jaar, die nasionale produktiwiteitstoekenning, pluimveeman van die jaar asook die World's Poultry Science se goue medalje vir buitengewone wetenskaplike bydrae tot die pluimveebedryf.

Hy behaal gedurende 1981 beide die BVSc Hons en die M Med Vet (virologie) grade en het reeds 12 wetenskaplike en 50 populêr-wetenskaplike artikels publiseer.

Dit is teen dié agtergrond dat dit 'n voorreg vir die Vereniging is om die silwermedalje toe te ken aan 'n man wie se naam sinoniem geword het met pluimvee en die pluimveebedryf.

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GOUE MEDALJE VAN DIE SUID-AFIKAANSE VETERINÊRE VERENIGING VIR 1987

DR A POLSON

Die goue medalje van die Suid-Afrikaanse Veterinêre Vereniging word toegeken aan Suid-Afrikaanse burgers in erkenning vir uitstaande wetenskaplike bydrae in die algemeen of vir 'n voortreflike bydrae tot die veeartsenykundige wetenskap. Die medalje word vanjaar toegeken aan dr. Alfred Polson wat by uitstek voldoen aan die vereistes gestel deur die Vereniging vir die toekenning van die medalje. Dr Polson het 'n uitstekende akademiese rekord en is 'n geëerde lid van die plaaslike en internasionale wetenskaplike gemeenskappe. Gedurende die verloop van sy wetenskaplike loopbaan was hy lank verbonde aan die Navorsingsinstituut vir Veeartsenykunde te Onderstepoort en het hy daarna nog sy verbintenis met die beroep behou deurdat hy verskeie probleme met 'n veeartsenykundige inslag ondersoek het.

Dr Polson is op 11 Maart 1912 te Brandfort in die Oranje Vrystaat gebore; hy ondergaan ook sy skoolopleiding in Brandfort en matrikuleer aan die einde van 1929. Daarna behaal hy die BSc graad met hoofvakke chemie en geologie in 1932 en die MSc graad met die verhandeling 'Die invloed van waterdamp op gasreaksies'. In 1937 word die DSc graad deur die Universiteit van Stellenbosch aan hom toegeken op sterkte van die verhandeling 'Navorsing in verband met die diffusiekonstantes van eiwitte' onder die promotors proff. Meiring Naude en D du Toit. Die DSc graad word ook deur die Universiteit van Kaapstad in 1958 aan hom toegeken op grond van publikasies van hoogstaande gehalte en in 1985 word 'n eredoktorsgraad deur die Universiteit van die Oranje Vrystaat aan hom toegeken.

Verdere erkenning vir sy werk word verleen deurdat hy in 1975 die goue medalje van die Suid-Afrikaanse Mediese Navorsingsraad ontvang asook die toekenning van die Claude Harris Leon Stigting in 1981 wat gegee word aan individue wat 'n wesentlike bydrae gelewer het op die gebied van die Wetenskap, Menslike Gesondheid, Opvoeding en Maatskaplike Welsyn.

Sy wetenskaplike loopbaan wat oor baie jare strek, begin in 1938 toe hy 'n betrekking by die Navorsingsinstituut vir Veeartsenykunde aanvaar waar hy in hoofsaak sy aandag wy aan die ondersoek van die fisiese eienskappe van virusse wat van belang is in die veebedryf in Suid-Afrika veral met die oog op entstofontwikkeling.

Die mees belangrike oorspronklike bydraes wat uit sy werk voortvloei is:

1. Die vervaardiging van kolloïedmembrane om virusekstrakte te steriliseer.
2. Die kweking van anaerobiese organismes in sellofaansakkies wat gelei het tot die vervaardiging van 'n doeltreffende entstof teen lamsiekte in beeste en 'n voorkomende entstof teen tetanus in die mens.
3. Hy was die eerste persoon wat die voorkoms van beskermende teenliggaampies in kolostrum kon demonstreer.
4. Hy het fisies-chemiese tegnieke op 'n wye reeks veeartsenykundige probleme toegepas insluitende die bepaling van serumparameters vir siek diere.
5. Hy was die eerste persoon wat in die VSA die tegniek van papier chromatografie vir die kwantitatiewe analise van aminosuurmengsels toegepas het.
6. Hy was die eerste persoon wat poliovirus en sommige ander virusse wat eie aan Afrika is, geisoleer het.
7. Gedurende 1952 ontwikkel hy 'n metode vir die deurlopende fraksionering van mensserum vir die vervaardiging van immunoglobuliene vir intramuskulêre toediening. Hierdie metode word vir lank deur die firma SERAVAC in samewerking met die Weskaapse Bloedoortappingsdiens gebruik vir die voorbereiding van gammaglobuliene op groot skaal. Die metode is ook toegepas op die voorbereiding van slangbytantiserum.

Van 1946 tot 1951 werk hy by die National Institute of Health in Bethesda, Maryland waarna hy terugkeer na Onderstepoort waar hy aandag gee aan die aminosuurbenodigdhede van mikro-organismes. Vanaf 1952 tot 1977 is dr Polson verbonde aan die virusnavorsingseenheid van die WNNR en later die Suid-Afrikaanse Mediese Navorsingsraad aan die Universiteit van Kaapstad. Gedurende die tyd werk hy veral op die fisieschemiese eienskappe van poliomiëlitis-, hondsdolheid-, slenkdalkoors- en influensa virusse. Hy ontwikkel verskeie nuwe tegieke, o.a. gelfiltrasie met behulp van gelatien en agarose; elektroforese in agarose en spesiale rotors vir ultrasentrifugasie.

Na sy "aftrede" in 1977 word hy aangestel as senior lektor in virologie aan die Universiteit van Kaapstad waar hy sy navorsing voortsit en die isolasie van plantvirusse en teenliggame uit dooiers van eiers bestudeer.

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Sedert 1980 is hy navorser in die departement biochemie aan die Universiteit van Stellenbosch waar hy die voorkoms van sekere antigene in die bloed van kankerpasiënte ondersoek.

Gedurende sy akademiese loopbaan promoveer 10 nagraadse studente onder sy leiding en tree hy ook as adviseur op vir drie MD proefskrifte.

Hy is lid van die 'Society of Experimental Biology and Medicine', stigterslid van die Eksperimentale Biologiegroep (VK en VSA) en 'n redaksielid van die tydskrif 'Preparative Biochemistry'. Hy is ook die stigter van die firma SERAVAC in 1954 wat 'n belangrike vervaardiger word van biologiese reagense en veral versuiwerde ensieme wat ook na die buiteland uitgevoer word.

Onder die talle bydraes wat hy op kongresse gemaak het, is die volgende waar hy as genooide spreker opgetree het, seker van die belangrikstes: The 14th Colloquim, Bruges, Belgium; The 18th Colloquim, Bruges, Belgium; The American National Red Cross Association; The International Congress of Virology en die Gordon Research Conferences in the VSA

Van die ongeveer 170 navorsingsartikels wat onder die naam van dr Polson verskyn het, meesal as senior of alleenouteur, het slegs 7 nie in wetenskaplike tydskrifte verskyn nie. Hierdie publikasies sluit ook 'n aantal hoofstukke in wetenskaplike handboeke in wat handel oor virologie, immunologie en analitiese metodiek in proteinchemie. Van hierdie publikasies verskyn ten minste 10 na sy aftrede.

Dit is veral in die ontrafeling van die geheime van die mens-, dier-, en plantvirusse waar sy groot bydrae lê. Sy besondere eksperimentele vindingrykheid het gelei tot die ontwikkeling van tegnieke wat vandag nog internasionaal gebruik word.

Dr Polson is getroud met mej Susanna le Roux en die egpaar het twee kinders.

As mens is dr Polson ongekunsteld en nederig met 'n besondere voorliefde vir die natuur. Tussen al sy wetenskaplike aktiwiteite het hy nog die tyd gevind om 'n kundigheid te ontwikkel op die gebied van inheemse struike en vetplante.

Dit is 'n voorreg vir die Vereniging om die Goue Medalje vanjaar aan dr. Polson toe te ken.

PROF DR BEREND CORNELIS JANSEN

Berend Cornelis Jansen, alombekend as Ben Jansen, is op Maandag 13 Julie in die ouderdom van 65 jaar na 'n lang en uitputtende siekbed oorlede. Daarmee het 'n einde gekom aan die prestasieryke loopbaan van een van die mees gerespekteerde en toegewyde veeartsenykundige wetenskaplikes wat hierdie land nog geken het.

Prof. Jansen is op 12 Augustus 1921 op Cradock, in die Kaapprovinsie gebore. Reeds op skool, en later as student in die veeartsenykunde, het hy getoon dat hy uitermate begaafd is deur die hoogste moontlike toekennings te verower. Sy skoolloopbaan is byvoorbeeld in 1939 bekroon toe hy in klas I aan Cradock se Hoërskool matrikuleer het en ook die Dux Medalje verower het. Hy sit sy briljante akademiese loopbaan aan die Fakulteit Veeartsenykunde te Onderstepoort voort deur die BVSc-graad *(cum laude)* te verwerf, terwyl die gesogte Theiler-medalje ook aan hom toegeken word.

Sy loopbaan as veearts neem in Johannesburg 'n aanvang waar hy vir ongeveer vyf jaar praktiseer. Daarna begeef hy hom as navorser na die Navorsingsinstituut vir Veeartsenykunde te Onderstepoort.

Prof Jansen se navorsingsloopbaan begin op 'n hoë noot in die afdeling Protosoölogie waar hy ontdek dat tetrasikliene 'n chemoterapeutiese werking teen die moeilik behandelbare *Babesia equi* het. Later word hy na die Afdeling Bakteriologie oorgeplaas waar hy hom in dié vakgebied verdiep en heel gou navorsingswerk van internasionale gehalte verrig. Aanvanklik gee hy heelwat aandag aan bloednier, een van die heel belangrikste siektes van skape in die Republiek van Suid-Afrika. Sy besonder oorspronklike werk op die oorsaak, eksperimentele verwekking en, veral, die immunologie van die siekte, vorm die fondament waarop die beheer van die siekte, soos dit tans in Suid-Afrika daar uitsien, gebou is. Op sterkte van hierdie en ander werk van uitstaande gehalte word die gesogte "Nuffield Fellowship for Research in Biological Sciences" vir studies in Brittanje in 1956 aan hom toegeken.

Voorts wend hy hom ook tot navorsing op lamsiekte. Onder andere karakteriseer hy deur middel van gesofistikeerde basiese navorsing die antigeniese toksienfraksie van die veroorsakende mikrobe. Hierdie inligting is nie net van groot waarde vir die vervaardiging van 'n entstof en antiserum teen lamsiekte nie, maar werp ook lig op die genetiese samestelling van die organisme, wat op sy beurt die taksonomie toelig. Hy gee ook heelwat aandag aan ander siektes wat deur anerobe organismes veroorsaak word.

Vanweë sy sterk leierseienskappe en onteenseglike administratiewe bekwaamheid, word Prof. Jansen in 1961 op die relatief jeugdige ouderdom van 41 jaar aangestel as Direkteur van die navorsingsinstituut vir Veeartsenykunde, te Onderstepoort, een van die jongste direkteure in die geskiedenis van die wêreldberoemde Instituut. In 1963 kry hy ook die gewigtige portefeulje van Dekaan van die Fakulteit Veeartsenykunde, Universiteit van Pretoria, by.

Ten spyte van al hierdie administratiewe verpligtinge, bly hy steeds aktief by navorsing betrokke. Hy verwerf inderdaad tussen 1960 en 1971 die volgende drie doktorsgrade op sterkte van sy omvattende studies op immunologiese aspekte van bloednier, lamsiekte en tetanus:

* In 1960, 'n DVSc *(cum laude)* aan die Universiteit van Pretoria
* In 1966, 'n DSc aan die Potchefstroomse Universiteit vir CHO
* In 1971, die PhD in Mediese Wetenskap aan die Universiteit Stellenbosch

Benewens die 43 wetenskaplike publikasies van besonder hoë gehalte wat uit prof Jansen se navorsing voortgevloei het, het hy talle voordragte op diverse wetenskaplike byeenkomste en menige openings- en geleentheidsredes gelewer. So het die besondere eer hom byvoorbeeld in 1986 te beurt geval om op uitnodiging van die Fakulteit vir Veeartsenykunde, Universiteit van Pretoria, die Theiler Gedenklesing te lewer.

In 1968 word hy tot Hoofdirekteur van Veeartsenydienste in die Department Landbou-tegniese Dienste bevorder en in 1974 word Veeteelt en Suiwel bygevoeg. Hy dien dan ook op die Bestuur van genoemde Departement. As Programbestuurder van Veeartsenykundige Dienste en Veeteelt en Suiwel beheer hy die toekenning van fondse vir navorsingsprojekte en oefen sodoende 'n onmiskenbare invloed op die gang van navorsing en ont-

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wikkeling op hierdie wetenskappe uit. Sy dinamiese leiding van hierdie veterinêre en veekundige instansies, tot groot voordeel van die veebedryf in Suid-Afrika, en die invloed wat hy daardeur laat geld, besorg aan hom 'n blywende plek in die dieregesondheids-annale van ons land.

Terwyl hy as Hoofdirekteur werksaam is, word hy in sy persoonlike hoedanigheid tot die gradering van 'n departementshoof bevorder. Met 'n enkele uitsondering is dit die hoogste sport wat 'n veearts nog ooit in die hiërargie van die betrokke Departement bereik het.

Prof Jansen was ook een van die mees bekende en beste dosente aan die Fakulteit Veeartsenykunde van die Universiteit van Pretoria. Sy loopbaan as dosent begin in 1958 toe hy as senior lektor in Bakteriologie aangestel word. In 1963 word hy bevorder tot Professor en Hoof van die Departement Infeksiesiektes, 'n posisie wat hy tot in 1973 met uitlywing van die Fakulteit Veeartsenykunde behou. In 1976 val die eer hom te beurt om as Ere-Professor in die Departement Infeksiesiektes van die Fakulteit aangestel te word, en in 1980 volg nog 'n prestasie toe hy in die nuwe stoel van Professor in Kleinveesiektes aan die Fakulteit aangestel word.

Sy entoesiasme vir sy vak, en die lewendige en interessante wyse waarop hy sy lesings aangebied het, het menige student in die veeartsenykunde geïnspireer, soos talle veeartse sal getuig. Sy bydrae tot die uitbouing van Suid-Afrikaanse veeartse as wetenskaplikes van formaat, of hulle nou navorsers, diagnostici, voorligters, dosente of praktisyns is, is onteenseglik groot.

Prof Jansen se aansien en bekwaamheid het hom talle aanstellings besorg op wetenskaplike liggame en in die wetenskaplike verenigingslewe. Sy bydraes was nie net tot die veeartsenykunde beperk nie, maar het die hele biologiese spektrum gedek. Slegs die belangrikste voorbeelde word hier genoem. Hy was byvoorbeeld sedert 1961 voorsitter van die Veeartsraad, 'n statutêre pos wat groot aansien geniet. In 1983 is hy tot President van die "nuwe" Veeartsraad verkies. Hy was vanaf 1975 tot 1978 vise-president van die Wêreld Veeartsenykundige Vereniging. Sedert 1960 was hy ook reeds 'n Volle Lid van die S.A. Akademie vir Wetenskap en Kuns. Hy was vanaf 1961 tot 1978 Raadslid van die S.A. Veterinêre Vereniging, eers as verkose en later as gekoöpteerde lid. Vanaf 1969 is hy lid van die Uitvoerende Komitee van die Mediese Navorsingsraad en sedert 1970 lid van die Komitee vir Biologiese Wetenskap van die S.A. Atoomkragraad. Van dieselfde datum is hy Raadslid van die Transvaal Museum. Hy het voorts vanaf 1984 op die Wetenskaplike Adviesraad van die Eerste Minister gedien.

Eerbetonings en toekennings het ook nie agterweë gebly nie. In 1975 is die Havenga prys van die S.A. Akademie vir Wetenskap en Kuns aan hom toegeken. Die Goue Medalje van die Suid-Afrikaanse Mediese Navorsingsraad verwerf hy in 1980. Daarop volg die Goue Medalje van die Suid-Afrikaanse Veterinêre Vereniging in 1983; die Claude Harris Leon and Percy Fox Foundation-toekenning in 1986; die Goue Medalje van die Mikrobiologie-vereniging, ook in 1986 en in 1987 die Goue Ram Toekenning van die Nasionale Wolkwekersvereniging. Die graad DSc *(honoris causa)* van die Potchefstroomse Universiteit vir CHO word ook in 1987 aan hom toegeken.

In 1978 kom hy as Hoofnavorser van die Suid-Afrikaanse Wolraad terug na dit wat altyd naaste aan sy hart gelê het, naamlik veeartsenykundige navorsing waaroor hy by herhaling verklaar het dat hy sy hele lewe sou wy. In die relatiewe kort tydjie wat nog vir hom beskore is, doen hy baanbrekerswerk op bepaalde skaapsiektes, en werp veral nuwe lig op die oorsake en beheer van onvrugbaarheid van ramme. Na sy aftrede in 1986 word hy by die Navorsingsinstituut vir Veeartsenykunde heraangestel waar hy sy navorsing op, en die ontwikkeling van entstowwe teen skaapsiektes voortsit, terwyl hy steeds die stoel in kleinveesiektes by die Fakulteit Veeartsenykunde beklee.

Ten spyte van die wete dat sy lewensdraad uitloop en sy fisiese vermoëns afneem, stel sy onblusbare energie en innoverende gees hom in staat om tot op die laaste braak grond te ploeg en die nuut-gegenereerde kennis aan sy kollegas vir verdere ontwikkeling beskikbaar te stel. Hy sterf inderdaad in die tuig, soos hy dit sekerlik wou gehad het.

Ben Jansen was egter nie net verknog aan sy werk nie. *'n Hegte gesinsverband en 'n vriendelike tuiste was* deurgaans besonder belangrike waardes in sy lewe. Hy was diep gelowig en het op kenmerkende energieke wyse ook getrou sy Skepper en sy Kerk gedien, selfs toe sy liggaam reeds aansienlik deur siekte verswak was.

Hy is in Januarie 1948 met Joyce de Jong getroud en uit die huwelik is 'n dogter en drie seuns gebore. Sy vrou het hom ongelukkig in Januarie 1982 na 'n kort siekbed ontval. Sy kinders het almal presteer en ten tye van sy dood was hulle reeds stewig in hulle onderskeie loopbane gevestig. Hy kon ook die groot geluk en plesier wat met sewe kleinkinders gepaard gaan, ervaar. In September 1983 is hy met Magda Duvenage getroud wat hom oorleef.

Alhoewel hy ontydig vroeg oorlede is, het hy 'n besonder vol en gelukkige lewe gehad. Sy eggenote en kinders en hulle gesinne sal troos en krag asook inspirasie kan put uit die ryk bron van aangename herinneringe afkomstig uit hulle intieme verbintenis met hierdie merkwaardige man.

BOOK REVIEW BOEKRESENSIE

RUMINANT UROGENITAL SURGERY

C F B HOFMEYR

1st Edn. The Iowa State University Press, Ames, Iowa, 500100. 1987 174 pp, illustrations 154, Price $36.95 (ISBN 0-8138-1591-6)

Professor Hofmeyr's surgical expertise and intellectual capacity is held in high esteem by all who have worked with him.

This book is the culmination of many years' experience in all aspects of surgery of the bovine uro-genital system. Although it is particularly in the fields of surgery of the penis of cattle where he has received national and international recognition, the text deals in detail with the male and female reproductive and urinary tracts as well as surgery of the udder.

The book is well illustrated and contains many suitable photographs but should not be seen as an atlas. For this reason, pre-graduate students might find the interpretation of certain surgical procedures difficult.

The text constitutes an excellent survey of a field of surgery which is second in importance only to that of surgery of the gastro-intestinal tract. However, due to the rapid development in veterinary surgical techniques, certain of the procedures described in the text, have been improved on.

S.S. van den Berg

VETERINARY GENETICS

F W NICHOLAS

Clarendon Press Oxford; Oxford University Press, Southern Africa, P.O. Box 1141, Cape Town, 8000, 1987, pp xvii and 580. Illustrations 158, Tables 55, Price R155,85. (ISBN 0 19 857569 6)

Veterinary Genetics encompasses those aspects of genetics that are relevant to animal diseases and to animal production.

Part I deals with Basic Genetics and concentrates on general principles of genetics that apply to normal, healthy animals. The exceptions to these principles are often the basis of genetic diseases. This is followed by a review of recombinant DNA techniques which offers substantial prospects in the production of particular proteins, in the use of probes for the detection of genes and disease organisms in animals.

Part II discusses genetic aspects of animal disease. It is primarily concerned with the principles of medical genetics, illustrated whenever possible with examples from domestic animals. The different sections are subdivided in Biochemical Genetics, Chromosomes and Chromosome Aberrations, Single Genes in Populations, Immunogenetics and Pharmacogenetics. Furthermore, two sections are particularly relevant to animal disease, the first one on genetical aspects of interaction between hosts on the one hand and parasites and pathogens on the other. Special emphasis is given to the development of resistance in parasites and pathogens. The second one examines the environmental and genetic options available for the control of inherited diseases in animals.

Part III covers genetic aspects of animal breeding, with an emphasis on the application of genetic knowledge to improvement of animal populations. The different sections are listed as follows: Single genes in animal breeding, Relationship and Inbreeding, Variation and Heritability, Animal Improvement by Means of Selection and Crossing.

This new book is a most useful contribution to the relatively new field of Veterinary Genetics. The genetic aspects of animal breeding has been outlined in many previous books, but the real value lies in Part II. Genetic aspects of animal disease have in my opinion never been as thoroughly discussed as in this book.

D.R. Osterhoff

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BOLTON'S HANDBOOK OF CANINE AND FELINE ELECTROCARDIOGRAPHY

N. JOEL EDWARDS

2nd Edn. W.B. Saunders Company, West Washington Square, Philadelphia, PA 19105. 1987 pp xix and 381, numerous illustrations and tables, Price $30,00 (ISBN 0-7216-1847-2)

Most veterinary practices today can afford to buy electrocardiographic equipment and the recording of an electrocardiogram is no longer an unusual event. The electrocardiogram, despite its many limitations, has become a most useful diagnostic tool. The reading of electrocardiograms and application of findings to the veterinary patient may, however, baffle the uninitiated — to the extent that it may prompt the practitioner to store his paid-for-machine for good in the cellar or lower drawer of a seldom-used cabinet. Bolton's Handbook of Canine and Feline Electrocardiography does, however, provide an answer. This no-nonsense, soft-cover, ready-for-use-in-the-clinic textbook provides a concise introduction to the subject, followed by a logical step by step approach to the up's and down's of an electrocardiographic tracing as well as its relevance to the patient on the table. There is no painful struggling through a long-winded text in this publication. The very liberal use of illustrations allows for easy understanding which makes it a pleasure to try and diagnose one's own tracings.

The self-assessment section in the book allows for evaluation of one's newly-gained knowledge. The text also includes a most useful glossary of terms as well as a list of drugs with recommended dosages.

I have no hesitation in strongly recommending this book to all canine and feline practitioners as well as to students with a special interest in cardiology.

J. van Heerden

AN ATLAS OF X-RAY TOMOGRAPHICAL ANATOMY OF THE SHEEP

A DAVIES, K GARDEN, M YOUNG and C REID

DSIR Science Information Publishing Centre, Wellington, New Zealand. 1987. 140 Tomographic image plates Price US$ 79,95 (ISBN 0-477-02506-4)

The atlas illustrates the topographical anatomy of a two year old New Zealand Romney ewe by utilising a X-ray computer assisted tomographic machine. Transverse slices were made at constant intervals of 10 mm along the body axis. X-ray computer assisted tomography greatly increases tissue discrimination compared to conventional radiography resulting in an image in which most soft tissue structures can be differentiated.

Part I contains 122 photographic plates in a cranio caudal sequence with two plates per page. Each plate is accompanied by an annotated sketch identifying major structures.

Part II shows 18 photographic plates, selected for their regional interest from Part I and enlarged to show more detail.

This atlas will benefit veterinary anatomists, scientists with an interest in ruminants and radiologists utilising sectional display techniques such as computer tomography and ultrasonography. Private practitioners may well find the atlas interesting but with little practical application.

R.M. Kirberger

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