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GESKEDULEERDE MEDISYNES

Daar bestaan skynbaar baie onkunde by die professie oor die voorskryf en uitreik van veterinêre medisynes. Aangesien daar egter gevalle voorkom waar nie-professionele persone betrokke is by die hantering van potensieel nadelige en selfs uiters gevaarlike middels, moet kollegas gewaarsku word om ernstig kennis te neem van die verpligtinge wat op die veearts rus wanneer 'n voorskrif uitgereik word of middels in die hand van die eienaar geplaas word.

Dit is uiters belangrik dat die veearts daarvan bewus moet wees dat hy deurgaans aanspreeklik bly vir die gebruik van enige medisyne wat direk aan die publiek verskaf word, of waarvoor 'n voorskrif uitgereik word. Dit kan slegs geskied na 'n konsultasie waartydens die kliënt se besondere probleem bespreek word, of waar 'n diagnose gemaak word na aanleiding van die ondersoek van 'n dier of diere, òf, alternatiewelik, 'n behoefte vir die voorkomende gebruik of gebruik van middels vir bestuurdoeleindes, bepaal word.

Nou eers is die veearts geregtig om behandeling toe te pas, 'n middel te voorsien of 'n voorskrif uit te reik. Hiermee aanvaar hy volle verantwoordelikheid vir sy optrede betreffende die betrokke middel se gevolge. Hy moet oortuig wees dat die gebruik van die middel geregverdig is nadat die risiko-voordeelverhouding beide ten opsigte van die betrokke dier en die persone wat dit toedien, oorweeg is en verder moet hy verseker dat die kliënt volledig oor die gebruik van die middel en alle voorsorgmaatreëls ingelig is. Dit sluit bv. die toediener se eie beskerming in die geval van wildvangmiddels in, of geskikte onttrekkingsperiodes met betrekking tot residu's in die geval van middels wat vir voedseldiere bedoel is.

Die veearts se optrede moet in alle gevalle sulks wees dat hy dit ter eniger tyd en geredelik voor die Veeartsraad of in 'n hof kan regverdig.

Die uiters kragtige en gevaarlike neuroleptiese analgetika en ander middels vir die vang van wild behoort slegs in hoogs uitsonderlike gevalle deur nie-veeartse gebruik te word en dan slegs na al die spesifieke voorsorgmaatreëls waarop in 1977 deur die Veeartsraad en die Medisynebeheerraad besluit is, nagekom is. Gevalle het aan die lig gekom waar verantwoordelike kollegas, wat vertroud is met die besondere gebruik en gevare van hierdie groep middels, na grondige oorweging dit nie aan 'n betrokke boer wou uitreik nie en lg. dan tog 'n voorskrif van 'n ander veearts wat min of geen praktiese kennis van gebruik en gevare van die middels het nie, verkry het. Sulke onverantwoordelike optrede kan nie gekondoneer word nie en stel dit die professie in 'n uiters swak lig. Dit behoort oorweeg te word of die gebruik van hierdie middels nie 'n aksie moet wees wat slegs by die veearts, en dan spesifiek dié wat die nodige opleiding in en ondervinding van die gebruik daarvan het, tuishoort nie.

'n Ander bekommerende faktor is die teenwoordigheid en oorsprong van rakke vol geskeduleerde medisynes op plase wat meestal nie oordeelkundig en met voordeel deur die boer benut kan word nie. Dit is 'n vereiste van die wet dat 'n veearts of apteker wat 'n middel aan 'n boer verskaf dit o.a. ook moet voorsien van 'n etiket of ten minste 'n plakker waarop sy naam en adres verskyn. Indien hierdie vereiste konsekwent nagekom word sal die ongeoorloofde bronne van hierdie middels baie gou opdroog.

SUCCINYLDICHOLINE AND GAME CULLING

Modern western society dictates that whenever man should kill, be it man or beast, for whatever reason, he should do so in a way that would result in instantaneous death of the victim so as to minimize any stressful effects of such an act. Few people would therefore applaud a kill that is conducted in a way considered by society as inhuman.

The use of drugs in the humane euthanasia of mammals is therefore directed at causing quick instantaneous death with minimal stressful perceptions by the animal. It is therefore not difficult to understand why succinyldicholine, a depolarizing neuromuscular blocking agent, has not been condoned as a drug for humane killing of animals. This drug results in muscular paralysis with complete retention of consciousness, and death by paralysis of the respiratory apparatus. The use of succinyldicholine in culling programmes in the Kruger National Park has subsequently became a highly controversial subject amongst naturalists including hard-stance realists, armchair conservationists and devoted animal lovers. In culling operations in the Kruger National Park, succinyldicholine is used as an immobilising agent and as such it probably aggravates the state of anxiety in animals. This concept is clearly supported by the work of Dr Hattingh and co-workers published in this issue of the journal. Herding free-living wild animals and subsequently immobilising them with a neuromuscular blocking agent resulted in highly elevated levels of cortisol. If we accept the fact that elevated peripheral serum cortisol concentrations under these conditions can be regarded as a parameter of stress, then there is little doubt that these animals were experiencing severe stress. This unfortunate state of events in the elephant and buffalo culling operations should, however, be compared to what happens almost daily in most parts of our country: animals (domestic) are herded, transported by road and/or rail sometimes over days, kraaled at abattoirs surrounded by the smell of blood (and who knows by which other chemical signs of fear and anxiety!) and only then are they eventually slaughtered in a humane way. We are not aware of a national outcry against this practice. And perhaps the reason for this silent approval simply lies in the fact that it is at present a practical way of supplying the man in the street with a source of protein. This then should also be at present our answer to the culling programme dilemma: For want of a better method, succinyldicholine is used.

We should, however, constantly be harrassed and embarrased by the stressful nature of the use of succinyldicholine in culling operations. Our search towards a more humane way should thus be continued in haste and because of our incomplete knowledge of life and death, this may well prove to be a never-ending process.

As a profession which concerns itself greatly with ehtical codes of conduct towards animals, it is our duty to ensure that the matter is never shelved but that it remains topical.

BLOOD COMPOSITION IN CULLED ELEPHANTS AND BUFFALOES

J. HATTINGH*, P.G. WRIGHT*, V. DE VOS**, I.S. McNAIRN*, M.F. GANHAO*, M. SILOVE*, G. WOLVERSON* and S.T. CORNELIUS*.

ABSTRACT: Hattingh J.; Wright P.G.; de Vos V.; McNairn I.S.; Ganhao M.F; Silove M.; Wolverson G.; Cornelius S.T. **Blood composition in culled elephants and buffaloes.** *Journal of the South African Veterinary Association* (1984) **55** No. 4, 157-164 (En). Department of General Physiology, University of the Witwatersrand, 1 Jan Smuts Avenue, 2001 Johannesburg, Republic of South Africa.

Blood composition of succinyldicholine culled elephants and buffaloes was compared with that of undisturbed animals shot in the brain. The results show statistically significant differences in a number of variables including plasma ACTH and cortisol concentrations. The observed changes are attributed to stress induced by a combination of herding and darting with succinyldicholine and asphyxia. Extrapolation from blood oxygen tensions suggests that this stress may be perceived for an undetermined period which is probably longer in elephants than buffaloes.

Key words: elephant, buffalo, succinyldicholine, culling, stress.

INTRODUCTION

The controversy concerning the use of succinyldicholine (SDC) in game cropping operations has recently been revived[4 5]. These authors reported a massive increase in plasma catecholamines following SDC induced asphyxia in calves. From EEG records they concluded that psychic stress was induced and persisted for several minutes. In sheep, similarly paralysed, plasma catecholamine and glucose levels increased more in conscious than in a pentobarbitone anaesthetised control group. The difference in the values was ascribed to fear resulting from asphyxia.

The blood composition of game animals culled using SDC has not been thoroughly investigated so that whether changes similar to those reported for undisturbed calves and sheep develop is unknown. We collected blood from elephants and buffaloes during culling operations in the Kruger National Park and measured the plasma concentration of components likely to be affected by stress. The values for blood obtained from undisturbed animals killed by a shot into the brain were compared with values from animals killed by several variants of the culling procedure. The entire study was carried out over a 2 week period in the Kruger National Park.

MATERIALS AND METHODS

Animals

All elephants (*Loxodonta africana*) and buffaloes (*Syncerus caffer*) used in this study were animals included in the current game management programme in the Kruger National Park. They came from the same geographical area (around Letaba) and were culled during August 1983.

The following groups were investigated (in all cases shot means that animals were killed by a bullet fired into the brain from close range):

Elephants

1. Undisturbed, shot, sampled. Individual animals were spotted by helicopter, the location given to the hunter who approached undetected on foot or in a vehicle and shot the animal. All animals in this group died instantaneously (brain death) and blood samples were obtained as soon as possible thereafter (Table 1).
2. Herded, shot, sampled. The animals were herded by the helicopter as during normal culling (see 4 below) but were then shot with a rifle from the helicopter. They were herded for variable times (Table 1) but no SDC was used. Most animals died instantaneously (brain death) and blood was then obtained as soos as possible.
3. Undisturbed, SDC, sampled. Individual animals were approached rapidly by helicopter, given the culling dose of SDC (see below), and then left alone until they collapsed. They were not herded in any way and were only minimally disturbed by the darting procedure. Blood samples were taken as soon as possible after the animal collapsed. All animals showed respiratory movements at the time of sampling and were then shot.
4. Herded, SDC, sampled. A selected group of animals was herded by helicopter and darted with SDC in the usual culling procedure. When the last animals had collapsed, blood samples were taken immediately before the animals were shot. The darts were numbered so that the time from darting to sampling could be measured (Table 1). All animals in this group showed respiratory movements when blood samples were taken.
5. Herded, SDC, shot, sampled. The same procedure as in 4. except that the animals were shot and blood samples were taken immediately afterwards. All animals showed respiratory movements up to the time of shooting.
6. Herded, SDC, delayed sampling. The same procedure as in 4. but blood samples were taken after all respiratory movements had ceased for at least 5 minutes.
7. Undisturbed, SDC, repeated sampling. For a special series of experiments, animals were treated as in 3. above, but sampling was continued on a regular basis until all respiratory and circulatory function had ceased.

Except for animals in Group 6, cardiac arrest had not occurred when blood was sampled. Arterial and venous samples were taken from ear arteries and veins. When

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Table 1: GROUPS OF ANIMALS USED

Groups	No.	*Herding duration min	**Sampled after min	Sex	***Number darts or shots per animal		Shoulder height m	Chest circumference m x 2
Elephant								
1 Undisturbed, shot, sampled	5	—	1,5-3,0 arterial blood in 45 sec	5 adult ♂	1	$\bar{x}$ SD	2,04 0,17	3,05 0,10
2 Herded, shot, sampled	7	3-19	1-3 arterial blood in 60 sec	5 adult ♂ 1 young ♂ 1 young ♀	1-3	$\bar{x}$ SD	2,72 0,11	—
3 Undisturbed, SDC, sampled	6	—	4,5-17 $	4 adult ♂ 1 old ♂ 1 young ♀	1-4	$\bar{x}$ SD	2,36 0,40	2,90 0,51
4 Herded, SDC, sampled	17	3-20	7-26 $$	2 adult ♂ 5 adult ♀ 4 young ♂ 4 young ♀ 2 calves ♂	1-4	$\bar{x}$ SD	1,85 0,36	2,55 0,63
5 Herded, SDC shot, sampled	5	6-20	7-29 $$∅	2 adult ♀ 3 young ♂	1-2	$\bar{x}$ SD	2,17 0,55	3,04 0,31
6 Herded, SDC, delayed sampling	7	8-11	18-42 $$	2 adult ♀ 5 young ♀	1-2	$\bar{x}$ SD	2,09 0,39	3,20 0,84
Buffalo								
1 Undisturbed, shot, sampled	5	—	1,5-8	5 adult ♂	1-2	$\bar{x}$ SD	—	—
2 Herded, shot, sampled	8	4-8	5,5-13	8 adult ♂	1-3	$\bar{x}$ SD	—	—
3 Undisturbed, SDC, sampled	6	—	7-16 ◫	2 adult ♀ 3 adult ♂ 1 old ♂	1-4	$\bar{x}$ SD	—	—
4 Herded, SDC, sampled	5	12-19	8,0-13 ◫	4 adult ♂ 1 young ♂	1-4	$\bar{x}$ SD	—	—

* Herding duration – from the start of chase until the animal was darted or shot.
** Sampled after – from the time of darting or brain shot until blood sampling was complete.
*** Number of darts or shots – if more than one shot or dart was fired this was in rapid succession.
$ animals down after 3,75 to 13 min.
$$ animals down after 4 to 10 min.
$$∅ blood taken immediately after brain shot.
◫ animals down after 1½ to 5 min.

SDC was used, the animals received from 1 to 4 darts depending on their size, fired from a dart gun. Each dart contained 7,5 ml SDC (Scoline, Glaxo) at a concentration of 56 g/100 ml water.

Buffaloes

1. Undisturbed, shot, sampled. These animals were approached carefully with a vehicle and then shot at close quarters. All died (brain death) instantaneously with no disturbance and blood was then taken as soon as possible.
2. Herded, shot, sampled. These animals were herded by the helicopter for variable times (Table 1) and then shot with a rifle from the helicopter. No SDC was used. Most animals died instantaneously (brain death) and blood was taken as soon as possible thereafter.
3. Undisturbed, SDC, sampled. Individual animals were approached carefully with a vehicle and then given the culling dose of SDC (see below). They were only minimally disturbed by the darting procedure and were observed until they collapsed. Blood was thereafter taken as soon as possible.
4. Herded, SDC, sampled. These animals were herded by the helicopter and darted from it in the usual culling procedure. Blood sampling commenced as soon as the last animals in the group collapsed. (Buffaloes are not normally shot as part of the culling procedure). The darts were numbered and times from darting to blood sampling noted (Table 1).

No respiratory movements were evident at the time of sampling in any of the buffaloes studied. Except for 1 or 2 animals in Group 4, cardiac arrest had not occurred by the time blood samples were taken. Blood samples were obtained after rapidly exposing the carotid artery and external jugular vein. Buffaloes

Table 2: BLOOD GAS, ACID-BASE BALANCE, HAEMATOCRIT AND GLUCOSE VALUES OF THE DIFFERENT GROUPS

Groups		pH	P_{CO_2} mmHg	P_{O_2} mmHg	Lactate mmol/ℓ	Base excess ECF mmol/ℓ	Haematocrit %	Glucose mmol/ℓ
Elephant								
1 Undisturbed, shot, sampled	$\bar{x}$	7,29	50,3	44,3	1,3	−3	39,3	4,16
	SD	0,04	6,9	18,4	1,3	2	6,1	0,66
2 Herded, shot, sampled	$\bar{x}$	7,07	63,3	34,3	9,4	−11	47,7	5,07
	SD	0,08	19,5	17,3	2,6	6	4,8	1,02
3 Undisturbed, SDC, sampled	$\bar{x}$	7,12	66,2	45,0	5,5	−9	45,4	5,10
	SD	0,12	15,2	23,6	1,1	5	2,1	0,87
4 Herded, SDC, sampled	$\bar{x}$	7,01	81,4	29,3	9,4	−11	46,7	7,97
	SD	0,13	23,3	19,5	1,9	4	4,2	2,35
5 Herded, SDC, shot, sampled	$\bar{x}$	7,05	77,8	33,8	8,1	−11	45,8	5,67
	SD	0,07	16,6	14,8	1,9	6	2,2	1,21
6 Herded, SDC delayed sampling	$\bar{x}$	6,87	108,3	14,7	11,2	−13	46,7	8,72
	SD	0,03	6,5	10,1	2,6	1	8,1	1,59
Buffalo								
1 Undisturbed, shot, sampled	$\bar{x}$	7,30	56,2	19,0	1,4	2	34,0	3,98
	SD	0,05	2,3	5,3	1,8	3	2,7	1,17
2 Herded, shot, sampled	$\bar{x}$	7,02	76,3	23,5	11,6	−10	38,3	5,28
	SD	0,11	18,3	7,5	2,9	2	3,3	1,45
3 Undisturbed, SDC, sampled	$\bar{x}$	7,23	67,8	19,6	3,3	0	40,3	6,07
	SD	0,09	11,6	5,8	1,2	3	4,0	1,69
4 Herded, SDC, sampled	$\bar{x}$	7,03	70,4	21,2	10,8	−12	38,0	6,86
	SD	0,06	7,9	5,5	1,8	3	6,5	1,43

received from 1 to 4 darts depending on their size. Each dart contained 2,0 ml SDC at a concentration of 56 g/100 ml water.

Analytical Techniques

The following blood samples were obtained from each animal and processed as indicated. Results were compared statistically using the two-tailed Student's *t* test.

1. Heparinized arterial blood. This was collected anaerobically in glass syringes whose dead space was filled with heparin solution (5 000 U/ml Pularin) and immediately placed on ice. pH, P_{O_2} and P_{CO_2} were measured within 2 to 3 hours using a Radiometer PHM71 analyser and BMS 3 MK2 Blood micro system. Base-excess was calculated from the Siggaard-Andersen nomogram. The haematocrit was determined by centrifugation.
2. Heparinized venous blood (1 000 U/ml). These samples were immediately centrifuged for 10 minutes at 3 000 *g* and the separated plasma placed on ice for transportation to the laboratory where it was divided into 2 portions.
   One portion of each sample was deep frozen and stored at − 70 °C for up to 10 days. When all frozen samples had been collected they were assayed for ACTH (Immunoassay kit, code IM.66, Amersham), cortisol (Amerlex radio-immunoassay kit, code IM.2021, Amersham), total T_3 and T_4 (Amerlex radio-immunoassay kits, codes IM. 200/2001 and IM. 2010/2011, Amersham) and TSH (Amerlex radio-immunoassay kit, code IM. 2060/61/64, Amersham). All samples were counted with a portable Ekcoγ − counter type N 664A.
   The other portion was immediately analysed for sodium and potassium concentrations (Radiometer FLM3 flame photometer), chloride concentration (Radiometer CMT 10 chloride titrator), blood glucose concentration (GOD-Perid colorimetric test kit cat. no. 124028, Boehringer Mannheim), total protein (Biuret method using bovine serum albumin as standard), colloid osmotic pressure (electronic colloid osmometer after Prather et al.[11] using Amicon PM-10 semipermeable membranes) and total lipid (colorimetric test kit cat. no 124303, Boehringer Mannheim). Lipid and protein electrophoresis was carried out and albumin/globulin ratios calculated using Gelman electrophoresis equipment and a Beckman scanner equiped with an integrator.
3. Venous blood plasma obtained by mixing 80 μl fluoride/EDTA reagent (Cat. no. 243710, Boehringer Mannheim) with 5 ml blood and centrifuging immediately for 10 min at 3 000 *g*. The plasma was placed on ice and analysed for lactate concentration (enzymatic UV-method cat. no. 256773, Boehringer Mannheim).
4. Venous blood plasma containing 4 mmol/l reduced glutathione and 5 mmol/l EGTA obtained after centrifugation as in 3. above. These samples were transported to the laboratory on ice and analysed for total catecholamine concentration according to the radio-enzymatic technique of Callingham and Barrand[6]. Samples were counted with a portable β-scintillation counter (Ecko type N664A).

RESULTS

Data concerning the animals included in the various groups and the results obtained are displayed in Tables

Table 3: ELECTROLYTE, PROTEIN, OSMOLALITY, COLLOID OSMOTIC PRESSURE AND LIPID VALUES OF THE DIFFERENT GROUPS

Groups		Na mmol/ℓ	K mmol/ℓ	Cl mmol/ℓ	Total Protein g/ℓ	Osmolality mosm/kg	C.O.P. mmHg	Total Lipid mg/100 mℓ
Elephant								
1 Undisturbed, shot, sampled	$\bar{x}$	125,4	4,3	84,6	99	243,0	31,1	234,4
	SD	4,6	0,5	2,9	3	11,3	1,9	41,7
2 Herded, shot, sampled	$\bar{x}$	129,3	5,4	88,0	106	271,3	32,4	276,7
	SD	3,0	0,4	3,5	6	6,6	1,8	63,8
3 Undisturbed, SDC, sampled	$\bar{x}$	123,5	4,3	84,6	117	252,5	36,2	318,6
	SD	3,2	0,4	3,1	23	5,9	6,4	59,1
4 Herded, SDC, sampled	$\bar{x}$	126,0	5,4	88,4	105	267,4	35,4	338,1
	SD	2,5	0,9	4,0	8	7,2	3,9	37,1
5 Herded, SDC, shot, sampled	$\bar{x}$	126,2	4,9	90,6	101	268,2	29,8	387,8
	SD	2,7	0,4	1,8	8	7,1	0,8	49,7
6 Herded, SDC delayed sampling	$\bar{x}$	123,9	6,0	85,7	99	268,0	36,0	337,7
	SD	3,9	0,7	5,3	5	7,0	3,0	48,7
Buffalo								
1 Undisturbed, shot, sampled	$\bar{x}$	132,4	4,4	104,2	96	263,2	23,6	272,0
	SD	3,9	1,2	6,6	9	3,6	2,3	72,6
2 Herded, shot, sampled	$\bar{x}$	138,5	6,5	98,9	103	287,1	21,2	358,4
	SD	3,5	1,1	2,5	12	9,7	1,9	88,4
3 Undisturbed, SDC, sampled	$\bar{x}$	132,4	8,2	93,0	96	266,3	23,9	379,8
	SD	2,7	1,8	4,8	7	7,1	10,5	79,3
4 Herded, SDC, sampled	$\bar{x}$	136,4	7,3	97,0	98	288,6	23,0	451,7
	SD	3,4	0,4	2,2	4	13,1	1,6	65,3

Table 4: LIPID FRACTIONS, PERCENTAGE COMPOSITION AND PROTEIN FRACTIONS OF THE DIFFERENT GROUPS

Groups		Lipid Fractions %		Lipid percentage composition mg/100 mℓ		Total Protein Fractions %				
		1	2	1	2	Alb	$\alpha 1$	$\alpha 2$	β	γ
Elephant										
1 Undisturbed, shot, sampled	$\bar{x}$	68,8	31,2	162,4	72,0	38,2	9,4	11,0	12,4	35,0
	SD	9,2	9,2	40,9	20,6	6,2	0,9	0,7	3,0	4,3
2 Herded, shot, sampled	$\bar{x}$	69,9	30,3	196,7	79,9	41,6	8,0	9,9	12,9	27,4
	SD	10,0	9,9	69,2	16,3	2,0	0,1	0,9	1,8	2,4
3 Undisturbed, SDC, sampled	$\bar{x}$	70,0	30,0	224,6	94,1	36,0	7,7	10,0	10,0	31,0
	SD	7,6	7,6	57,9	21,7	4,5	1,4	1,3	2,9	5,3
4 Herded, SDC, sampled	$\bar{x}$	72,0	28,3	247,0	93,0	41,0	8,0	11,0	13,4	27,5
	SD	8,6	8,6	49,8	23,7	4,0	1,1	1,4	1,4	3,2
5 Herded, SDC, shot, sampled	$\bar{x}$	77,2	22,8	301,8	86,7	44,2	9,0	9,8	10,6	26,6
	SD	5,5	5,5	56,9	10,3	7,3	2,4	1,3	2,9	3,2
6 Herded, SDC, delayed sampling	$\bar{x}$	65,3	34,9	177,2	117,1	40,0	8,0	10,0	13,0	29,0
	SD	10,9	10,9	84,8	38,2	2,0	1,0	1,0	2,0	3,0
Buffalo										
1 Undisturbed, shot, sampled	$\bar{x}$	26,8	73,2	75,8	196,2	33,4	—	15,2	11,6	43,2
	SD	8,2	8,2	35,8	46,5	3,4	—	1,5	4,0	2,8
2 Herded, shot, sampled	$\bar{x}$	35,9	64,1	127,2	233,9	32,7	—	15,5	8,5	43,4
	SD	4,8	4,8	18,2	72,1	4,3	—	1,0	1,0	4,3
3 Undisturbed, SDC, sampled	$\bar{x}$	32,8	66,6	122,4	255,8	32,6	—	15,3	7,3	44,6
	SD	5,5	4,9	19,2	67,9	3,7	—	2,2	1,0	3,8
4 Herded, SDC, sampled	$\bar{x}$	36,6	63,4	163,6	287,9	39,2	—	14,4	7,6	39,4
	SD	3,8	3,8	23,5	51,8	8,5	—	2,2	0,9	5,5

1-7 (means ± SD).

Blood gas and acid-base balance values in normal undisturbed elephant and buffalo populations are unknown. By acccepted normal human limites of pH 7.36 and P_{CO_2} 46 mmHg, all animals in all groups can be said to exhibit acute hypercapnia and acidaemia with a small base excess or a base deficit. Similarly they were hypoxic. For the purpose of statistical evaluation we have used the values found in Group 1 of each species as control levels; these animals were shot after minimal disturbance. Compared with these levels all the other groups showed increased hydrogen ion and lactate concentrations with raised P_{CO_2} values. The hydrogen ion and lactate values in elephant in Groups 2-6 were significantly greater than the control ($P<0,01$). P_{CO_2} values in elephants were significantly higher than controls in Groups 4 and 5 ($P<0,01$) and 6 ($P<0,001$). The hydrogen ion and lactate values in buffaloes were significantly higher than controls in Groups 2 and 4 ($P<0,01$), and for P_{CO_2} in Group 2 ($P<0,05$) and Group 4 ($P<0,01$). The animals of both species in Group 3 which were minimally disturbed before being darted showed least variation from the control groups. Only elephant in Group 6 had P_{O_2} values significantly lower than controls ($P<0,01$).

The haematrocrit values (Table 2) indicate a trend towards haemoconcentration in all groups, the differences being significant (P between $<0,02$ and $<0,01$) except for elephant Group 6 and buffalo Group 4. In addition, osmolality values (Table 3) were significantly increased ($P<0,01$) in the herded groups (elephant 2, 4, 5 and 6, buffalo 2 and 4) as were the plasma glucose concentration (Table 2) in elephant Groups 4 and 6 ($P<0,01$) and 5 ($P<0,05$) and buffalo Groups 3 ($P<0,05$) and 4 ($P<0,01$). Plasma potassium concentration (Table 3) increased significantly (P between $<0,02$ and $<0,01$) in the herded groups in both species except elephant Group 5. The values for total plasma protein and sodium were unchanged whilst the plasma lipid concentration was raised in all groups; the increase being significant (P between $<0,05$ and $<0,001$) except for Groups 2 of each species. Chloride concentration decreased significantly in buffalo Groups 2 ($P<0,01$), 3 ($P<0,01$) and 4 ($P<0,05$) (Table 3). Colloid osmotic pressure increased significantly in elephant Groups 4 ($P<0,05$) and 6 ($P<0,01$) (Table 3). Table 4 shows that the lipid electrophoretic pattern and percentage composition in all groups of both species was not different from the controls. Similarly, the plasma protein fractions, their percentage composition and the albumin/globulin ratios were also not different (Tables 4 & 5).

Table 6 shows the results obtained for the hormonal composition of blood. In the case of TSH no significant differences from control values were evident except in the case of Group 6 elephants (decrease, $P<0,01$) and Group 3 buffalo (increase, $P<0,01$). In buffaloes, T_3 concentrations did not differ amongst the groups but in elephants a general increase in mean concentrations was observed whenever the animals were exposed to SDC with the values for elephant Groups 4 and 5 being significant ($P<0,01$). Also, T_4 concentrations did not differ from control values in buffaloes but in elephants Groups 2 and 3 ($P<0,05$) and 4 ($P<0,02$) the increases were significant. The results for ACTH and cortisol concentrations were the same for elephants and buffaloes in that only a combination of exercise (herding) and SDC resulted in significantly increases in concentration (P between $<0,05$ and $<0,001$). Animals which had died from the effects of SDC (both elephants and buffaloes) showed the highest values for ACTH. The catecholamine results indicate that undisturbed elephants and buffaloes killed by SDC had lower concentrations than both undisturbed and herded, shot animals (significant in the case of elephants: $P<0,001$) and that culled elephants which died of SDC had significantly lower values than culled elephants both before and after the brain shot ($P<0,05$). Although these results may be explained (see Discussion), they are probably artefactual because, in our experience, the radioenzymatic technique used is fraught with technical difficulties and open to significant experimental error.

The variables investigated which differed most amongst the various groups in both elephants and buffaloes are blood pH, P_{CO_2}, P_{O_2}, base excess and the concentrations of lactate, glucose, lipid, ACTH, cortisol, T_3, T_4 and catecholamines. It is to be expected that amongs other factors, the duration of herding and/or exposure to SDC would have an effect on the magnitude of the observed differences. The time ranges involved in the various groups are shown in Table 1. Table 7 shows the mean times involved for each group as well as statistically significant correlation coefficients between total time and the means of the variables mentioned above for the different groups. From this Table it is clear that the concentration of neither T_3 nor T_4 was time dependant in either species, that base excess and glucose, lipid, ACTH and cortisol concentrations in both buffalo and elephants were time dependant and that blood pH, P_{CO_2} and lactate concentrations were time dependant in elephants only. In general, the same was observed when individual results (within the different groups for both elephant and buffalo) were correlated with time. In a further special series of experiments (Group 7), 6 individual undisturbed elephants were darted with SDC and blood samples obtained at regular intervals from as soon as the animals were down until all respiratory and circulatory functions had ceased (up to 35 min in some cases). Blood T_3, T_4, TSH and lipid concentration did not correlate with time, but glucose ($r=0,8271$; $y=0,165X+2,96$), lactate ($r=0,8798$; $y=0,026X+1,13$), cortisol ($r=0,7778$; $y=110X-606$) and ACTH ($r=0,773$; $y=0,73X+9,1$) concentrations did. These results underline the effect of time on the above values in elephant exposed to SDC.

DISCUSSION

There is very little published data on blood constituents of elephants and buffaloes. Values for plasma proteins, serum electrolytes, lipids and cortisol measured in blood from elephants shot in East Africa are similar to the values we have obtained[2 3 10]. The possibility of seasonal variation was indicated in the latter 2 papers although possible circadian variation was not considered. In our series sampling was carried out at the same time of day, over a period of 10 days and in the same geographical area. We collected blood from undisturbed animals, very shortly after they were killed by a bullet in the brain. This blood provided the control values for comparison with values from blood collected in the course of a number of different culling procedures. Blood was readily obtained from the ear vessels

Table 5: PROTEIN PERCENTAGE COMPOSITION AND A/G RATIOS OF THE DIFFERENT GROUPS

Groups		Total Protein Percentage Composition g/ℓ					A/G
		Alb	α1	α2	β	γ	
Elephant							
1 Undisturbed, shot, sampled	$\bar{x}$	39,0	9,5	11,1	13,0	35,0	0,63
	SD	6,2	0,8	0,7	2,9	3,8	0,18
2 Herded, shot, sampled	$\bar{x}$	41,2	7,9	9,8	13,1	27,5	0,71
	SD	3,7	0,9	0,9	2,8	2,7	0,06
3 Undisturbed, SDC, sampled	$\bar{x}$	41,0	9,1	11,0	12,0	35,0	0,57
	SD	4,0	1,9	6,0	3,4	3,9	0,11
4 Herded, SDC, sampled	$\bar{x}$	43,0	8,1	11,5	14,0	29,4	0,70
	SD	4,0	1,0	1,8	1,8	4,8	0,12
5 Herded, SDC, shot, sampled	$\bar{x}$	44,0	9,0	10,0	11,0	27,0	0,72
	SD	4,0	3,0	2,0	4,0	5,0	0,26
6 Herded, SDC, delayed sampling	$\bar{x}$	39,0	8,0	10,0	13,0	29,0	0,67
	SD	1,0	1,0	2,0	2,0	4,0	0,05
Buffalo							
1 Undisturbed, shot, sampled	$\bar{x}$	35,0	—	16,0	12,0	45,0	0,51
	SD	4,0		2,0	4,0	5,0	0,07
2 Herded, shot, sampled	$\bar{x}$	33,5	—	16,0	8,6	44,0	0,48
	SD	7,6	—	2,4	1,5	4,1	0,08
3 Undisturbed, SDC, sampled	$\bar{x}$	37,0	—	17,0	8,0	51,0	0,49
	SD	6,0	—	3,0	3,0	11,0	0,08
4 Herded, SDC, sampled	$\bar{x}$	38,5	—	14,0	7,4	38,9	0,67
	SD	8,0	—	2,0	1,0	6,2	0,28

Table 6: HORMONAL COMPOSITION OF BLOOD OF THE DIFFERENT GROUPS

Groups		TSH μU/mℓ	T_3 nmol/ℓ	T_4 nmol/ℓ	ACTH pg/mℓ	Cortisol nmol/ℓ	Catechola-mines mg/ℓ
Elephant							
1 Undisturbed, shot, sampled	$\bar{x}$	3,57	1,94	70,2	17,3	111,38	271,5
	SD	0,77	0,49	42,2	1,4	24,75	42,3
2 Herded, shot, sampled	$\bar{x}$	3,62	1,87	114,0	16,3	132,28	327,8
	SD	0,53	0,62	8,8	2,6	26,95	78,2
3 Undisturbed, SDC, sampled	$\bar{x}$	3,21	3,08	116,6	13,3	125,40	158,5
	SD	0,76	1,76	17,0	3,2	104,50	25,8
4 Herded, SDC sampled	$\bar{x}$	3,11	3,07	111,9	23,2	858,00	326,1
	SD	0,73	0,75	29,3	5,1	283,25	64,2
5 Herded, shot, SDC, sampled	$\bar{x}$	3,36	3,64	98,8	29,4	687,50	355,7
	SD	1,18	1,02	40,6	5,4	269,50	68,7
6 Herded, SDC, delayed sampling	$\bar{x}$	2,10	2,80	97,8	39,3	501,88	217,6
	SD	0,43	0,85	35,2	6,4	144,93	28,2
Buffalo							
1 Undisturbed, shot, sampled	$\bar{x}$	2,22	1,00	6,8	32,8	23,65	351,4
	SD	1,31	0,58	10,4	15,3	21,45	71,2
2 Herded, shot, sampled	$\bar{x}$	2,50	1,00	10,0	27,4	16,23	368,5
	SD	1,58	0,45	6,7	13,0	20,08	—
3 Undisturbed, SDC, sampled	$\bar{x}$	4,42	1,46	14,8	63,1	33,55	303,3
	SD	0,72	1,68	17,6	26,5	31,35	77,7
4 Herded, SDC sampled	$\bar{x}$	2,53	1,32	3,7	155,5	115,50	—
	SD	0,55	0,30	5,7	41,5	46,75	—

Table 7: THE RELATIONSHIP BETWEEN MEAN TOTAL TIMES AND THE MEANS OF DIFFERENT BLOOD VARIABLES EXPRESSED AS STATISTICALLY SIGNIFICANT CORRELATION COEFFICIENTS ($P<0,01$).

Groups	Mean Total Time* min	Mean SDC Time** min	pH	PO_2	P_{CO_2}	Base excess	Lactate	Glucose	Lipid	ACTH	Cortisol	T_3	T_4
Elephant													
1	2,2 ± 0,5	—											
2	10,7 ± 5,0	—											
3	10,8 ± 5,7	—											
4	33,9 ± 7,4	19,3 ± 5,0	−0,894	−0,879	0,944	0,988	0,786	0,903	0,794	0,900	0,824	—	—
5	33,4 ± 9,8	19,5 ± 10,8											
6	45,1 ± 10,0	32,0 ± 8,7											
Buffalo													
1	4,4 ± 2,6	—											
2	8,7 ± 4,8	—	—	—	—	0,849	—	0,933	0,948	0,968	0,938	—	—
3	14,0 ± 1,8	—											
4	26,2 ± 2,6	10,4 ± 1,8											

* Total time – from the time of the shot or SDC dart in undisturbed animals or the start of herding in the other groups until blood sampling was complete

** SDC time – from the time of SDC dart in herded animals until blood sampling was complete

in elephants but in buffaloes it was necessary to cut down to the carotid and jugular vessels; this lengthened the sampling time slightly.

With the exception of elephants killed by SDC alone, all animals had some degree of circulatory function when blood samples were taken and in elephant Groups 3, 4 and 5 some respiratory function also persisted. Although our Group 1 control animals died immediately from a shot into the brain, so that respiration ceased, the maintained circulation has implications for our base line values. Apart from its effect on blood gas and hydrogen ion values, the progressive asphyxia may cause peripheral and central stimulation of autonomic pathways leading to catecholamine release from the adrenal glands. However, this will depend upon the remaining integrity of the medulla oblongata although it is also probable that the percussive integrity shock to the reticular formation would depress medullary function. The shot into the brain may also result in a massive sympathetic discharge which would increase blood catecholamine levels[7] and it is possible that some degree of pituitary function could persist. If there is release of catecholamines this could in turn cause an increase in plasma glucose and lactate through increased glycogenolysis and possibly increased potassium by an effect on membrane permeability[8]. Gericke et al.[7] have noted increases in heamatocrit and glucose, lactate, and potassium concentrations in Springbok similarly shot, compared with the values from conscious tranquilised animals. However, at present there are no normal resting values from conscious elephants and buffaloes to provide a better basis for comparison than our Group 1 values.

The raised P_{CO_2} and lowered P_{O_2} values in arterial blood from all our animals is indicative of acute respiratory insufficiency. The immediate consequence of this is the development of a combined respiratory and metabolic acidosis with increased blood lactate concentrations. The hypoxic metabolic acidosis was exacerbated by muscular activity and there was no possibility of renal compensation during the short time interval with which we are concerned. The values of P_{CO_2} and pH in our control samples are only slightly outside normal human limits and the hypercapnia is attributable to asphyxia before sampling. The longer sampling delay in buffaloes would also account for the lower P_{O_2} values we obtained in this species. It is apparent therefore that SDC alone (Group 3) causes a hypercapnia and acidemia and that the muscular effort associated with herding in the other groups (which in itself is probably experienced as stress because the animals are not accustomed to prolonged running) exacerbates these states. Herding also causes a lower P_{O_2} in elephant but the low value in buffalo is little affected. The elephants in Group 6, where sampling was delayed, had the highest CO_2 and hydrogen ion concentrations and the lowest P_{O_2}. The relationship between the magnitude of the change and the time interval from the commencement of the group procedure to the eventual blood sample is apparent in Table 7.

The hormone measurements were all made using radioimmunoassay kits designed for human hormones. The degree of cross-reactivity of elephant and buffalo material with anti-human antibodies is unknown so that our results can only be used to indicate relative differences between groups; they may not indicate absolute hormone levels in these species. Varying ages of animals within groups may also affect our values. In the case of buffaloes, zero values for T_3, T_4 and cortisol were occasionally recorded and this explains the large standard deviations (greater than mean values) reported.

As cortisol and thyroid hormone concentrations are regulated by hypothalamic releasing factors, variations in hypothalamic and pituitary function will affect plasma levels. The degree to which these regions are damaged and their residual circulation (as discussed above) may influence values in brain shot animals. There was no difference between ACTH and cortisol levels in either elephants or buffaloes killed by a shot into the brain, undisturbed (Group 1) or herded (Group 2). However, elephants which were herded, given SDC and eventually shot (Group 5) had significantly higher levels of both ACTH and cortisol. Elephants in Groups 4 and 6 which were not shot before sampling, also had significantly higher levels of ACTH and cortisol. The common factors in Groups 4, 5 and 6 are the forced exercise during herding and SDC suggesting a relationship between activity and raised concentrations of these hormones. Thurley & McNatty[13] reported increased cortisol levels in unrestricted ewes subjected to exercise. Van Heerden & Bertschinger[14] showed an increase in cortisol in response to stress in the black-backed jackal and the same has been reported for pigs[9]. In man the increased secretion rate of cortisol in stress situations of all kinds is well documented. It is possible that the stress of forced exercise in elephants is compounded by the effects of

the subsequent SDC and causes the higher levels we have observed. In buffaloes, SDC alone (Group 3) caused an increase in ACTH and a non significant increase in cortisol but SDC in combination with forced exercise (Group 4) resulted in a significant increase in both hormones.

In man, rhesus monkey and guinea pig, thyroid function appears to be stimulated by stress but in a number of other species thyroid response is complicated by concommitant corticosteroid activity. There was no trend apparent in TSH values here. In elephants treated with SDC alone or after herding (Groups 4 and 5) there was a significant increase in T_3 levels. There was also an increase in T_4 levels after herding and/or SDC administration (Groups 2, 3, 4, 5 and 6) but a large variation in the control (Group 1) values prevents the estimation of significance. In buffaloes there were no significant changes in either T_3 or T_4 levels in any groups. If it is accepted that the shot into the brain influenced catecholamine concentrations then the values obtained in Group 3 animals probably reflect an increase above normal levels. Apart from this, we are unable to draw any conclusions from the determinations; the values in all groups being extremely high.

Bearing in mind the many unknown factors to which we have drawn attention, we consider that the changes we have measured in the levels of certain blood components can be attributed to stress induced by all the culling procedures which involve darting with SDC. Individual animals differ in their response as is indicated by the large standard deviations. In general it would seem the greater the activity induced by herding, the greater the resultant stress. It is an observation that the respiratory system in buffaloes is more susceptible to paralysis induced by SDC at the high dose-level used than is the case in elephants. The resultant asphyxia is more acute as indicated by the lower P_{O_2} values.

The persistence of circulatory function complicates the interpretation of observed changes if we attempt to link stress and its conscious appreciation. Asphyxial stress produces blood changes in conscious and anaesthetized animals alike, although the changes may be greater in the conscious animals as Button & Mülders found[5]. When the brain is destroyed by a bullet the problem of fear, or the conscious appreciation of stress, does not arise. What is pertinent is the question of what degree of asphyxia is likely to be incompatible with consciousness? Button et al.[4] interpreted EEG changes in an SDC apnoeic calf after 4 minutes, as indicative of cerebral depression, whilst the EEG pattern before this suggested a heightened alertness and stress. The haemoglobin of the African elephant has a half saturation oxygen tension of 22 mmHg[12]. We do not have a value for buffalo haemoglobin but the S_{50} for the *Bos* species is 32 mm Hg[1]. We may assume that, as in other species, cerebral vasodilatation is caused by asphyxia. It is a reasonable conclusion therefore, that at the P_{O_2} values in elephant arterial blood that we have measured after SDC paralysis, the oxygen supply to the elephant brain would be adequate to sustain consciousness for a considerable time if circulation of blood was present. The effects of SDC may thus be perceived for some time after the animals collapse and they should be shot as soon as possible after this, as is indeed the case in the usual culling procedure in the Kruger National Park. Buffaloes on the other hand, would probably lose consciousness far sooner as their respiratory paralysis is more profound, their P_{O_2} is lower, and their S_{50} is higher. As indicated before, they are usually not shot after collapsing when culled. The above would seem to be factors to consider in future in relation to culling techniques.

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EFFICACY OF IVERMECTIN AGAINST INTERNAL PARASITES OF SHEEP

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Ivermectin in an oral formulation was evaluated by the non-parametric method against a wide range of endoparasites of sheep in 15 trials involving a total of 297 sheep. Ivermectin at 200 μg/kg was more than 80 % effective in more than 80 % of the treated animals (i.e. Class A) against induced infestations of 3rd and 4th stage larvae and adults of *Chabertia ovina, Dictyocaulus filaria, Haemonchus contortus* (including a benzimidazole-resistant strain), *Nematodirus spathiger, Oesophagostomum columbianum, Ostertagia circumcincta* (including a benzimidazole-resistant isolate), and *Trichostrongylus colubriformis*; an "A" class was also obtained against the 4th stage larvae and adults of *Gaigeria pachyscelis* and *Strongyloides papillosis* and natural infestations of adult *Trichuris* spp. Efficacy against the 3rd stage larvae of *G. pachyscelis* and *S. papillosus* fell to Class B (i.e more than 60 % effective in more than 60 % of the treated animals). Ivermectin completely eliminated *Oestrus ovis* larvae (all 3 instars).

In 2 additional trials the efficacy of concurrent oral treatment with ivermectin at 200 μg/kg and rafoxanide suspension at 7,5 mg/kg was evaluated against the 3rd stage larvae of *N. spathiger, O. columbianum* and *S. papillosus* and 56 day-old *Fasciola gigantica.* Concurrent medication did not adversely affect the individual efficacies of either ivermectin or rafoxanide.

Keywords: Endoparasites, nematodes, *Fasciola gigantica, Oestrus ovis,* sheep, ivermectin, rafoxanide.

INTRODUCTION

Ivermectin (22, 23-dihydroavermectin B_1) (Ivomec liquid: MSD) has been shown to have a high degree of efficacy against gastrointestinal nematodes[2 4 5 6 8 10], lungworms[4 10] and nasal worm[11] in sheep at very low dose rates. This paper describes a series of 15 South African trials to determine the efficacy by the modified non-parametric method (NPM) of ivermectin against induced infestations of all 3 parasitic stages of 9 nematode species (including benzimidazole-resistant isolates of two species) and natural infestations of *Trichuris* spp. and *Oestrus ovis.* Two further efficacy trials were performed to establish the efficacy of concurrent administration of ivermectin and rafoxanide (Ranide: MSD) against 3 nematode 3rd stage larvae and immature *Fasciola gigantica.*

MATERIALS AND METHODS

Experimental Animals:

The number of experimental animals used in each trial is given in Table 1. A total of 297 South African Mutton Merino lambs, aged 6 to 9 months, was used in 15 trials with induced nematode infestations. These lambs were all reared under conditions which precluded as far as possible previous helminth exposure, but were also treated with either cambendazole (Bonlam: MSD), or thiabendazole (Thibenzole: MSD), or thiabendazole plus rafoxanide (Ranizole: MSD) or levamisole (Ripercol: Janssen), 3 to 24 d before infestations were induced. Two further trials included 10, 5 to 6 year-old SA Mutton Merino ewes, reared under similar conditions to those described above, and 21 Dorper wethers, aged 6 to 12 months, selected on the presence of nasal discharge.

Target parasites:

Ivermectin was tested against induced infestations of all 3 parasitic stages of *Chabertia ovina, Dictyocaulus filaria, Gaigeria pachyscelis, Haemonchus contortus* (including the benzimidazole-resistant Boshof strain[1]), *Nematodirus spathiger, Ostertagia circumcincta* (including the benzimidazole-resistant Swellendam isolate[15]), *Oesophagostomum columbianum, Strongyloides papillosus* and *Trichostrongylus colubriformis* and against a natural infestation of 1st, 2nd and 3rd instars of *Oestrus ovis.*

Concurrent medication of ivermectin and rafoxanide was tested against induced infestations of 3rd stage larvae of *N. spathiger, O. columbianum* and *S. papillosus* and 56 day-old *F. gigantica.*

A summary of parasites included in each trial is contained in Table 1. In addition, natural infestations of *Trichuris* spp. were present in 13 of the trials with induced infestations.

Induced infestations:

Nematode infestations, except the 3rd stage larvae of *D. filaria,* were induced according to the procedures described by Reinecke[13]. For this exception each animal was infested orally with 2 daily doses of 502 or 503 infective 3rd stage larvae of *D. filaria* 1 and 2 d prior to treatment.

In the *F. gigantica* efficacy trial each animal was orally infested with 150 viable metacercariae 56 d prior to treatment.

Treatment:

The treatment schedules employed in the various trials are summarized in Table 1. The sheep in Trials 1 to 15 were treated orally with a single dose of ivermectin 0,08% m/v solution at 200 μg/kg. In trials 16 and 17 the sheep were treated concurrently with ivermectin 0,08 % m/v solution orally at 200 μg/kg and rafoxanide 2,5 % m/v suspension orally at 7,5 mg/kg. Two different formulations of ivermectin, viz propylene glycol and aqueous micelle, were used in different trials.

Larval indicator controls (LIC):

One LIC animal was included for each of the induced nematode trials. On the day of treatment for each trial the LIC animal was slaughtered to confirm the stage of development of the various parasitic nematodes at treatment.

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*** Kiepersol, Hazyview.

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Table 1: SUMMARY OF 17 TRIALS SHOWING THE NUMBER OF ANIMALS USED, THE TARGET PARASITES AND THE INTERVAL BETWEEN TREATMENT AND SLAUGHTER FOR EACH TRIAL

Trial Number+°	Number of animals		Target parasites++	Treatment to slaughter intervals
	Treated	Control		
1	12	7	L_3 *Haemonchus contortus* (H.c); L_3 *Trichostrongylus colubriformis* (T.c); L_3 *Chabertia ovina* (C.o); L_4 *Strongyloides papillosus* (S.p)	34d-37d
2	12	7	L_4 *Nematodirus spathiger* (N.s); *Oesophagostomum columbianum* (O.col); L_4 *Dictyocaulus filaria* (D.f); L_4 H.c; Ad T.c.	23d-25d
3	12	8	Ad *Gaigeria pachyscelis* (G.p); Ad H.c; Ad T.c; Ad O. col; Ad N.s	14d-16d
4	12	8	L_3 *Ostertagia circumcincta (Ost. c); L_3 G.p; L_3 O. col*	34d-36d
5	12	7†	L_4 Ost. c; L_4 C.o; L_4 G.p.	38d-40d
6	12	7	Ad Ost. c; Ad S.p; Ad C.o; Ad D.f	14d-16d
7	12††	8	L_3 H.c; L_3 S.p; L_3 N.s; L_3 O. col	41d-43d
8	14	7	L_1,L_2 and L_3 Oestrus ovis	9d
9	12	7	L_3 Ost. c; L_3 O. col; Ad H.c; Ad N.s	35d-37d
10	12	6	L_3 H.c*; L_3 T.c; L_3 C.o; L_3 G.p; L_3 D.f; L_3 S.p	35d-37d
11	12	6	L_3 T.c; L_3 S.p; L_3 Ost.c**; L_3 C.o; L_3 G.p; L_3 D.f	35d-36d
12	12	7	L_3 D.f; L_4 N.s; L_4 S.p; L_4 C.o; L_4 Ost.c**; L_4 H.c*; Ad G.p	28d-29d
13	12	6	Ad H.c*; Ad Ost.c**; Ad G.p; L_4 S.p; L_4 N.s; L_4 C.o	28d-30d
14	12	6	L_3 N.s; L_3 H.c; L_3 G.p	35d
15	12	6	L_3 N.s; L_3 S.p	21d
16	12	6	L_3 O.col; L_3 N.s; L_3 S.p	34d-35d
17	5	5	56d *Fasciola gigantica*	14d

+ Trials 1-6 Propylene glycol formulation; Trials 7-17 aqueous micelle formulation.
++ L_1 – First larval stage; L_3 – third larval stage; L_4 – fourth larval stage; Ad – adult
°Induced infestations in all trials except Trial 8 in which natural infestations were used
*Boshof benzimidazole – resistant strain** Swellendam benzimidazole – resistant isolate.
† One control died on Day 37 and was excluded from the trial
†† One treated animal died one day after treatment – from pharyngeal trauma

Table 2: EFFICACY OF IVERMECTIN AT 200 µg/kg AGAINST THIRD STAGE LARVAE OF PARASITIC NEMATODES. MEAN WORM BURDENS, PERCENTAGE REDUCTIONS AND NPM CLASS FOR ALL TRIALS

Third larval stages	No. of trials	Mean worm burdens[1]		Percentage reductions	NPM class
		Control	Ivermectin		
C. ovina	3	334	4,3	99	A+
D. filaria	3	117,1	1,1	>99	A
G. pachyscelis	4	59,7	3,9	93	B†*
H. contortus	3	1392	11,1	>99	A
H. contortus[2]	1	123	6,7	>99	A
N. spathiger	3	1507	1,6	>99	A
O. columbianum	3	443,9	32,8	93	A*
O. circumcincta	2	1537	3,1	>99	A
O. circumcincta[3]	1	1390	5,2	>99	A
S. papillosus	4	942	27,3	97	B
T. colubriformis	3	1799	2,6	>99	A

1 Geometric means after transformation to natural logarithms, with or without substitution of 0,25 for zero counts in an aliquot
2 Boshof benzimidazole-resistant strain
3 Swellendam benzimidazole-resistant isolate
+ A = More than 80 % effective in more than 80 % of treated sheep
† B = More than 60 % effective in more than 60 % of treated sheep
* Apparent anomaly in claims arises because percentage reduction is based on geometric means and NPM claims on median parasite count of the control animals.

Necropsy and worm recovery:

The intervals between treatment and slaughter of animals for each trial is given in Table 1. For the greater part worm recovery procedures were conducted according to the methods described by Reinecke[13].

To facilitate recovery of parasites, the gastro-intestinal ingesta were processed using the following variations of technique: the modified Baermann apparatus was used for incubations in some instances whereas in other cases no incubations were done; washing was done either on a 37, 63 or 150 µm aperture sieve; digests of the mucosa were not always done. Variations in technique depended upon the parasitic nematode species and stage of development which was being recovered.

For lungworm the trachea and bronchi were opened and all visible worms removed. The trachea and lungs were then washed over a 37 µm aperture sieve whereafter the lung tissue was cut into blocks and incubated in physiological saline at 37-40°C for 3-4 h. The lung

Table 3: EFFICACY OF IVERMECTIN AT 200 μg/kg AGAINST FOURTH STAGE LARVAE OF PARASITIC NEMATODES. MEAN WORM BURDENS, PERCENTAGE REDUCTIONS AND NPM CLASS FOR ALL TRIALS

Fourth larval stages	No. of trials	Mean worm burdens[1]		Percentage reductions	NPM class
		Control	Ivermectin		
C. ovina	3	248	0,1	>99	A+
D. filaria	1	71	0,5	>99	A
G. pachyscelis	1	171,3	2,6	99	A
H. contortus	1	1150,7	13,3	98	A
H. contortus[2]	1	119	1,3	99	A
N. spathiger	3	523	0,1	>99	A
O. columbianum	1	461,2	2,8	>99	A
O. circumcincta	1	1168,3	2,8	>99	A
O. circumcincta[3]	1	64,9	0	100	A
S. papillosus	3	1202	3,8	>99	A
T. colubriformis	1	1573,8	2,7	>99	A

[1] Geometric means after transformation to natural logarithms, with or without substitution of 0,25 for zero counts in an aliquot.
[2] Boshof benzimidazole-resistant strain.
[3] Swellendam benzimidazole-resistant isolate
+ A = More than 80 % effective in more than 80 % of treated sheep

Table 4: EFFICACY OF IVERMECTIN AT 200 μg/kg AGAINST ADULT PARASITIC NEMATODES. MEAN WORM BURDENS, PERCENTAGE REDUCTIONS AND NPM CLASS FOR ALL TRIALS

Third larval stages	No. of trials	Mean worm burdens[1]		Percentage reductions	NPM class
		Control	Ivermectin		
C. ovina	1	403	2,5	>99	A+
D. filaria	1	156,1	0,3	>99	A
G. pachyscelis	3	74,9	0,4	>99	A
H. contortus	2	1659	3,6	>99	A
H. contortus[2]	1	77,3	0,1	>99	A
*N. spathiger**	2	783	1,9	>99	A
O. columbianum	1	500,9	2,9	>99	A
O. circumcincta	1	1677,5	5,6	>99	A
O. circumcincta[3]	1	2358	0,2	>99	A
S. papillosus	1	1209,3	4,9	>99	A
T. colubriformis	1	2177	3,5	>99	A
Trichuris spp.[4]	13	5,5	0,3	95	A

[1] Geometric means after transformation to natural logarithms, with or without substitution of 0,25 for zero counts in an aliquot.
[2] Boshof benzimidazole-resistant strain
[3] Swellendam benzimidazole-resistant isolate
[4] Natural infestations
* At slaughter 40,9 % of worms recovered from control animals in one of the two trials (Trial 3) were in the fourth larval stage. The remaining worms were adult
+ A = More than 80 % effective in more than 80 % of treated sheep

pieces were then removed, thoroughly washed and the worms collected by sieving the washings and saline through a 37 μm aperture sieve.

In Trial 15 the liver of each animal was collected and the gall bladder and bile ducts opened. All visible parasites were recovered and the liver was then cut into slices approximately 5 mm thick. Pressure was exerted on the exposed edges to extrude all parasites still remaining. The liver slices were then placed on a coarse sieve in a pan and incubated in physiological saline at 37°C for 3 h. Thereafter the slices were thoroughly washed and the worms collected by sieving the washings and saline suspension from the pan through a 150 μm aperture sieve.

The nasal cavity (turbinates, septum and ethmoid bone), the frontal and maxillary sinuses and the cornual cavity of sheep from Trial 8 were examined for *O. ovis* parasites.

Statistical analysis:

The gastrointestinal parasite burdens were ranked for each species and treatment group for determination of NPM claims[13].

In addition, geometric means and percentage reductions were calculated for all parasites. The geometric means were calculated after transformation to natural logarithms, with or without substitution of 0,25 for zero counts in an aliquot.

RESULTS

Nematode infestations:

Mean worm burdens for all trials for each parasite and different treatment groups, percentage reductions and non-parametric efficacy claims for the different stages

of development appear in Tables 2 to 4 for nematode parasites.

The percentage reductions of the mean worm burdens of ivermectin treated animals relative to the control animals were 99 % and more for all stages of the induced infestations with the exception of 3rd stage larvae of *G. pachyscelis* (93 %), *O. columbianum* (93 %), *S. papillosus* (97 %) and 4th stage larvae of *H. contortus* (98 %).

All 3 parasitic stages of benzimidazole-resisant *H. contortus* and *O. circumcincta* were also reduced by 99 % and more.

For 13 trials the mean burden of naturally acquired adult *Trichuris* spp. was reduced by 95 % in ivermectin treated animals when compared with the control animals. Small numbers of adult *Trichuris* (a mean of approximately 5,5 worms per animal) were encountered in the control animals.

With the exception of the 3 rd stage larvae of *G. pachyscelis* and *S. papillosus* for which only a Class B (> 60 % reduction in > 60 % of sheep) were achieved, the reduction of all other induced infestations (species and development stages) and naturally acquired adult *Trichuris* spp. rose to Class A (> 80 % reduction in > 80 % of sheep) by the NPM.

Two formulations of ivermectin, viz an aqueous micelle formulation and a propylene glycol formulation were used in the trials (Table 1). Without exception both formulations showed comparable efficacy when evaluated against the same target parasite.

Table 5: EFFICACY OF IVERMECTIN AT 200 µg/kg AGAINST *O. ovis*

Larval stage	*Mean*[1] number of *O. ovis*		Percentage reduction
	Control	Ivermectin	
1st Instar	3,2	0	100
2nd Instar	4,3	0	100
3rd Instar	5,8	0	100

[1] Geometric means after transformation to natural logarithms

Oestrus ovis:

The efficacy of ivermectin at 200 µg/kg against *O. ovis* is summarized in Table 5. Ivermectin completely eliminated all larval stages of *O. ovis*.

Concurrent medication with rafoxanide:

The results of concurrent medication of ivermectin and rafoxanide against the 3rd stage larvae of *N. spathiger, O. columbianum* and *S. papillosus* and 56 d old *F. gigantica* appear in Table 6. The mean percentage reductions were > 99 % for all nematode parasites, which qualified for Class A by the NPM; and 97 % for 56 day-old *F. gigantica*.

DISCUSSION

The trials reported in this paper are the first in which the efficacy of ivermectin has been tested by the modified NPM in sheep. The results demonstrate the high efficacy of ivermectin at 200 µg/kg against both larval and adult stages of gastrointestinal and pulmonary nematodes and *O. ovis* in sheep and confirm overseas reports by other workers[4 5 6 10 11]. It is interesting that the lowest efficacy was obtained against 3rd stage larvae of *G. pachyscelis* and *S. papillosus*, both of which infest the host percutaneously.

An apparent anomaly exists between the mean percentage reduction and the class by the NPM of the 3rd stage larvae of *O. columbianum* and *G. pachyscelis* (Table 2). This arises because percentage reductions are calculated from geometric means and NPM claims are based on the median parasite counts of the control animals.

Small numbers of naturally infested adult *Trichuris* spp. occurred in most trials. A combined analysis of the results of all trials gave an "A" efficacy claim by the NPM. Efficacy of ivermectin against immature *Trichuris* spp. has been observed (Leaning, W.H.D.L., 1981 unpublished data) and has not been evaluated locally.

The biochemical mode of action of ivermectin is unique and differs from any currently available parasiticides[2]. It paralyses parasitic nematodes, arachnids and insects by stimulating the release of the inhibitory neurotransmitter *gamma* amino butyric acid (GABA) from the presynaptic nerve terminals as well as by potentiating GABA binding to the post synaptic receptor sites[7 9 12]. Therefore it is unlikely that cross resistance against benzimidazole-resistant nematode strains will occur. This is illustrated by the high efficacy of ivermectin against benzimidazole-resistant isolates of *H. contortus* (Boshof) and *O. circumcincta* (Swellendam) shown in the present trials. Other workers have also reported a high degree of efficacy against benzimi-

Table 6: EFFICACY OF IVERMECTIN AT 200 µg/kg AND RAFOXANIDE AT 7,5 mg/kg CONCURRENT TREATMENT AGAINST SOME THIRD STAGE LARVAL NEMATODES (L3) AND IMMATURE *F. gigantica*

Parasite	Mean worm burdens[1]		Percentage reduction	NPM class
	Control	Ivermectin/ Rafoxanide		
Trial No. 16				
O. columbianum (L3)	173,4	1	99	A+
N. spathiger (L3)	1198,8	0	100	A
S. papillosus (L3)	1276,5	1,9	99	A
Trial No. 17				
56d F. gigantica	73,4	1,6	97	—

+A = More than 80 % effective in more than 80 % of treated sheep.
[1] Geometric means after transformation to natural logarithms.

dazole-resistant strains of *H. contortus, O. circumcincta* and *T. colubriformis*[3 5]. This property could find significant practical application in dosing programmes for sheep in South Africa where at the moment there are at least 4 confirmed field isolates of benzimidazole-resistant *H. contortus*[14] and 2 isolates of *O. circumcincta*[15].

Ivermectin is not effective against trematodes and cestodes, which appear not to use GABA as neurotransmitter[4 9]. Since sheep in liver fluke enzootic areas are often treated for both liver fluke and nematodes at the same time, the efficacy of concurrent medication of ivermectin and rafoxanide given orally was also determined. The parasitic 3rd stage larvae of *G. pachyscelis, N. spathiger* and *S. papillosus* were selected to evaluate ivermectin efficacy and immature *F. gigantica* (56 day-old) was selected for rafoxanide evaluation. Concurrent medication did not result in any reduction in the individual efficacy of either compound against these parasitic stages tested.

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BOOK REVIEW **BOEKRESENSIE**

VETERINARY HELMINTHOLOGY

R.K. REINECKE

First Edition. Butterworth Publishers, Durban 1983 pp 392 Figs 118 Tables 17 and 8 plates comprising 48 colour photographs Price R55 ISBN 0 409 11262 3

For 50 years students of veterinary helminthology, not only in South Africa, but in many other countries as well, have used the contemporary edition of Mönnig's *Veterinary Helminthology and Entomology* as their standard reference. This work has its roots in South Africa. Considerable advances have been made since the first edition was published in 1934 and, although excellent textbooks (for example those by Soulsby and Dunn) have augmented the updated editions of Mönnig's work for other areas of the world, most new local data pertinent to this essential department of South African science have remained inaccessible to the professions – virtually all developments have been bound in lonely library volumes, filed in dusty collections of reprints and theses or, even worse, lost with their originators.

In "Veterinary Helminthology" Professor Reinecke has boldly attempted to rectify this deficiency. Using relevant sections from overseas texts, his own extensive student notes and expertise, constant reference to the most recent publications and liberal use of personal communications, he has produced a masterful hybrid which satisfies most of the major requirements for a textbook for South African undergraduates and practitioners. In addition, through comprehensive and carefully selected reference source, a path to more advanced studies is shown.

To understand the author's style of presentation one must examine the Preface where he justifies the inclusion of key words and short cuts as an aid to learning and diagnosis. There are critics who would argue that teaching aids of such kinds will perpetuate the "parrot fashion" style of learning which has historically bedevilled the study of helminthology and, ironically, led to its misunderstanding. Clearly, he is concerned that the reader has rapid access to the information he requires and he has succeeded, unlike some other authors, in producing a book with the fundamental attribute of being readable. But he has done this without losing sight of the necessity for a detailed knowledge of the subject. The book is designed to be constantly and easily used, not scrutinized and memorized and, as such is innovative.

Essential terminology and the basics of classification (also essential) are given in the Introduction. Here the definition of "hypobiosis" is sufficiently vague to reflect our knowledge of the phenomenon, but the term "definitive host" should be expanded to include an asexually-reproducing parasite such as *Strongyloides*. Although it is economically and practically convenient to study the pathogenesis of parasitic disease in artificially infested hosts such results are, at best, only a guide to what may happen in the field. Perhaps, herein, lies one of the greatest deficiencies in our understanding of the problems that confront us in the field.

In Part 1 the diagnosis of parasites, resistance, immunity and chemotherapy are presented. The long-awaited section concerning diagnosis is excellent and provides a simple, practical standard procedure for field and laboratory examinations which includes collection, preservation and examination of worms, eggs or larvae. In addition a working checklist of what may be found in various organ systems is a constant reminder to the examiner of what *not* to overlook. One major deficiency, however, is that nothing is said concerning interpretation of the findings, nor for the greater part does such information appear later in the text. This section would benefit from expansion in a subsequent edition to include estimates of what are acceptable or normally encountered values for parasite burdens, faecal egg counts, etc. All too often we find veterinarians failing to diagnose parasitism on the basis of negative faecal egg counts or, less acceptably, diagnosing parasitic disease having found 5-6 *Oesophagostomum* and 50-60 *Haemonchus* at necropsy. This is such a constant criticism that it needs to be urgently addressed at the level of basic training. The larval differentiation diagrams are invaluable, but for ease of understanding could have been cross-referenced to subsequent places in the text, where explanatory notes are to be found.

It is stated that "in horses and ruminants it is reasonably simple to diagnose the common genera macroscopically" yet no indication is given here, or later in the book how this can be done. A welcome addition would be a series of black and white photographs (such as those found in Angus Dunn, *Veterinary Helminthology*) with explanatory text illustrating the macroscopic differences between the more common and important genera. I am sure it would receive wide use in the field and help prevent some of the embarrassing misdiagnoses which occur from time to time.

With due respect to the author I rather suspect that Mr Linnaeus would not approve of his unsurpassed contribution to biology (viz. a classification system) being referred to as a "mania of man".

Two additional suggestions are that the indiscriminate use of formalin and iodine (both serious health hazards) should be clearly discouraged and that at least some worms in tissue lesions should be teased from the lesions for positive identification prior to fixing in formalin.

A brave bid is made to summarize, from diverse literature sources, the state of our ignorance concerning the complex mechanisms of immunity to parasitic infections. The text, although logical, includes perhaps too much detail of laboratory experiments with *Nippostrongylus braziliensis* for an undergraduate helminthology student (who in any case should have detailed concepts of immunity covered in other courses) and insufficient for the specialist. Nevertheless, this dynamic subject cannot be overlooked and subsequent editions will undoubtedly see this section amended and expanded.

Chemotherapy is a field in which Professor Reinecke has decades of experience and, as expected, there follows an excellent essay on anthelmintic tests. Although historical perspectives are invaluable for a student's textbook there would seem to be unnecessary detail concerning individual trial results of early studies. It is suggested that Stampa's observations (1959) be referred to in the past tense or not at all, as his findings are no longer valid. The statement that "the requirements for the registration of anthelmintics are far more advanced in the R.S.A. than elsewhere in the world" will not find acceptance by many overseas experts who regard the N.P.M. evaluation and the requirements

for testing against third larval stages of nematodes to be unnecessary. First, the test involves huge expenses, second, the third larval stage because of its short duration usually constitutes a very small percentage of the total parasite population in a grazing animal, and third, this stage is usually non-pathogenic. One wonders also why some of the parasites considered to be "rife" or "widespread" in South Africa (e.g. *Bunostomun trigonocephalum* (p 173) *Dictyocaulus viviparus* (p 178) are not permitted to appear on product labels). Perhaps some guidance should also be given to the field and diagnostic veterinarians who consistently confuse an "A" claim with 100 % removal of *all* worms from *all* animals. Adequate practical as well as statistical interpretation of label claims is essential.

The use of the words "mixture" or "combination" of compounds (pp 64-65) is misleading. There are few products available which are recommended for mixing or combination with other products. Most (including so-called "mixtures") are specifically formulated using careful procedures and efficacy, safety and stability may be adversely affected with indiscriminate mixing.

Other topics which could have found a place in this section include label claims against resistant parasites or hypobiotic larvae in R.S.A., the withdrawal periods for meat and milk following anthelmintic administration, the various modes of application/administration of anthelmintics (oral, injectable, topical, slow release, etc.) and, where applicable, their broad toxicologic and teratologic properties.

Part 2, which forms the bulk of the book, presents the parasites systematically, including aspects of their pathogenesis, clinical signs, clinical pathology, epizootiology and chemotherapy. It begins with a very useful and comprehensive introduction to the morphology, life cycle and ecology of the Strongylorida. Nematodes, trematodes and cestodes are dealt with in that order. Generally speaking this part is excellent, but I experienced some difficulty in interpreting the conclusions drawn from the presented data; some seem to be too dogmatic. The underlying problem is that considerable attention is given to epidemiological studies in other parts of the world without appropriate clarification of the South African circumstances. Names such as Brunsdon, Anderson, etc. do not necessarily lead a student to associate the reported findings with phenomena which have been observed in New Zealand or Australia. Too little is known of microclimates and parasite strains for the findings in other southern hemisphere countries to be considered valid for local conditions. Most South African parasite surveys have been done with small numbers of animals over one, or at most, two seasons. Their repeatability cannot be assumed, and in most cases confirmatory data should be gathered before conclusions can be drawn.

Throughout Part 2 reference is made to numerous place and area names (eg. Vryburg, Outeniqua, Border, etc.). This information would be more valuable to those without a detailed geographical knowledge of R.S.A: if a map was provided. At the risk of being labelled a pedant I would suggest that Vryburg, which receives 400-600 mm of summer rains be allocated to the Far Northern Cape Region and that arid Namaqualand, some 500 km west of there, to the North Western Cape.

There are some minor errors, omissions and necessary additions which should receive attention in subsequent editions: According to the work of Allonby and Preston (1977) the susceptibility of sheep does not decrease from Red Masai through Merino to Corriedale, but *increases* (p 81); the "spring rise" as it applies to *H. contortus* in R.S.A. is not clearly defined (p 83); The wisdom of using disophenol against *H. contortus* as late as March invites challenge from those who could contend that its incomplete efficacy against third and fourth larval stages, combined with the onset of hypobiosis at this time of year, could, in the presence of falling plasma levels, predispose to the emergence of resistant, strongly hypobiotic parasites (p 86) – it may also prove to be ineffective; *Ostertagia* is probably much more widespread in R.S.A. than has hitherto been believed (p 95); ivermectin, in addition to fenbendazole is effective against hypobiotic L4 *Ostertagia* (p 96); *Bunostomum phlebotomum*, which has a *characteristic* egg, is a particular problem in dairy calves (p 170).

If errors are not to be perpetuated there is a need to radically amend Figure 2.12 (p 84) which reflects guesses concerning epizootiology which were current 10 years ago. Horak has recently described several additional ecological areas. Furthermore we have long known that *Nematodirus* is rare in the northern third of the Karoo (*sic*) area, yet the text claims it is common there. Moreover the Boshoff benzimidazole-resistant strain of *H. contortus* originates from an area where it is "not supposed" to occur.

The section on Strongylinae of horses is delightful, with concise summaries of the latest information, excellent line drawings and keys and some lovely SEM photographs. It is indeed unfortunate that morphologic characteristics of the genera are referred to plate 2.57 and not 2.56. One questions the logic of Levine (p 161) who maintains that *Mammomonogamus* is non-pathogenic, yet animals may cough and lose mass. A comprehensive summary of data on pigeon roundworms is a most useful inclusion.

Some other general comments are appropriate. The fecundity of *Ascaris* should not be confused with fertility. No indication is given of the occurrence in R.S.A. of *Neoascaris vitulorum*, yet it is found quite frequently in calves slaughtered for veal. A photograph of the eggs of *Toxocara* and *Toxascaris* would have been useful as they are, together with *Ancylostoma*, the most common eggs a practitioner will see. It is also a little confusing that mice are mentioned as being both transport hosts (life cycle) and intermediate hosts (epizootiology) of *Toxascaris leonina*. Notwithstanding this, the comprehensive presentations on the ascarids of dogs and cats *Oslerus osleri* and *Spirocerca lupi* are excellent.

The section concerning *Thelazia* provides an example of the errors that can be made when information from different sources is pooled. Although many would disagree with him, Professor Reinecke considers that *Thelazia* "is believed to be non-pathogenic" – presumably in South Africa. The listed invertebrate hosts (*Musca larvipara* and *M. convexifrons*) are not, however, found in R.S.A., but in Russia where thelaziosis is a severe seasonal problem. The transmission of *Parafilaria bovicola* via seasonal bleeding points, which are the only external manifestation of infestation, is omitted. It could also be mentioned that *Cordophilus sagitta* needs to be differentiated from cardiac cysticercosis in cattle. Whilst *Trichinella spiralis* is concisely reviewed, *Trichuris globulosa* which was the only *Trichuris* found by Mönnig in the Onderstepoort collection (1933) is not mentioned.

Trematodes are dealt with thoroughly, although sometimes no indication is given whether they are found in R.S.A. (e.g. *Eurytrema pancreaticum*). There is an excellent section on snail morphology and a most useful diagram from Dawes illustrating the life cycle. Any such work must mention Boray and this is done in an appropriately unobstrusive way. It must be borne in mind, how-

Continued overleaf.

ever, that the epizootiology of fascioliasis is probably quite different in southern Africa and Australia. Perhaps more use could have been made of personal communications here to give a better idea of local circumstances. The section on paramphistomiasis is outstanding as is that on schistosomiasis – but schistosomiasis is over done. Eight text pages, 4 figures and 30% of the colour plates are devoted to a disease which occurs sporadically in South Africa and is not recognized as a consistent cause of major stock losses.

There is an excellent introduction to the morphology and life cycle of cestodes. The order Taeniidae is dealt with logically and interestingly – indeed this is the first time that the extensive studies of Drs Verster and Rausch have been put together. One wonders, however, whether detailed morphological descriptions and figures (genus Taenia (10); genus *Echinococcus* (4)) are necessary in a book of this nature. First the required techniques of staining, sectioning and mounting are not covered in the text and second such taxonomy is a specialist's function. It can justifiably be contended that this book meant "to go further". If so, it certainly has. The author states "The order Anoplocephalidea is of great importance to veterinary surgeons", yet 5 lines suffice to describe what we know of its pathogenicity in ruminants. In this regard little has changed since Mönnig's first edition. Bearing this in mind, the importance of *Stilesia hepatica* which, in clearly defined instalments, costs South Africa probably as much as any other single parasite, is understated. Missing from this section is a guide to diagnosis of *Moniezia* spp. and *Thysaniezia giardi* by faecal examination, which could be essential in selecting appropriate chemotherapy.

The book concludes with useful sections on zoonoses and control procedures, although many parasitologists in South Africa will not agree that low level dosing of either phenothiazine or benzimidazoles is justified. (Incidentally no products have registered claims for such use.)

Preventive medicine is vital to successful farming and if the section on control is to retain its value it will have to be updated to include recommendations which are much more specific for specialist sheep and cattle farmers. The young rural veterinarian is an expensive commodity who requires a thorough knowledge of investigating a flock problem; if he does not have this knowledge given to him in an undergraduate course he is dispensible – there is no time for him to learn at the farmer's expense. In view of the importance of the horse industry and its clearly defined veterinary components viz. studs, foals to yearlings, two and three year olds, older race and leisure horses, pony clubs etc. it would be worthwhile expanding this section to reflect its significance.

The glossary is a valuable inclusion and will certainly be extensively used.

The book is attractively presented with a well-spaced, large type face on firm paper. The binding is solid and the cover firm and durable – so essential for a volume which will see much practical use. It is excellently illustrated and liberally supplied with explanatory notes, diagrams and keys. There are 8 attractive plates. Unfortunately, two components of Plate 1 are transposed. There are a number of typographical errors (I counted 11) and the word "injectable" is consistently misspelled. At R55 it is expensive as a student's text, but it is worth it.

"*Veterinary Helminthology*" represents a milestone in South African veterinary history and is a tribute to the contributions that Professor Reinecke has made. The framework has been laid for a textbook that will grow with time. It should be a constant companion of students and field veterinarians and, although insufficient as a primary source for overseas colleagues, will provide them with an invaluable reference. Its support is essential and will have long term benefits to the profession in South Africa.

I.H. Carmichael

BOOK REVIEW — BOEKRESENSIE

LIVE ANIMAL REGULATIONS

11th edition International Air Transport Association, Montreal 1984 pp vi and 232

This publication is an updated specifications manual for the carriage of live animals by air. Its regulations are applicable to all Member Airlines of the International Air Transport Association (IATA) in both scheduled and unscheduled operations who are parties to the IATA Interline Traffic Agreement on Cargo. The regulations are explicit and binding and cover general aspects which must be considered to ensure that animals are carried without the risk of harm to either themselves or handling personnel. These specifications have also been adopted as guidelines for the transportation of animals by air by the Convention on International Trade in Endangered Species of Wild Fauna and Flora (CITES) and the Office International des Epizooties (OIE).

This 11th Edition became effective from the 1st Janaury, 1984.

The Manual is divided into 12 sections which provide general background information which facilitates the use of the regulations and detailed specifications for shipping procedures, carriage procedures, container design and requirements, governmental regulations and air carrier regulations. The role of the International Organisation of Epizooties and Convention on International Trade in Endangered Species in meeting acceptable sanitary standards and the control of epizootic diseases and control of the commercial exploitation of endangered animals is briefly reviewed.

The text is supported by general information on the scientific nomenclature of animals, live animals acceptance sheets and animal handling procedures.

The regulations cover air transportation requirements for insects, birds, amphibians, mammals, reptiles, crustacea and fish. They have drafted to ensure the welfare of animals in transit on an international basis. The Manual will be of value to veterinarians engaged in zoo animal medicine and to dealers in wild, companion and laboratory animals. It is an essential reference document for all engaged in the transportation of animals by air.

J.C. Austin

EPIDIDYMITIS OF RAMS IN THE CENTRAL AND SOUTHERN DISTRICTS OF THE ORANGE FREE STATE

J.A.L. DE WET* and J.A. ERASMUS**

ABSTRACT: De Wet J.A.L.; Erasmus J.A. **Epididymitis of rams in the central and southern districts of the Orange Free State.** *Journal of the South African Veterinary Association* (1984) 55 No. 4, 173-179 (En). Veterinary Laboratory, P.O. Box 502, 9300 Bloemfontein, Republic of South Africa.

Scrotal palpation, microscopical examination of semen smears and the application of bacteriological techniques, revealed the incidence of *Actinobacillus seminis* and *Brucella ovis* infection of rams in the central and southern districts of the Orange Free State to be 2,9 % and 4,3 %, respectively. More Dorper than Merino rams were affected. Although clinically detectable epididymitis was found in 5,9 % of rams examined, infection with *A. seminis* and *B. ovis*, as measured by the presence of neutrophils in semen and positive semen cultures, could only be demonstrated in a small minority of affected cases. These organisms were found more regularly in clinically negative rams excreting neutrophils in their semen.

Possible reasons for the high incidence of sheep brucellosis in rams in the particular area as well as the higher incidence of infection in Dorper rams are discussed.

Key words: Epididymitis, *Actinobacillus seminis, Brucella ovis*, rams.

INTRODUCTION

Epididymitis of rams can be caused by a variety of bacterial organisms such as *Acinetobacter lwoffi, Actinobacillus actinomycetem comitans, Actinobacillus seminis, Bacteroides spp. Brucella abortus, Brucella ovis, Corynebacterium pseudotuberculosis, Corynebacterium pyogenes, Haemophilus spp., Moraxella spp., Pasteurella haemolytica, Pasteurella multocida, Pasteurella pseudotuberculosis, Staphylococcus spp.* and *Streptococcus spp.*[18,14]. It appears, however, that *A. seminis* and *B. ovis* can be regarded as the main causes of the disease in the Republic of South Africa[13].

About 2 decades ago van Rensburg et al.[12] determined the incidence of sheep brucellosis in the Cape Province and some of the southern districts of the Orange Free State (OFS) to be about 12 %. In this particular survey the diagnosis of brucellosis was only confirmed by the microscopic examination of semen smears from the selected rams. Although the presence of *B. ovis* organisms can be detected with a high degree of accuracy by employing Stamp's staining technique[9,16], experience has shown that the microscopical demonstration of the organism in bacteriologically positive semen may not always be possible. The incidence of infection may thus well have been much higher than the suggested figure.

Recently van Tonder[15] conducted a similar survey in which bacteriological examinations and the complement fixation test formed the basis of the laboratory diagnosis of both *A. seminis* and *B. ovis* infections. The majority of these rams originated from the Cape Midlands and the Karoo and represented stud and commercial Dorper, Karakoel and Merino rams. From bacteriological examinations performed on 1 058 semen samples 401 or 37,9 % were positve for *A. seminis* and 22 or 2,1 % were positive for *B. ovis*. Of a total of 33 656 rams examined, 2,5 % revealed a clinically detectable epididymitis.

Vaccination of rams with the attenuated Elberg Rev. 1 strain of *Brucella melitensis* (Rev. 1 vaccine) which was introduced by van Drimmelin[10], proved to be a highly effective method of control sheep brucellosis[11]. The less favourable results obtained with vaccination reported subsequently[20], were attributed to faulty vaccination procedures, which together with a disregard for regular vaccination[19], could be taken as the main causes for the continued, but decreased occurrence of this disease in South Africa.

Judging by the reported incidence of sheep brucellosis in the adjacent area, a widespread infection of *B. ovis* could be expected in both stud and flock rams in the central and southern districts of the OFS. Data obtained from routine diagnostic tests done on rams at the Veterinary Laboratory in Bloemfontein were used to confirm this hypothesis and to compare the incidence of *A. seminis* infection in rams in the latter area with results obtained by van Tonder[15]. *A. seminis* as referred to in this study embraces 3 different bacteria viz. *A. actinomycetem comitans, A. seminis* and *H. ovis*[5,6].

MATERIALS AND METHODS

The scrotal contents of 15 225 rams of various breeds from 671 farms in the central and southern districts of the OFS were examined by visual inspection and palpation. Semen samples were collected electrically from these animals according to the method of Van Tonder et al.[18]. Smears from these specimens were stained with either CAM's Quick stain (C.A. Milsch (Pty) Ltd., Krugersdorp) or Loeffer's methylene blue[4] before being microscopically examined for the presence of neutrophils. The classification as set out in Table 1 was used for this purpose.

All semen samples with a 1+ or more neutrophils were subjected to a second smear examination and bacteriological culture. These smears were stained using Stamp's staining method[9] and examined for the presence of acid-fast bacilli as well as non acid-fast pleomorphic bacilli in the cytoplasm of the neutophils.

From each of the samples containing neutrophils a 0,1 mℓ aliquot was plated out on tryptose blood agar containing 5 % (v/v) sterile horse blood*. All samples were cultured for at least 4 days at 37 °C in an atmosphere of 10 % CO_2. A culture was regarded as being positive for *B. ovis* if, after 4 days, dome shaped, light grey in colour, translucent and about 1 mm in diameter,

Continued on page 176.

*Veterinary Laboratory, P.O. Box 502, 9300 Bloemfontein.
**Veterinary Laboratory, Kroonstad.

*Obtained from the Veterinary Research Institute, Onderstepoort.

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Curatik is an extremely stable dip and **safe,** even at 20 times the recommended strength. Even for sick and weak animals, young and old, lactating and pregnant. It allows a **dipping interval** of 14 **days** depending on the tick population.

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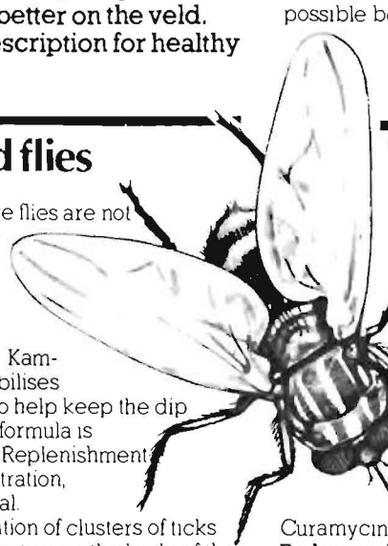
Regular Curatik dipping eliminates the carrier, the face fly. Summer **dehorning** is possible because Curatik reduces the danger of screw-worm infestation. This leads to a drastic **reduction in fly and screw-worm population.**



Tick-borne diseases and other bacterial infections.

Heart-water: Curamycin 123 and Curamycin L.A.

Tick-borne gall-sickness: Curamycin 123 and Curamycin L.A.

Foot-rot, navel-ill, pneumonia and septic joint infections: Curamycin 123, Curamycin L.A. and Sulphadimidine
Redwater: Phenamidine solution

CURAMYCIN 123 is a broadspectrum antibiotic with 123 mg oxytetracycline per mℓ. A high concentration allowing a smaller dose. It halves the cost of oxytetracycline treatment

CURAMYCIN L.A. injectable solution contains 200 mg oxytetracycline per mℓ. It ensures sustained blood levels for 3 to 5 days in cattle, sheep, goats and pigs after a single intramuscular injection of 1 mℓ per 10 kg body mass. It is clear, sterile, stable and ready for use

SULPHADIMIDINE: SODIUM B.P. 33⅓%: For the treatment of pneumonia, bacterial diarrhoea, cocsidiosis, septic joint infections and navel-ill in cattle.

PHENAMIDINE injection against Redwater in cattle. A single dose usually i effective.

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- Curatik dipping helps to control blowfly strike.
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DAZ-DUST powder contains 2% Diazinon, one of the safest insecticides. Kil blowfly maggots on contact. Helps healing by drying out the wound.

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Spray with **CURADINE.** Containing povido iodine, it works in the presence of blood, p and dead tissue. Helps against **foot-rot,** infectious **eye infection** and **ringworm in cattle.** Effectiveness at very low cost.

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- Eliminate the carrier with **CURATIK** dipping
- Inject infected animals with

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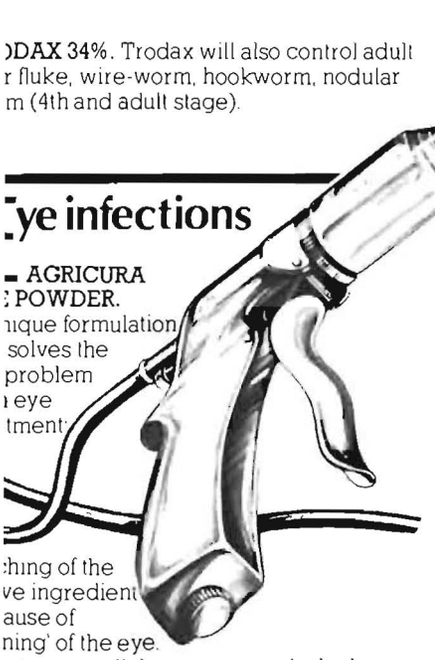
;ura
n for veld cattle

)DAX 34%. Trodax will also control adult r fluke, wire-worm, hookworm, nodular m (4th and adult stage).

Eye infections

– AGRICURA
: POWDER.
nique formulation solves the problem eye tment:



:hing of the ve ingredient ause of ning' of the eye.
ontains a cellulose compound which is a jelly-like layer. This dissolves slowly, easing the active ingredient to tinuously fight infection. It works so well ause it stays on the eye so long.

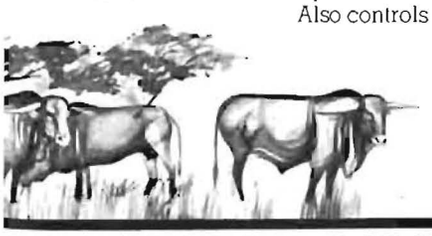
ternal parasites

EPONVER Injectable Roundworm nedy, also effective against ordinary liver :e and giant liver fluke. It has a 3-week idual action against wire-worm. Use it) when liver fluke occurs.

RIPERCOL-L 150 Injectable Solution or use when liver fluke is **not** a problem. Also controls wire-worm, brown stomach worm, bankrupt worm, nodular worm and hookworm by intramuscular injection.

RIPERCOL POUR-ON Systemic Roundworm Remedy for the treatment of large numbers of animals. It is sprayed or poured on to the animal's back or side. It is absorbed through the skin to act against all the common roundworm species.

Tapeworm In Calves: Use Multispec Oral Suspension



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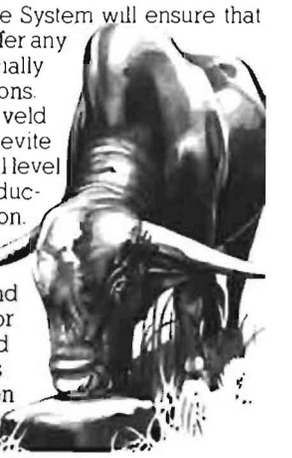

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Table 1: INTERPRETATION OF THE NEUTROPHIL DENSITY IN A SEMEN SMEAR

Number of neutrophils per microscopic field[a]	Arbitrary allocation
1 – 2	+
4 – 5	+ +
6 – 19	± ±
20 – 30	+ + +
Too many to count	+ + + +

a: 10 fields counted per smear (x 1000)

colonies were found. A final positive microscopic diagnosis was made, if smears made from these colonies and stained with Stamp's[9] technique, revealed the presence of actinomorphic bacilli occurring either singly or in pairs showing acid-fast properties.

A diagnosis of *A. seminis* was made if the colonies exhibited the following characteristics after 48 h incubation on the above-mentioned medium: 1-2 mm diameter, low convex or dome-shape, greyish white appearance. The diagnosis was confirmed if pleomorphic Gram-negative bacilli were obtained which showed no growth on MacConkey's agar and if, in the absence of serum in the medium, oxidation or fermentation of glucose did not occur within 48 h at 37 °C.

RESULTS

The area in which the survey was conducted, is shown in Fig 1.

The incidence of epididymitis

Summarized data of the clinical and bacteriological

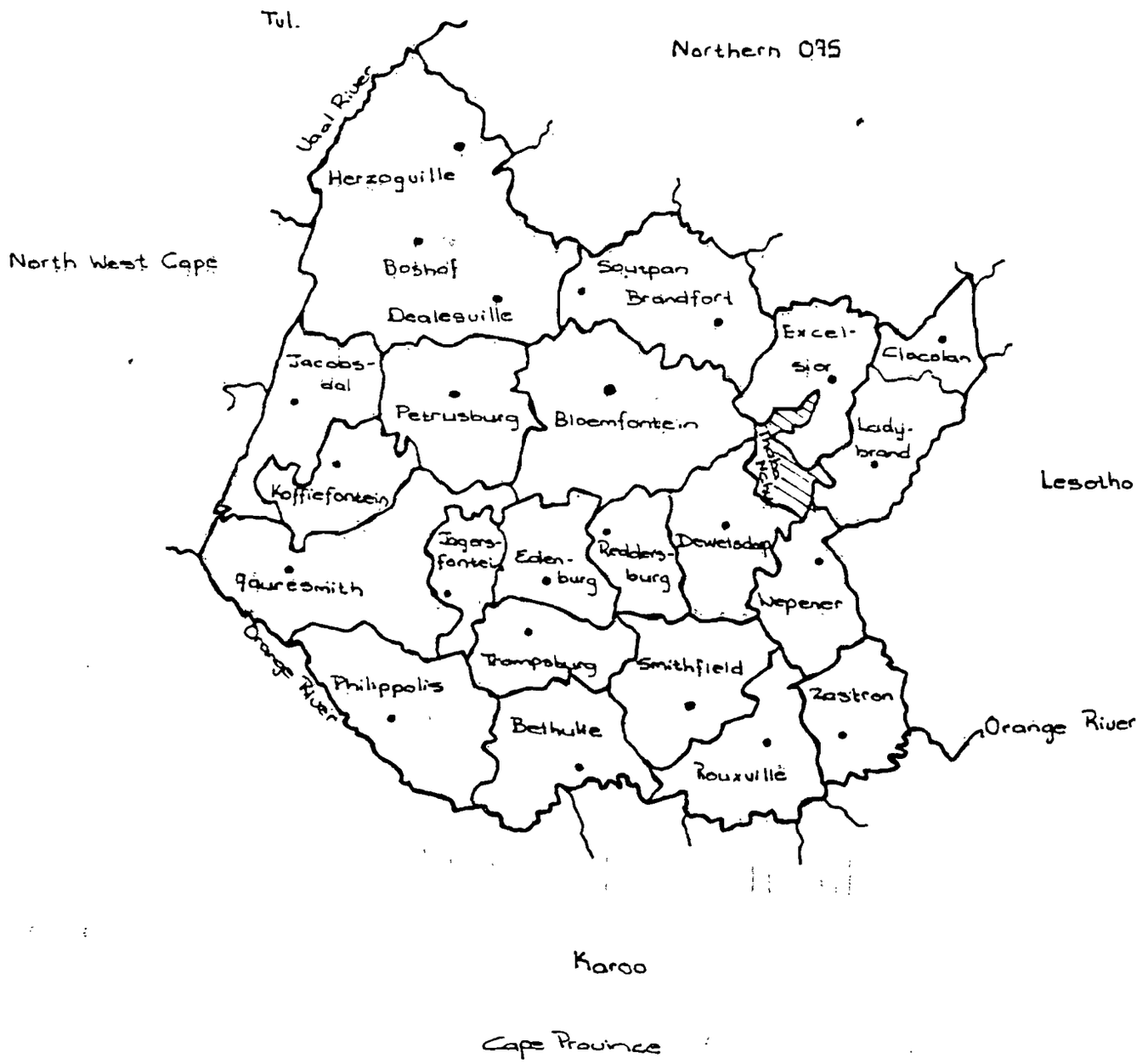

Fig. 1 Districts of the central and southern Orange Free State in which the survey was conducted.

Table 2: THE INCIDENCE OF *ACTINOBACILLUS SEMINIS* AND *BRUCELLA OVIS* INFECTION IN RAMS IN THE CENTRAL AND SOUTHERN DISTRICTS OF THE ORANGE FREE STATE

Year	1980-81	1981-82	1982-83	Total	% Rams
Number of rams tested	5 034	5 211	4 980	15 225	—
Rams with lesions[b]	287	272	346	905	5,9
Rams infected with *A. seminis*[c]	235	158	50	443	2,9
Rams infected with *B. ovis*	303	187	172	662	4,3
Number of farms	240	212	219	671	—

b: lesions = clinically detectable epididymitis
c: *A. seminis* represents *A. actinomycetem comitans, A. seminis* and *H. ovis*

Table 3: THE INCIDENCE OF CLINICALLY DETECTABLE EPIDIDYMITIS IN DORPER AND MERINO RAMS

Age of rams	Testicular lesions				Total number of	
	present in		absent in			
	Dorper rams	Merino rams	Dorper rams	Merino rams	Dorper rams	Merino rams
Lambs	20 (4,3 %)	9 (1,5 %)	446 (95,7 %)	591 (98,5 %)	466	600
2-Tooth	24 (4,8 %)	13 (1,6 %)	474 (95,2 %)	821 (98,4 %)	498	834
4-Tooth	18 (7,5 %)	20 (3,4 %)	222 (92,5 %)	570 (96,6 %)	240	590
6-Tooth	19 (7,0 %)	37 (5,9 %)	254 (93,0 %)	492 (94,1 %)	273	529
Full-mouthed	44 (8,2 %)	48 (3,4 %)	395 (91,8 %)	1 380 (96,6 %)	439	1 428
Old	11 (13,1 %)	20 (8,1 %)	73 (86,9 %)	227 (91,9 %)	84	247
Total	136 (6,5 %)	147 (3,4 %)	1 864 (93,5 %)	4 081 (96,6 %)	2 000	4 228
Survey by van Tonder[15]	161 (5,7 %)	641 (2,1 %)	2 653 (94,3 %)	29 609 (97,9 %)		

X^2 (Dorper rams) = 2,501; P>0,05
X^2 (Merino rams) = 46,15; P>0,001

Table 4: THE INCIDENCE OF *A. SEMINIS*[c] AND *B. OVIS* INFECTION IN CLINICALLY NORMAL RAMS AND IN RAMS WITH CLINICAL EPIDIDYMITIS

Age of rams	Number of rams						Total number of rams examined
	with clinical lesions			without clinical lesions			
	A. seminis positive	*B. ovis* positive	Bact. negative	*A. seminis* positive	*B. ovis* positive	Bact. negative	
Lambs	3	4	30	74(5,6)	30(2,3)	1171	1312
2-Tooth	7	8	50	49(3,1)	37(2,4)	1415	1566
4-Tooth	5	7	33	26(2,8)	47(5,0)	821	939
6-Tooth	1	23	27	28(2,7)	88(8,4)	885	1052
Full-mouthed	6	19	83	48(2,1)	176(7,5)	2005	2337
Old	4	9	22	8(2,0)	8(2,0)	353	404
Total	26(0,3)	70(0,9)	245	233(3,1)	386(5,1)	6650	7610

c: *A. seminis* represents *A. actinomycetem comitans, A. seminis* and *H. ovis* The figure in bracket = % rams

findings are given in Table 2. The results of the clinical examinations performed on 6 228 rams during 1980 – 1982 on Dorper and Merino rams with more complete data regarding age, appear in Table 3.

Approximately 5,9 % of the rams examined suffered from a clinically detectable epididymitis (Table 2). The incidence of this lesion in Dorper rams was about twice that seen in Merino rams (Table 3). Epididymitis in Merino rams occurred more regularly in the central and southern districts of the OFS (Table 3). With regard to the ram lambs that have reached puberty, 4,3 % of the Dorpers and 1,5 % of the Merino's were affected. In both breeds the incidence of epididymitis increased with age.

A. seminis and B. ovis in clinically normal and abnormal rams

The incidence of *A. seminis* and *B. ovis* infection in the 7 610 rams examined during 1980 – 1982 for which complete data regarding age was available, is given in Table 4.

B. ovis was found in 6 % of this group of rams (Table 4). At least 2,3 % of the clinically normal ram lambs excreted the organism at the time of sampling. As the rams aged, the rate of infection increased to such an extent that 7,5 % of the full-mouthed rams excreted the organism. The opposite tendency was noted in the case of *A. seminis* infection. Here the rate of infection decreased from 5,6 % in clinically normal

ram lambs to 2 % in the clinically normal full-mouthed animals.

The number of clinically positive rams in each group was very small. No conclusions could be drawn from this data except that about 1,2 % of them excreted either *A. seminis* or *B. ovis* organisms in their semen (Table 4).

DISCUSSION

Several methods, such as palpation of the scrotal contents, microscopical examination and semen culture as well as the complement fixation test are available for the detection of *A. seminis* and *B. ovis* infection in rams[1,3,7,14]. Palpation is a reliable technique in screening rams for the presence of clinical epididymitis, but is useless for the detection of the subclinical case. At least 5,9 % of all rams examined during this survey had a clinically detectable epididymitis. This figure is more than double the 2,5 % clinically positive cases found in especially the Cape Midlands and the Karoo region[15].

Semen smear examinations are one of the most important methods for the detection of genital infection in so far as it is useful for the demonstration of both neutrophils and bacterial organisms in semen. The presence of neutrophils in semen could be regarded as indicative of specific genital infection, since there is general agreement that normal semen should not contain any leucocytes[16]. As the presence of bacterial organisms is more difficult to establish in general, culture methods are more reliable to demonstrate their presence in semen samples.

Compared with information given by Van Tonder[15], the incidence of *A. seminis* infection in the relevant parts of the OFS was particularly small; the highest incidence of 5,6 % occurring in ram lambs that have reached puberty. Dorper rams in the 2 different areas were about equally affected clinically, with less Merino rams positive for clinical epididymitis in the Cape Midlands and the Karoo.

With regard to an *A. seminis* infection in a flock, Jansen[8] postulated that the preputial cavity of a ram becomes invaded with organisms through contact with the environment. When, under the influence of hormonal stimulation, the genitalia undergo development, suitable conditions are created for the migration of some bacteria in the sheath to deeper-lying organs of the genital tract such as the vesiculae seminales, epididymides and the testes. In these organs the bacteria may initiate a pathological process resulting in clinical lesions with the total occlusion of the epididymis or in subclinical lesions with or without tubular occlusion. The majority of subclinically affected ram lambs even rid themselves of the infection[13]. Jansen[8] further reported that the most vigorous and virile young rams showing the most rapid growth rate in a group of rams kept in an intensive system on a high plane of nutrition are more likely to develop epididymitis or orchitis.

The decreasing incidence of *A. seminis* infection can easily be seen from the data in Table 4. Although the particular reasons for the low incidence of *A. seminis* infection in the central and the southern districts of the OFS were not particularly looked for, it is possible that more extensive farming practices followed in this area could be one answer to this question.

In New Zealand, sheep brucellosis as caused by *B. ovis* is characterized by abortion[2]. In South Africa the presence of the disease in a flock usually passes unnoticed and is detectable only when semen of infected rams are examined or when the appropriate serological tests are performed on serum samples. Rev. 1 vaccination is widely applied in rams in the Karoo. Use of this vaccine is doubtlessly the main reason why only 2,5 % of the rams from that particular area were bacteriologically positive for *B. ovis*[12]. On the other hand the incidence of *B. ovis* infection in the central and southern districts of the OFS reported in this study was found to be 6 %.

In contrast to *A. seminis* infection, sheep brucellosis is thought to spread directly by contact between rams, or indirectly via the ewe during mating[3]. The application of Rev. 1 vaccine results in a solid immunity[17]. Therefore the high incidence of sheep brucellosis in the relevant districts of the OFS could only be the result of no or improper vaccination and contact with infected rams.

In comparison with the results obtained by Van Tonder[15], the incidence of epididymitis in Dorper rams was comparable with that seen in the central and southern districts of the OFS (Table 3). The increased occurence of epididymitis in Dorper rams was noted earlier[15]. Although the reasons for this phenomenon were not investigated during this survey, it is worth while to note that sexual activity in this breed commences earlier than in the Merino. It is also generally accepted that the growth rate of the Dorper ram exceeds that of the Merino. From the above it is possible to deduce that the Dorper should be more susceptible to these infections than the Merino.

In comparison with the Cape Midlands and the Karoo where *A. seminis* appears to be the main cause of ram epididymitis, the incidence of this type of infection in the central and southern districts of the OFS appears to be insignificant. Instead *B. ovis* appears to be the most important cause of genital disease of rams in the latter area, with an incidence of about twice as much as would be expected in the Cape Midlands and the Karoo.

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AN OUTBREAK OF *COTYLEDON ORBICULATA* L. POISONING IN A FLOCK OF ANGORA GOAT RAMS

R.C. TUSTIN*, D.J. THORNTON** and C.B. KLEU***

ABSTRACT: Tustin R.C.; Thornton D.J.; Kleu C.B. **An outbreak of *Cotyledon orbiculata* L. poisoning in a flock of Angora Goat rams.** *Journal of the South African Veterinary Association* (1984) **55** No. 4, 181-184 (En). Department of Pathology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

An acute outbreak of *C. orbiculata* L. poisoning in a flock of 16 Angora Goat rams is described. Typical signs of acute bufadienolide cardiac glycoside toxicity were manifested and 6 animals died. In the 2 animals examined histopathologically multiple foci of myocardial degeneration and necrosis were present. Treatment consisted, inter alia, of drenching with a mixture of activated charcoal, potassium chloride and a commercial preparation, Universal Antidote (Centaur) and parenteral administration of atropine sulphate.

Key words: *Cotyledon orbiculata* L. poisoning, krimpsiekte, bufadienolide cardiac glycoside, Angora Goat, cardiomyopathy.

HISTORY AND CLINICAL INVESTIGATION

One Sunday afternoon during July 1983 a flock of 16 adult Angora Goat rams in the Murraysburg district, Cape Provinve was placed in a small camp next to the homestead. Early on the following Tuesday morning it was discovered that 2 of them had died during the previous night and that 4 were prostrate. The remainder were anorectic, lethargic and disinclined to move although they would move off at a walk if approached too closely. Veterinary assistance was immediately sought.

At that time the farm was in the grip of a severe drought and the goats in question had previously been maintained for several months in a camp in which there was an abundant growth of old man salt bush (*Atriplex nummularia*) and nothing else. The camp in which the disease occurred was virtually devoid of all plant growth except for a small patch of short green weeds near a fenced-off sheep dip bath. The goats had been dipped in an organophosphate-containing dip wash 6 days previously.

The most apparent signs in the 4 severely affected animals were prostration, ruminal stasis, anorexia, tachycardia, a reluctance to move and, when induced to stand, the head was held low. Two of them showed a wetness around the mouth which was considered to be evidence of salivation while the visible mucous membranes were pale. In addition, faecal soiling of the hair of the buttocks indicated the possibility of a diarrhoea or passage of soft fluid faeces, and constricted pupils were present in 3 of the 4 animals. Six of the other animals in the rest of the flock also showed the latter clinical sign.

These clinical signs were interpreted as being due to possible organophosphate poisoning and, without further ado, 2 of the prostrate goats were injected subcutaneously with atropine sulphate (1 ml of a 10 mg/ml solution). About 10 minutes after this treatment the most severely affected animal stood up, walked a few paces and remained standing for some hours.

A necropsy of one of the dead animals was then commenced. On opening the rumen pieces of the leaves of a *Cotyledon* sp. were immediately noticed intermingled with the other contents. On being questioned about their presence and possible source, the farmer related that he had recently removed a relatively large quantity of these plants from a rockery near the fence of the camp in which the disease had occurred. The plants had then been thrown over the fence into the camp. Only a low spread-out heap of their stems remained on the day of the investigation; no leaves at all were present. The tentative diagnosis of organophosphate poisining was changed to a diagnosis of *Cotyledon* sp. poisoning and treatment of all the animals was immediately commenced as it was assumed that all had eaten the plant. The duration of treatment of the goats took about 1½ hours during which period another 2 animals died. Two more animals died on the fifth day after having ingested the toxic plant, the death of one of which was unexpected as it had appeared to be improving in health, while that of the other was expected, being one of the 4 severely affected animals that were first examined.

A specimen of one of the remaining plants concerned which was growing in the rockery was collected for later identification. It proved to be *Cotyledon orbiculata* L.

TREATMENT

The animals all received the following treatment:

1. A mixture of activated charcoal (5 g/kg live mass), potassium chloride (1 g/kg) and 37 g/goat of a commercial preparation (Universal Antidode (Centaur) containing 40 % activated charcoal, 20 % magnesium oxide, 20 % tannic acid and 20 % kaolin) was added to about 0,5 ℓ of water and then administered via a tube directly into the rumen. This was repeated on the following day.
2. Five ml of a preparation containing 200,000 i.u. procaine penicillin and 250 mg dihydrostreptomycin sulphate per ml (Streptopen, Milvet) was injected intramuscularly on 4 occassions at intervals of 12 hours.
3. One ml of a solution containing 10 mg/ml atropine sulphate was administered subcutaneously. The following day each animal received 0,5 ml subcutaneously of the same preparation.

PATHOLOGY

The first 3 animals to die were necropsied. All showed generalised congestion, severe lung oedema, a very watery ruminal content in which numerous pieces of the

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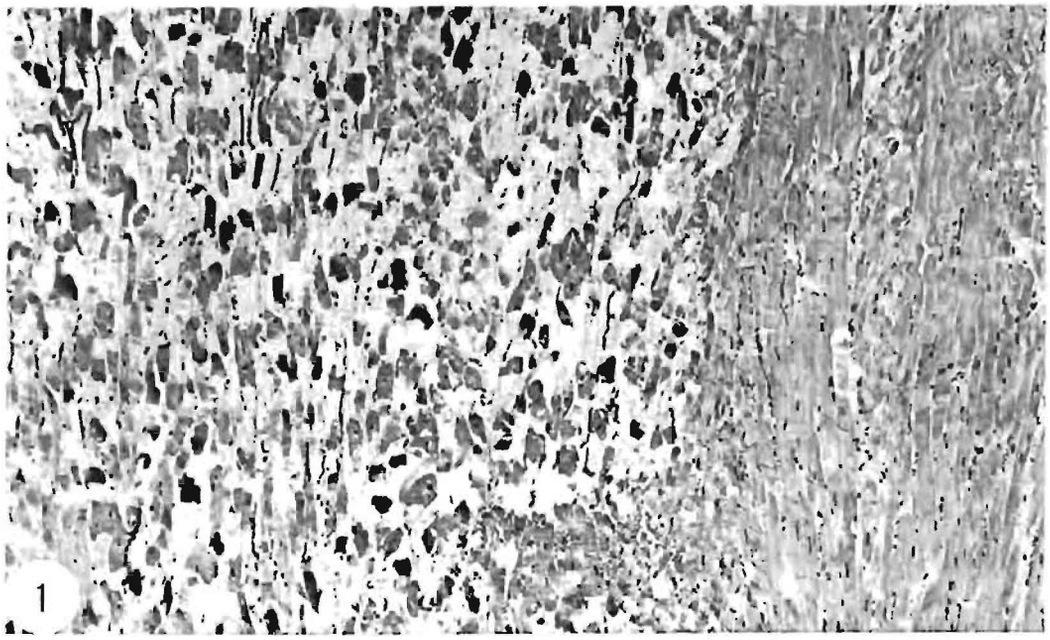

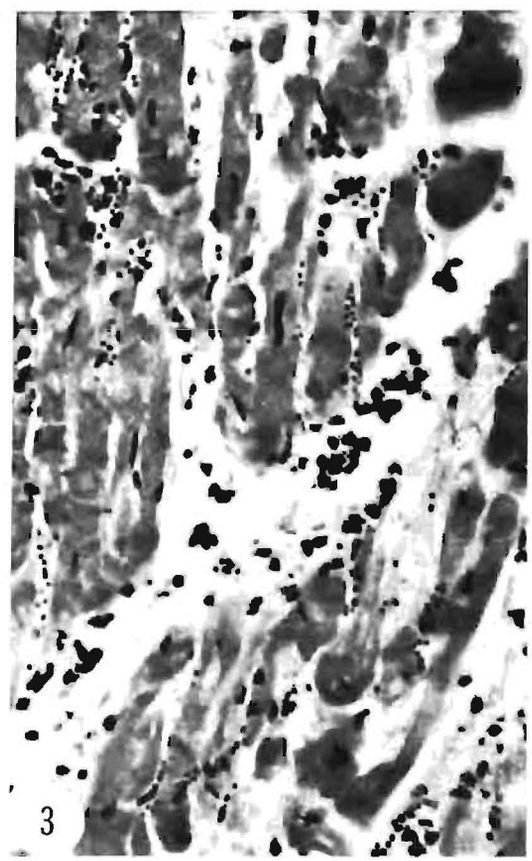

Fig. 1: Photomicrograph of the myocardium showing an area of necrosis and apparent fragmentation of muscle fibres. Haematoxylin and eosin stain (HE) X100

Fig. 2: Myocardial degeneration of necrosis manifested by karyopyknosis and swelling of some segments of fibres with coagulation, loss of structure and intense eosinophilia, and vacuolation and loss of sarcoplasm, of other segments. A few extravasated erythrocytes are present. HE X300.

Fig. 3: An area of myocardial necrosis associated with an infiltration of cells consisting predominantly of neutrophils and smaller numbers of macrophages and lymphocytes. HE X300.

leaves of a *Cotyledon* sp. could easily be identified and a softer than normal large intestinal content. The small and large intestinal mucosa of 2 of the goats was hyperaemic in parts and one animal exhibited a wetness of the skin around the mouth which was assumed to have occurred ante mortally. In addition, an early pneumonia was present in a lobe of the lungs of one of them, and quite extensive epi- and endocardial haemorrhages were evident in all.

Representative specimens of the lung, heart, spleen, liver, kidneys, intestine and various lymph nodes from 2 of the carcases were fixed in 10 % formalin for microscopy. The specimens from the hearts were taken at random but consisted of right and left ventricular walls and interventricular septa. With the exception of extensive myocardial lesions present in both animals, the histopathological examination confirmed the macroscopic findings. The pneumonia seen in one of the cases proved to be a purulent bronchopneumonia in which numerous bacteria were present. The myocardial changes which were present to a greater or lesser degree in all sections of the hearts examined manifested as multifocal areas of degeneration and/or necrosis of segments of muscle fibres, single fibres and bundles or larger groups of fibres being involved. In some sections they were quite extensive. Affected parts showed swelling of muscle fibres, intense eosinophilia, loss of striations and/or vacuolation and, in some areas, even apparent fragmentation. Many nuclei were pyknotic or absent. A small number of the foci was associated with an infiltration of cells, the neutrophil being the most numerous and macrophages and lymphocytes occurring in much lesser numbers (Figs. 1-3).

DISCUSSION

According to Vahrmeyer[13], krimpsiekte or nenta due to ingestion of certain plant species (plakkies) of the genera *Cotyledon, Tylecodon* and *Kalanchoe* (family Crassulaceae) has long been known in southern Africa, i.e. at least since 1775. It appears, however, that it was not until 1890 that the cause of the disease was unequivocally demonstrated by the feeding of *Cotyledon ventriculosa* Burm. (= *Tylecodon ventriculosus* [Burm. F.] Toelken[12]) to goats[16]. This experiment was conducted by Veterinary Surgeon Soga following a report by a farmer in the Somerset East district that he suspected the plant in question. Confirmatory experiments were carried out in the latter 1890's[16]. Henning (1926) described in detail 2 basic forms of the disease in goats and sheep; the acute or "opblaas" (bloat, ruminal tympany) syndrome and chronic or "dun" (thin) krimpsiekte[3]. The latter is a paretic syndrome characterized, inter alia, by the following: an affected animal lags behind the flock, easily tires when driven and lies down apparently exhausted when left, salivates excessively, and exhibits trembling and spasms of musles especially those of the head, neck and back, torticollis and a loose dangling of the head[3].

Krimpsiekte is a disease of considerable economic importance in the Republic of South Africa[7]. It occurs most frequently in goats, but sheep, cattle, horses and fowls are also susceptible[3 10 16]. It is of more than passing interest to note that the liver and meat of animals that have died from krimpsiekte are toxic when fed to dogs, eliciting the typical disease[3], and that even man can be affected by eating such raw or undercooked meat[16]. It is the only plant poisoning encountered in South Africa where this potential danger has ever been proved[7].

Bufadienolide cardiac glycosides have relatively recently been isolated from several of these plant species, such as *Tylecodon wallichii* (Harv.) Toelken subsp. *wallichii* (= *Cotyledon wallichii* Harv.)[12 14 15], *Kalanchoe lanceolata* Forsk[2] and *Tylecodon grandiflorus* (Burm. F.) Toelken[1]. Experimental reproduction of the syndromes resembling those encountered in the field may be achieved in sheep by the parenteral administration or drenching of these bufadienolides at various dosage levels: typical signs of cardiac glycoside poisoning occurring at higher dosage levels while the paretic syndrome regarded today as krimpsiekte only being elicited by repeated administration of smaller doses[1 2 8]; these bufadienolides being extremely cumulative and potent neurotoxins[8]. It is apparent that the condition we investigated corresponded more to the acute disease described by Henning[3], Terblanche & Adelaar[11], Naude[7], Naude & Schultz[8] and Anderson et al.[1 2].

C. orbiculata L., also known colloquially as pig's ear, hondeoor-plakkie, kooltrie, kouterie, kotrie, plakkie or varkiesblaar, occurs as entirely glabrous succulent shrublets of up to 800 mm in height throughout the Cape Province (with the exception of the north eastern areas), Lesotho, southern Natal, eastern Orange Free State and southern and central South West Africa/Namibia (Fig. 4)[11]. This plant species is very similar in appearance to the toxic species *C. leucophylla* R.A. Dyer ex C.A.Sm. which occurs in the Transvaal and this similarity has led to some confusion in the past as to their toxicity[11]. In 1965, however, Terblanche & Adelaar proved beyond doubt, following an outbreak of krimpsiekte in the Maltahöhe district of South West Africa that *C. orbiculata* L. was indeed toxic[11]. They also demonstrated its cumulative potential.

The rationale of our treatment was the following:

1. *Activated charcoal and potassium chloride.* In 1982 Joubert & Schultz described the treatment of ruminants suffering from bufadienolide glycoside

Fig. 4: *Cotyledon orbiculata* L.

poisoning induced by the administration of the blue tulp (*Moraea polystachya* (Thunb) Ker-Gawl) or Transvaal slangkop (*Urginea sanguinea* [Schinz]) with either activated charcoal alone or activated charcoal plus potassium chloride[4 5 6]. They concluded that activated charcoal alone was as therapeutically effective as the combination of activated charcoal and potassium chloride and, in a pilot trial, determined that in blue tulp poisoning in sheep, the minimal effective dose was 2 g activated charcoal per kg live mass[6].

2. *Atropine sulphate.* This was given for 2 reasons: We were initially uncertain as to whether the syndrome was caused by a combination of cotyledon and organophosphate poisoning or cotyledon poisoning alone, and the administration of the drug to a severely affected goat appeared to have a beneficial effect. It was therefore decided to administer it to all the animals.
3. *Antibiotics.* The necropsies had revealed that at least one of the 3 goats had suffered from pneumonia, that lung oedema (a potential medium for bacterial growth with consequent pneumonia development) was present in all, and that a possible early enteritis was present in 2. In order to control any possible complicating bacterial infections or to prevent their further development, it was decided to use antibiotic therapy.

There seems no doubt that the wetness around the mouth of several of the affected animals was due to salivation. Henning states that this occurs in some acutely ill animals and is due to spasm of the muscles of mastication and deglutition which makes chewing and swallowing impossible[3]. A muco-nasal catarrh has also been described in less acutely affected animals[11].

The macroscopic lesions were similar to those previousy described in animals dying from the acute to subacute disease[8 11]. Terblanche & Adelaar also recorded the presence of focal hyperaemia of the abomasum and small intestine in many instances[11]. The latter change was present in 2 of the animals we necropsied. In addition, the evidence of the presence of a diarrhoea or possible intestinal hypermotility in some of the animals has been noticed in experimental cases of *C. orbiculata* L. poisoning[11]. A bloody diarrhoea has been reported in *Kalanchoe* sp. poisoning[10].

The very watery ruminal content present in the 3 animals necropsied is of interest. No explanation can be offered as to its pathogenesis. It seems doubtful that the increased amount of fluid was derived entirely from the moisture in the succulent leaves of the ingested cotyledon and was retained because of the ruminal stasis, although this might have been partially responsible.

Similar myocardial lesions to those we observed have been recorded in the disease induced experimentally by the administration of bufadienolides or plant material of *K. lanceolata* Forsk and *T. grandiflorus* (Burm. F.) Toelken[1 2]. In the latter cases, however, the lesions were often infiltrated mainly by macrophages, lymphoblasts and lymphocytes and not by neutrophils as occurred in our cases. Newsholme & Coetzer suggest that morphological evidence of myocardial injury in cardiac glycoside poisoning of ruminants may be more consistent than has been recognized and state that microscopical examination of the myocardium should not be neglected if poisoning is suspected[9].

The monotonous diet of old man salt bush due to the drought probably played an important role in the pathogenesis of this outbreak, the goats being only too keen to vary the situation by sampling any other possibly palatable plants when the opportunity arose. *C. orbiculata* L. not only occurs in the wild state but is a popular rockery plant and this outbreak once again focusses attention to that fact that many garden plants and shrubs are indeed highly toxic.

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A BRUCELLOSIS SEROLOGICAL SURVEY ON BEEF CATTLE SLAUGHTERED AT CATO RIDGE ABATTOIR

G.C. BISHOP*

ABSTRACT: Bishop G.C. **A brucellosis serological survey on beef cattle at Cato Ridge Abattoir.** *Journal of the South African Veterinary Association* (1984) 55 No. 4, 185-186 (En). Allerton Regional Veterinary Laboratory, P. Bag X9005, 3200 Pietermaritzburg, Republic of South Africa.

A total of 5059 sera were collected from adult female beef cattle at Cato Ridge Abattoir and tested for *Brucella abortus* antibody levels over the period November, 1981 to August, 1982. The sera were screened using the Rose Bengal Plate Test, and the Complement Fixation Test was used as the definitive test. Seventy-seven sera (1,5 %) had titres of 30 IU complement fixing *B. abortus* antibody per ml serum or greater.

Key words: Brucellosis serological survey, *Brucella abortus*.

INTRODUCTION

Extrapolating from brucellosis serological test results obtained from the various veterinary laboratories and the Veterinary Research Institute at Onderstepoort in an attempt to establish the true prevalence of the disease, may result in most unreliable estimates. The sera routinely examined by these laboratories may be biased towards problem herds or towards known brucellosis-free herds participating in the certification process. There are no recently published findings of serological surveys of beef cattle in the Republic of South Africa, to indicate the brucellosis infection rate. In order to eradicate the disease it is essential that all foci of infection be located and, while this is fairly easily done in the case of dairy herds by making use of the Milk Ring Test on bulk milk samples, such an approach is not practicable in beef herds.

It was therefore decided to test unselected sera from beef cows slaughtered at Cato Ridge Abattoir in order to establish the prevalence of serological reactors and to investigate the possibility of using this approach to detect infected herds.

MATERIALS AND METHODS

The blood examined in this survey was collected by the abattoir staff at the slaughter-point into empty, sterile 10 ml test tubes. Unselected adult female beef cattle were bled and all obvious dairy or dairy-like cows were ignored for the purpose of this survey. Samples were collected from cattle as was convenient but there was no conscious selection of groups, individuals, days or times. This was in an attempt to obtain unbiased figures representative of the true field prevalence of brucellosis. The blood samples were forwarded within 24 hours to the Allerton Regional Veterinary Laboratory where the serum was decanted, following centrifugation at 1 000 g for 15 minutes, stored at 4°C and tested within 72 hours of arrival.

The Rose Bengal Test (RBT), Complement Fixation Test (CFT) and the tube Serum Agglutination Test (SAT) were carried out as described by Herr et al[1]. The CFT International Units were based on a serum control containing 1 000 IU ml^{-1} which gave a 50 % haemolysis end-point at a dilution of 1/220 as described by Herr et al[1]. For the SAT, the International Units were based on the International Unitage Table with 1 000 IU ml^{-1} end-point, defined as 50 % agglutination at a dilution of 1/500. The SAT and CFT were continued to end-points corresponding to 212 IU ml^{-1} and 784 IU ml^{-1} respectively.

The RBT was carried out on all sera and the CFT done on all RBT positive sera. All CFT sera containing 18 IU or more *B. abortus* antibody ml^{-1} were tested further using the SAT. The SAT was used primarily in this survey as a safe-guard against human error.

RESULTS

Table 1: RESULTS OF SEROLOGICAL TESTS

ROSE BENGAL TEST					
Positive		Negative		Total	
241		4818		5059	
COMPLEMENT FIXATION TEST (IU ml^{-1})					
0-15	18-24	30-49	60-196	≥240	Total
142	22	18	27	32	241
SERUM AGGLUTINATION TEST (IU ml^{-1})					
0-27	34-67	80-160	≥186	Total	
3	41	15	40	99	

DISCUSSION

Interpretation of the above results is complicated by the fact that the *B. abortus* vaccinal status of all animals was unknown. It was also often impossible to trace back reactors to their farms of origin as the slaughter stock was frequently bought at stock sales by agents who subsequently held the animals for varying periods at feedlots prior to despatch to the abattoir. The sera tested here were collected from animals originating from all four provinces of the Republic.

The official guidelines are that cattle, vaccinated as calves, be regarded as positive if their serum contains 30 IU or more *B. abortus* CF antibody ml^{-1} serum[1,2]. Adult vaccinated animals are regarded as positive if their serum contains 60 IU or more *B. abortus* CF antibody ml^{-1} serum. Applying the 30 IU or more cut-off

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point, 77 (1,5 %) of the total would fall into this category. If 60 IU or more CF antibody is regarded as indicative of infection then 59 (1,2 %) of the animals were positive. Most of the sera in the 18-49 IU ml^{-1} CF *B. abortus* antibody group yielded relatively high agglutination titres. Of the 40 animals in this category, 33 had agglutination titres which were higher than the CFT titres (82,5 %). This phenomenon is often taken as a strong indication of vaccine involvement so that the most probable estimate of infected animals was between 1,2 % and 1,5 %. The Annual Report of the Director General: Agriculture and Fisheries for the year 1980-04-01 to 1981-03-31[3] gives a figure of 150 458 head of cattle tested under the voluntary diagnostic eradication scheme. Of these, 5743 cattle tested positive, giving an incidence of 3,7 %. Earlier, for the year 1978/1979, Bosman[4] estimated the incidence to be approximately 6 %. These figures cannot, however, be compared with the findings in this survey, as both previous estimates were based on routine test returns for dairy and beef cattle obtained from the various veterinary laboratories in the Republic.

ACKNOWLEDGEMENTS

The author wishes to express his sincere thanks to Mr John Heath of the Cato Ridge Abattoir who collected the blood samples and Mrs Beverley Coetzer, Miss Peta Wood and Miss Lisa Hare who carried out the serological tests. The assistance of the Allerton-Cato Ridge Liaison Committee and in particular Dr Gareth Bath and Dr Paul Bosman who read the manuscript is also acknowledged. The author also thanks the Assistant Director of Natal for allowing this investigation.

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BOOK REVIEW

BOEKRESENSIE

THE VETERINARY ANNUAL

C.S.G. GRUNSELL and F.W.G. HILL

Twenty-fourth Issue, John Wright & Sons, Bristol, England 1984 pp xv + 348 Figs 103 Tabs 30 ISBN 0 85608 1
Price £18.00

After nearly a quarter of a century of issues the Veterinary Annual has firmly established itself as the one publication in book form which is essential reading for all veterinarians irrespective of their particular field of interest. Indeed, one of the forty three contributions deals with general aspects of veterinary specialization. This is one of four special articles, the other three dealing with advances in animal husbandry, reproduction and infertility and protein-losing enteropathy in the horse. The fifteen contributions dealing with food animals concentrate mainly on erosive diseases in which recent important advances have been made. These topics include subclinical ketosis and the role of the milking machine in udder infection in dairy cows, the epidemiology and control of hydatid disease in sheep and mange, cystitis and pyelonephritis in sows and piglet anaemia in pigs. The computer as an aid in herd recording systems has bot been neglected as there is an article on COSREEL (Computer System for Recording Events in Economically-important Livestock).

Contributions on Equine Veterinary Medicine are better represented than in previous Annuals and include articles on navicular disease and its treatment, diarrhoea in foals and the development of diagnostic techniques for equine viral disease.

The small animal section with nineteen contributions is diverse and is aimed at the small animal practitioner. I particularly enjoyed reading the articles on the prophylactic use of antibacterials, the application of herbal medicine in veterinary medicine, canine cardiomyopathy and exfoliative cytology in small animals.

Most of the articles are well written and concise and the book can best be read by selecting a topic when the odd half hour presents itself, much as one would read a collection of short stories or poems.

M.C. Williams

ANTIBIOTIC SUSCEPTIBILITY PATTERNS OF MASTITIS PATHOGENS ISOLATED FROM BLOEMFONTEIN DAIRY HERDS

RIANA SWARTZ*, P.J. JOOSTE* and J.C. NOVELLO*

ABSTRACT: Swartz, R.; Jooste, P.J.; Novello J.C. **Antibiotic susceptibility patterns of mastitis pathogens. isolated from Bloemfontein dairy herds** *Journal of the South African Veterinary Association* (1984) **55** No. 4, 187-193 (En). Department of Dairy Science, Faculty of Agriculture, University of the Orange Free State, P.O. Box 339, 9300 Bloemfontein, Republic of South Africa.

The antibiotic susceptibility patterns of bacteria associated with subclinical mastitis in Bloemfontein fresh milk dairy herds were determined. A total of 141 bacterial strains tested, consisted of *Staphylococcus aureus* (93 strains), coagulase negative staphylococci (17), streptococci (12), *Corynebacterium bovis* (8), *Pseudomonas aeruginosa* (7) and enterobacteria (4). Antibiotic susceptibility was determined qualitatively using the Kirby-Bauer disc diffusion method and quantitatively by determining the minimal inhibitory concentrations using the agar dilution method. The utilization of commercial intramammary antibiotic preparations on the dairy farms is also discussed. The Gram-positive bacteria were generally not exceptionally resistant to the antibiotics tested. *S. aureus* susceptibility figures for penicillin G were 66 % and for methicillin (or cloxacillin) 100 %. The coagulase negative staphylocci in contrast were relatively more resistant than the coagulase positive staphylococci. The enterobacteria and particularly the *P. aeruginosa* strains, were extremely resistant to all antibiotics tested. In the latter case even carbenicillin and gentamicin susceptibility figures were low. A general mastitis control programme is discussed.

Key words: mastitis pathogens, antibiotic susceptibility, bovine mastitis, *Staphylococcus aureus*.

INTRODUCTION

Information regarding the antibiotic susceptibility of mastitis pathogens, particularly the staphylococci, is limited and often not readily comparable[3]. The purpose of this paper is to report the antibiotic susceptibility patterns of mastitis pathogens isolated during a recent survey in the Bloemfontein area[18]. This study will serve to supplement the sparse Southern African data[2,19,20] available in the important field of mastitis therapy.

MATERIALS AND METHODS

Bacteria tested

A total of 141 bacterial strains from cases of bovine subclinical mastitis consisted of *Staphylococcus aureus* (93 strains), coagulase negative staphylococci (17), streptococci (12), *Corynebacterium bovis* (8), *Pseudomonas aeruginosa* (7) and enterobacteria (4). With the exception of 3 *P. aeruginosa* strains isolated from an industrial hand-milked herd, all organisms were isolated from machine-milked fresh milk dairy herds in the Bloemfontein district. The bacteria were stored in lyophilized form as previously described[18].

Antibiotic susceptibility testing

Disc diffusion test

Antibiotic susceptibility patterns of the bacteria were determined using the Kirby-Bauer disc diffusion method with strict adherence to the method and standards as described by the National Committee for Clinical Laboratory Standards[12]. Interpretive zone sizes for oleandomycin and novobiocin were obtained from a Federal Register publication[7]. Antibiotic discs were supplied by Mast Laboratories and were stored at −23°C. The following antibiotics and disc concentrations were used: ampicillin 10 μg, cephalothin 30 μg, chloramphenicol 30 μg, clindamycin 2 μg, kanamycin 30 μg, methicillin 5 μg, neomycin 30 μg, novobiocin 30 μg, oleandomycin 15 μg, penicillin G 10 IU, polymyxin B 300 IU, streptomycin 10 μg and tetracycline 30 μg. Enterobacteria en *P. aeruginosa* were also tested against carbenicillin (100 μg disc) and gentamicin (10 μg disc).

All tests were done in duplicate. Mueller-Hinton agar (Difco) was used as culture medium. The agar medium was poured in 25 ml counts per 90 mm diameter petri dish. Agar plates were dried at 35°C for 2 h immediately before use. The lyophilized bacterial cultures to be tested were reactivated by inoculation into Difco brain-heart infusion broth (staphylococci), Oxoid nutrient broth (*P. aeruginosa* strains and enterobacteria), Oxoid Todd-Hewitt broth (streptococci) and Oxoid nutrient broth enriched with 7 % human serum (corynebacteria). After 24 h incubation at 35°C a loopful of the broth culture was streaked onto a Difco plate count agar plate and incubated overnight. Five discrete, similar colonies were picked up and inoculated into an appropriate broth. After 12 – 18 h incubation the turbidity of the broth culture was standardized against a $BaSo_4$ turbidity standard[12]. A mechanical vortex mixer was used for shaking the cultures to obtain a homogeneous suspension. Agar plates were inoculated by flooding the plate with 1-2 ml of the standardized broth culture. The excess fluid was poured off and the remaining drops soaked up using a sterile tissue paper swab. Antibiotic discs were applied by means of sterile needles. The inoculated plates were incubated at 35°C for 16-20 h. Zone sizes were read using a sliding caliper. *S. aureus* ATCC 25923 was included as a Gram-positive control and *P. aeruginosa* ATCC 10662 and *Escherichia coli* ATCC 25922 as Gram-negative controls.

Minimal inhibitory concentration determinations

Minimal inhibitory concentration (MIC) determinations using the agar dilution method[21], were performed on all staphylococcal strains showing resistance to one or more of the following antibiotics used in the disc diffusion test. These antibiotics including penicillin G, streptomycin and tetracycline, are the most frequent active ingredients of commercial intramammary preparations. *P. aeruginosa* strains were tested against a range of streptomycin, tetracycline, polymyxin B, carbenicillin and gentamicin concentrations.

Standard antibiotic powders were kindly supplied by the following companies or institutions: oxytetracycline hydrochloride by Pfizer Laboratories, Johannesburg; streptomycin sulphate and procaine penicillin G by

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Milborrow, Germiston; and polymyxin B, carbenicillin and gentamicin sulphate by the Department of Medical Microbiology, University of the Orange Free State, Bloemfontein. Antibiotic powders were stored at −23°C.

Stock solutions of the various antibiotics were prepared at a concentration of 2 000 μg/ml or IU/ml, depending on the antibiotic. From the stock solutions a range of intermediate concentrations were prepared. These solutions were aseptically added to sterilized Mueller-Hinton agar (Difco), previously cooled to 50°C. The range of antibiotic concentrations in the prepared agar plates are given in Table 1. A ½ dilution range was used for all antibiotics.

Table 1: RANGE OF ANTIBIOTIC CONCENTRATIONS TESTED IN MIC DETERMINATIONS

Antibiotic	Range of Concentrations
Penicillin G	0,25 – 128 μg/ml
Streptomycin	0,25 – 256 IU/ml
Tetracycline	0,25 – 256 μg/ml
Polymyxin B	0,25 – 128 IU/ml
Gentamicin	0,25 – 128 μg/ml
Carbenicillin	0,25 – 512 μg/ml

The bacterial cultures to be tested were prepared in the same way as for the disc diffusion test. The standardized inocula were applied to the antibiotic agar plates using a replicating device (P.J. Jooste 1983 Dept. Dairy Science, University O.F.S., personal communication). Plates were incubated aerobically at 35°C for 16-20 h. *S. aureus* ATCC 25923 and *E. coli* ATCC 25922 were included as control strains. The MIC was regarded as the lowest concentration/ml at which no growth occurred on the plates.

Testing of penicillin G – streptomycin combinations

Concentration combinations of 2:1, 1:1 and 1:2 of penicillin G and streptomycin, respectively, were tested. The antibiotic concentration range tested was 3,75 – 240 IU streptomycin/ml and 3,75 – 240 μg penicillin G/ml. Methods used for preparation of antibiotic solutions, agar plates, broth cultures, inoculation and incubation, were as described for the MIC determinations.

Utilization of commercial intramammary antibiotic preparations on dairy farms

Information regarding the frequency of utilization and the type of antibiotic preparations used in the dairy herds was obtained from the milk producers in the Bloemfontein area.

RESULTS

Table 2 summarizes the results of the antimicrobial susceptibility testing using the disc diffusion method. Sixty-six percent of the *S. aureus* strains were susceptible to penicillin G. Susceptibility to other major antibiotics were as follows: streptomycin (76 %), tetracycline (97 %) and methicillin (cloxacillin) (100 %). *S. aureus* strains were also largely susceptible to cephalothin, chloramphenicol, clindamycin, kanamycin, lincomycin, neomycin, novobiocin and oleandomycin. The few *P. aeruginosa* and enterobacterial strains were resistant to a wide range of antibiotics. Low *P. aeruginosa* susceptibility figures were found even for carbenicillin (29 %) and gentamicin (57 %). The coagulase negative staphylococci appeared to be more resistant to the antibiotics than *S. aureus* strains. The streptococcal isolates were highly susceptible to most of the antibiotics tested. Streptomycin (25 % susceptible) and tetracycline (67 % susceptible) were however less effective against this group of organisms.

Table 2: ANTIBIOTIC SUSCEPTIBILITY OF MASTITIS PATHOGENS TESTED BY THE KIRBY-BAUER DISC DIFFUSION METHOD

ANTIBIOTICS TESTED	PREVALENCE OF SUSCEPTIBILE ISOLATES					
	S. aureus	Coagulase negative staphylococci	Streptocci	*C. bovis*	*P. aeruginosa*	Enterobacteria
	% of 93 strains	% of 17 strains	% of 12 strains	% of 8 strains	% of 7 strains	% of 4 strains
Ampicillin[1]	68	71	100	75	—	—
Cephalothin[2]	100	100	100	100	0	25
Chloramphenicol	99	100	100	100	0	25
Clindamycin[3]	99	94	100	100	0	0
Kanamycin[4]	95	76	8	88	0	25
Lincomycin	99	94	100	100	0	0
Methicillin[5]	100	100	100	100	—	0
Neomycin	99	82	0	75	14	0
Novobiocin	100	100	100	100	0	0
Oleandomycin	99	94	100	100	0	0
Penicillin G	67	71	100	75	—	—
Streptomycin	76	59	25	63	0	0
Tetracycline[6]	97	88	67	88	0	25
Gentamicin	—	—	—	—	57	100
Carbenicillin	—	—	—	—	29	75
Polymyxin B	—	—	—	—	100	25

1. Group-representative disc for ampicillin, hetacillin and amoxillin
2. Group-representative disc for cephalosporins
3. Clindamycin disc is used to test for clindamycin as well as lincomycin
4. Kanamycin disc also tests for framycetin
5. Group-representative disc for penicillinase-resistant penicillins, i.e. methicillin, cloxacillin, dicloxacillin, oxacillin and nafcillin
6. Group-representative disc for tetracyclines

Table 3: MIC VALUES FOR ANTIBIOTICS TESTED AGAINST MASTITIS PATHOGENS

ANTIBIOTIC	ORGANISM	No. of* strains tested	Cumulative % inhibited at various conc. (μg or IU/ml)										
			≤ 0,025	0,5	1	2	4	8	16	32	64	128	256
Penicillin G (μg/ml)	*S. aureus*	44	34	36	39	52	75	89	100				
	Coagulase negative staphylococci	10	50				60	80	90	100			
Streptomycin (IU/ml)	*S. aureus*	44				2	43	66	68				77
	Coagulase negative staphylococci	10						50			70		
	P. aeruginosa	7										100	
Tetracycline (μg/ml)	*S. aureus*	44				93						100	
	Coagulase negative staphylococci	10				80					90	100	
	P. aeruginosa	7									100		
Polymyxin B (IU/ml)	*P. aeruginosa*	7									14	100	
Gentamicin (μg/ml)	*P. aeruginosa*	7					14	100					
Carbenicillin (μg/ml)	*P. aeruginosa*	7									57	71	100

* All organisms tested for MIC values were resistant to one or more antibiotics as determined by the Kirby-Bauer method.

The results of the MIC determinations are shown in Table 3. The activity of penicillin G, streptomycin and tetracycline against the strains tested is illustrated in Figs. 1, 2 & 3. The MIC's of polymyxin B, carbenicillin and gentamicin against *P. aeruginosa* strains are illustrated in Fig. 4.

The MIC results confirm the findings of the disc diffusion tests in that the coagulase negative staphylococci were more resistant to antibiotics in vitro than *S. aureus* strains. Higher concentrations of penicillin G and tetracycline were required to inhibit the former group of organisms. At a streptomycin concentration of 256

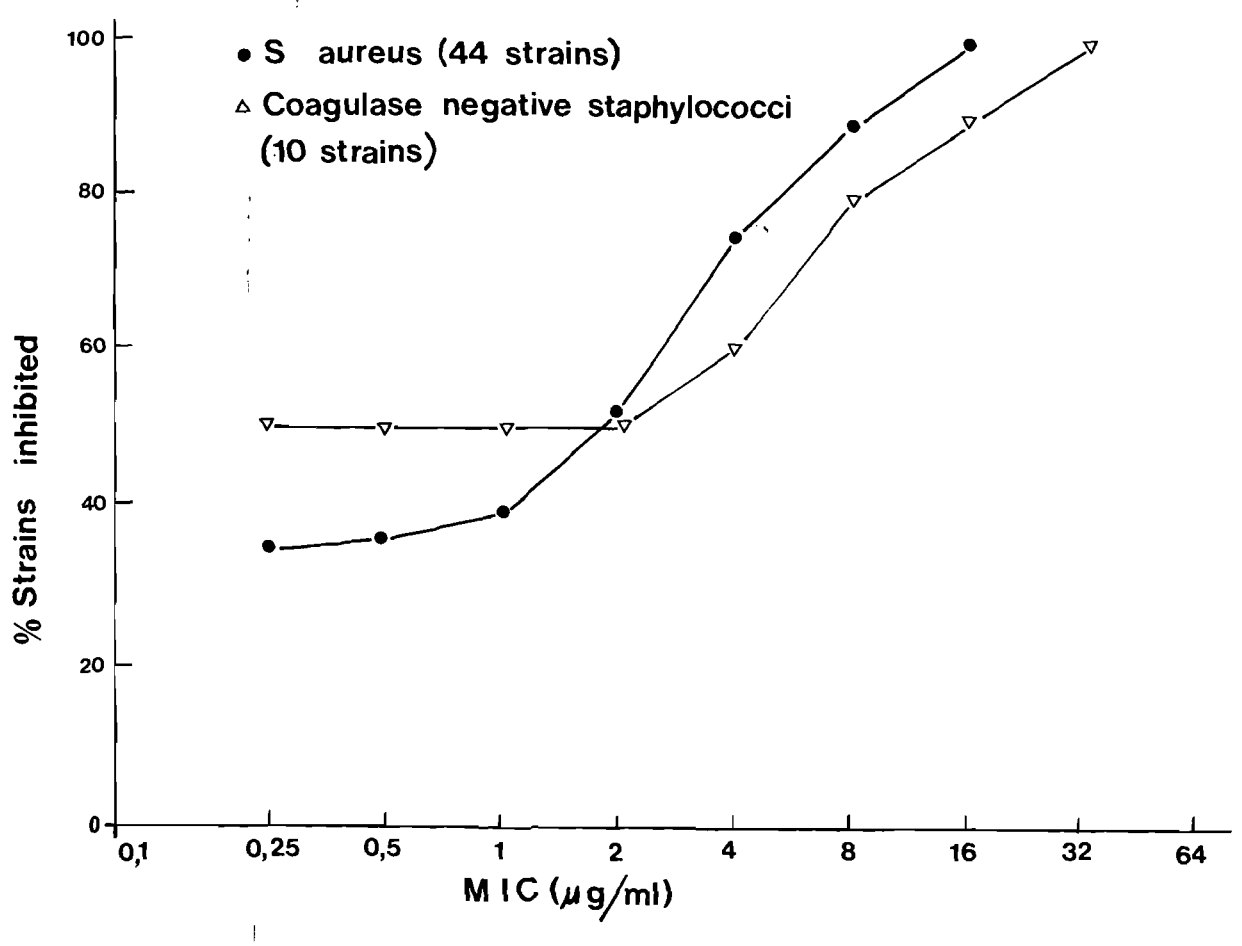

Fig. 1: Activity of penicillin G against antibiotic resistant mastitis bacteria.

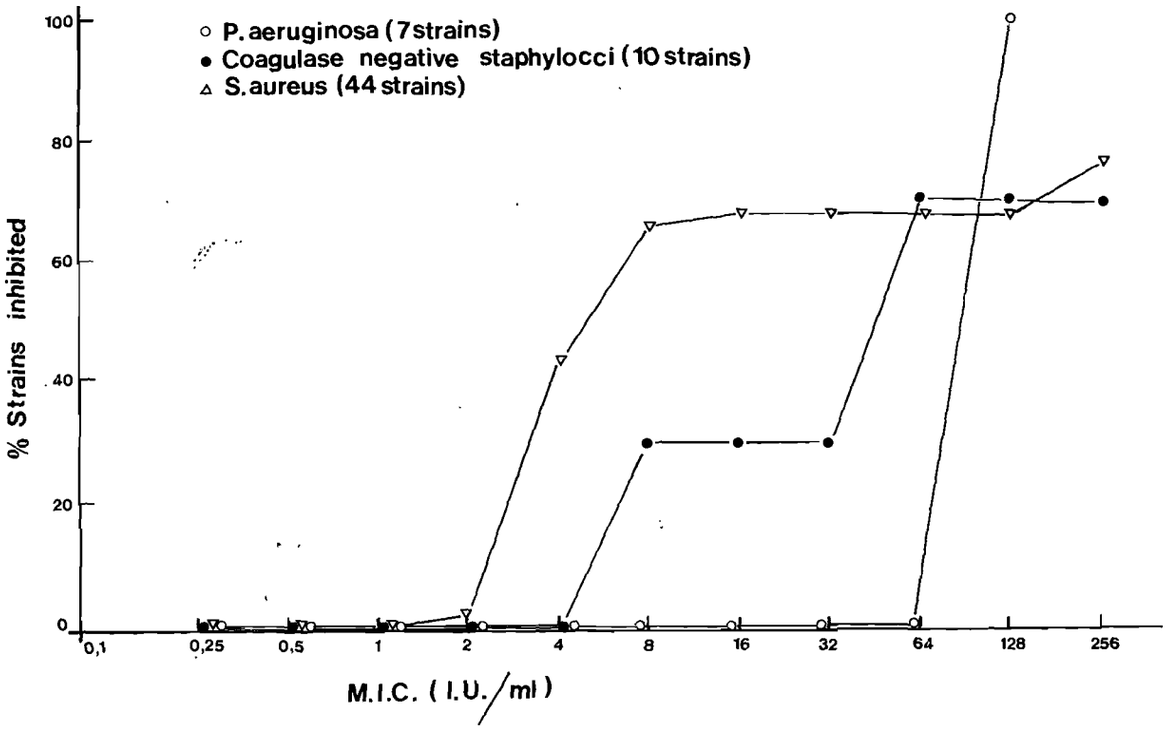

Fig. 2: Activity of streptomycin against antibiotic resistant mastitis bacteria.

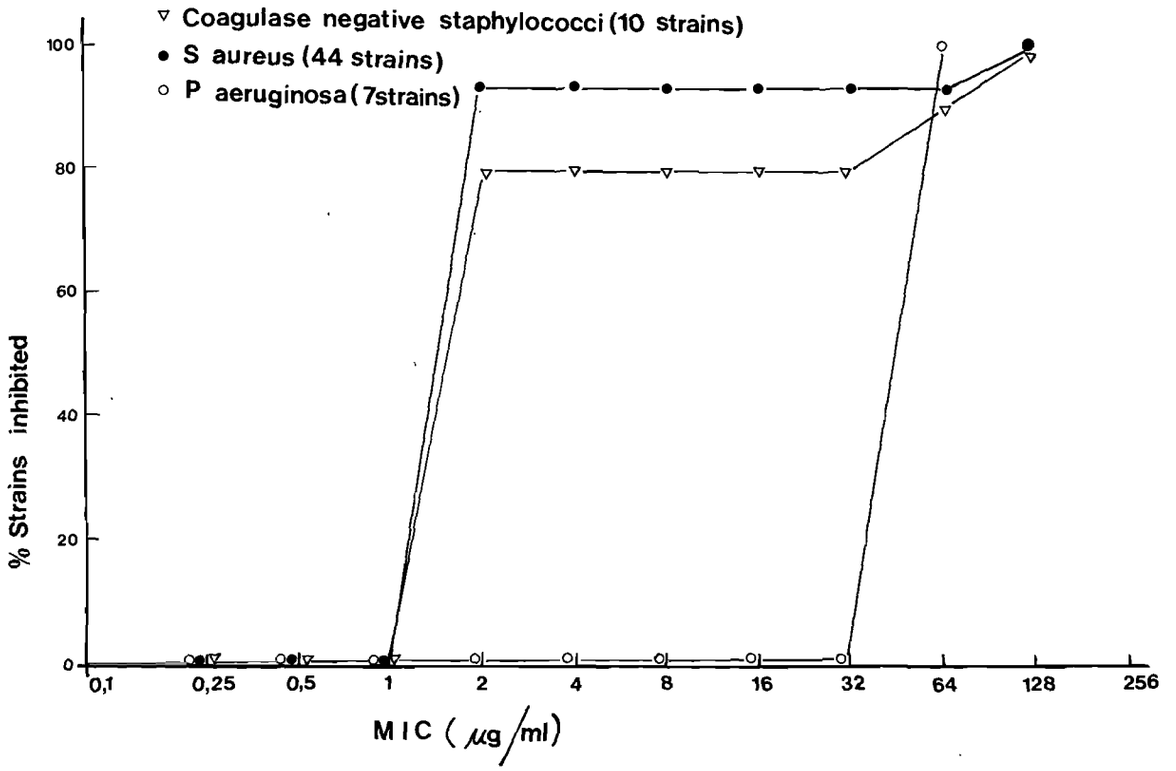

Fig. 3: Activity of tetracycline against antibiotic resistant mastitis bacteria.

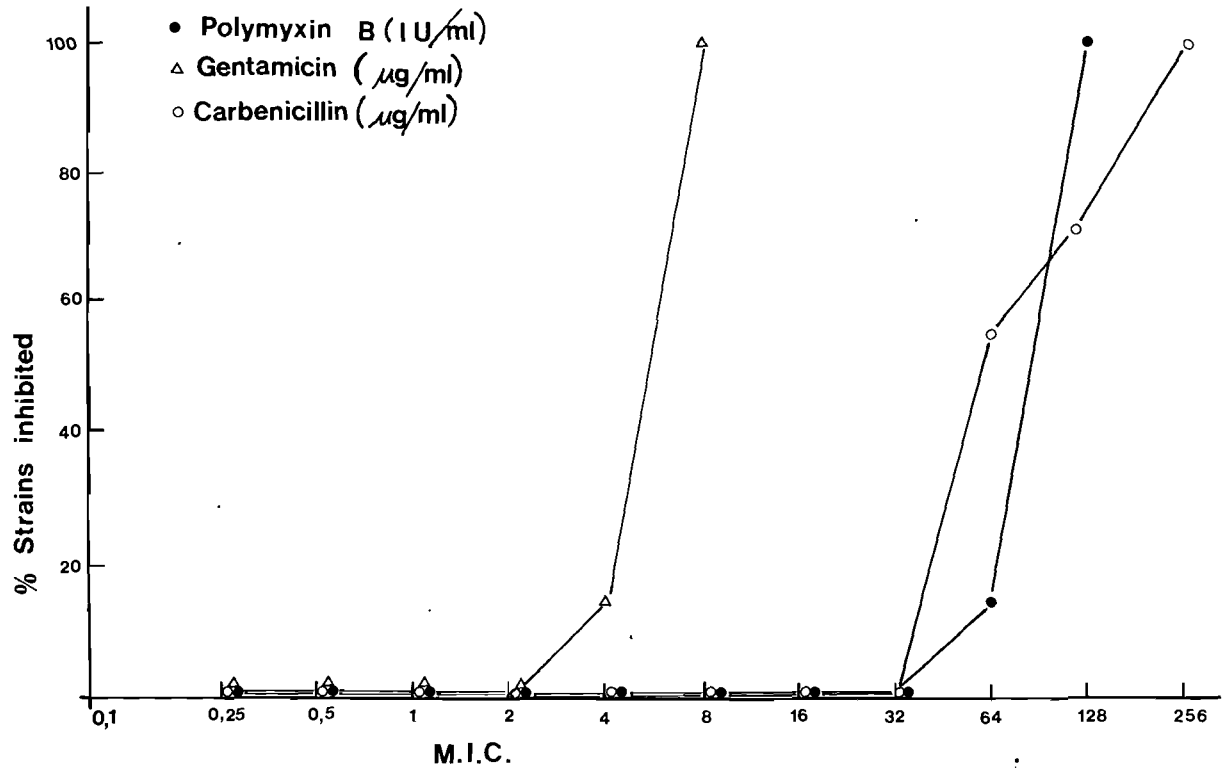

Fig. 4: Activity of polymyxin B, gentamicin and carbenicillin against *P. aeruginosa* (7 strains).

IU/ml, for example 77 % of *S. aureus* strains were inhibited compared with 70 % of coagulase negative staphylococci.

Results of the penicillin G-streptomycin combination testing indicated that the combinations were not more effective in inhibiting the test strains than the antibiotics individually. In the case of *S. aureus*, 16 μg penicillin G/ml and >256 IU streptomycin/ml respectively were required to inhibit 100 % of the strains. A combination of 15 μg penicillin G/ml and 7,5 IU streptomycin/ml gave similar results. No synergism could therefore be claimed in the case of *S. aureus*. All coagulase negative staphylococci were inhibited individually by 32 μg penicillin G/ml or by more than 256 IU streptomycin/ml. A combination of 15 μg penicillin G/ml and 7,5 IU streptomycin/ml had the same effect. Although these latter results display a trend towards synergism, no definite conclusions can be made. The lowest concentration combination that inhibited the *P. aeruginosa* strains was 120 μg penicillin G/ml and 120 IU streptomycin/ml. As all strains were susceptible to 128 IU streptomycin/ml used alone, no deduction of synergism could be made.

Sixty percent of the dairy farmers used intramammary preparations containing tetracycline, 50 % using the same brand. Penicillin G-streptomycin combinations were also popular and were used by 30 % of the farmers. The remaining 10 % used a cloxallin-ampicillin combination. In order to compare the efficiency of the popular commercial preparations the laboratory susceptibility figures of the antibiotics contained in these preparations are reported in Table 4. Susceptibility figures for penicillin G and streptomycin are reported individually as no synergism could be proven in this study. Table 4 clearly indicates that methicillin (cloxacillin) was the most effective popular antibiotic used against Gram-positive bacteria. All antibiotics listed were however ineffective against the Gram-negative bacteria tested.

DISCUSSION

Standardization of methods for antimicrobial susceptibility testing

Information regarding the antibiotic susceptibility patterns of mastitis pathogens in South Africa is limited and often not readily comparable. A serious shortcoming of publications is that the methods and materials used for testing are not clearly specified. It is highly recommendable to include control organisms with known susceptibilities to the agents being tested[15]. None of the South African publications however referred to the use of such control strains. Where a standardized method is not used or referred to, the media, antibiotics disc concentrations, criteria of interpretation and technique details should be described. Standardized susceptibility data will greatly facilitate comparison with similar surveys in different parts of the country by different workers.

Comparison of results with relevant surveys

The incidence of penicillin G resistant *S. aureus* strains in the Bloemfontein area was 32 %. This figure is slightly lower than that determined in one survey, i.e. 36,1 %[19], but considerably lower than in the other surveys, i.e. 41 % (Bulawayo)[2], 42,5 % (Pretoria)[20] and 47,8 % (Switzerland)[10].

There was considerable variation in the susceptibility of *S. aureus* to other antibiotics. Bloemfontein *S. aureus* strains were more susceptible to streptomycin (76 %) than in the other South African surveys, i.e. 59 %[2 19] and 69 %[20]. Susceptibility to tetracycline was on the same level in both the Bloemfontein survey and that of Van den Heever & Giesecke[20]. The figures in the

other surveys were however considerably lower, i.e. 83 %[19] and 70 %[2]. The low resistance of Bloemfontein isolates to tetracycline and streptomycin was a bit surprising since intramammary preparations containing these antibiotics were used extensively by dairy farmers in this area and a build-up of resistance might have been expected.

S. aureus strains were highly susceptible to chloramphenicol and neomycin in the Bloemfontein survey, as well as in the other 3 surveys[2 19 20], although the neomycin susceptibility figures for Bulawayo strains were slightly lower, i.e. 84 %[2].

The only recent South African survey on antibiotic susceptibilities of mastitis pathogens other than *S. aureus* was conducted in hand-milked cows with clinical mastitis in the Bulawayo surveys[2]. The considerable differences in results between this and the Bloemfontein survey can be summarized as follows:

1. The coagulase negative staphylococci in the Bloemfontein area were appreciably more resistant to streptomycin, tetracycline, chloramphenicol and neomycin. The susceptibility to penicillin G was similar.
2. Antibiotic susceptibilities of the corynebacteria were approximately the same, but Bloemfontein strains were more resistant to penicillin G.
3. Bloemfontein *P. aeruginosa* and enterobacteria were more resistant to all antibiotics tested than in the Bulawayo survey.

Antibiotic resistance of coagulase negative staphylococci and *P. aeruginosa*

An important finding in this study was the relatively high antibiotic resistance of the coagulase negative staphylococci. This group of bacteria was more resistant than *S. aureus* to the following major antibiotics: penicillin G, tetracycline, streptomycin, neomycin and kanamycin. As reported previously[18], 65 % of the coagulase negative staphylococci was isolated from udder-quarters with somatic cell counts of more than 10^6/ml, stressing the possible pathogenic role of these organisms.

The Gram-negative isolates, particularly *P. aeruginosa*, were extremely resistant to most antibiotics tested. Only polymyxin B, carbenicillin and gentamicin were relatively effective against these organisms. Since these antibiotics are not included in veterinary intramammary preparations, it is unlikely that infections will respond to available preparations. The use of gentamicin and carbenicillin for the treatment of bovine mastitis is undesirable for the following reasons: In the first place 86 % of Bloemfontein strains of *P. aeruginosa* were resistant to gentamicin (Table 2), having a MIC of > 8 µg/ml. Secondly, the indiscriminate use of gentamicin for animal therapy may cause a rise in the general incidence of gentamicin resistance as has been the case with *P. aeruginosa* strains from human clinical specimens in a general hospital[6]. This antibiotic, alone or in combination with carbenicillin has been the antibiotic of choice for the treatment of serious human *P. aeruginosa* infections since 1968[11]. It may therefore be wise to reserve this antibiotic solely for human therapeutic purposes and to employ culling of chronically affected cows as a means of stopping the spread of Gram-negative infections.

Effectivity of antibiotics in general use

It has been thought necessary to compare the results of antibiotic susceptibility testing with the types of antibiotics most commonly used on dairy farms in the district. Tables 2 & 3 clearly indicate that cephalothin, chloramphenicol, novobiocin and the semi-synthetic penicillins (methicillin, cloxacillin, dicloxacillin, oxacillin and nefcillin) were appreciably more effective against the pathogens isolated than the tetracycline and penicillin G-streptomycin combinations used by 90 % of farmers. As 94 % of all organisms isolated were Gram-positive bacteria, the use of cephalothin, novobiocin and the semi-synthetic penicillins is recommended for intramammary lactating and dry cow preparations. The pathogens tested were all 100 % susceptible to these antibiotics. Although chloramphenicol susceptibility figures for Gram-positive bacteria were exceptionally high, this drug is not recommended due to its poor activity in vivo[11].

Recommendations for an effective mastitis control program

It is estimated by Giesecke[8] that only 4 out of every 10 cows in South Africa have healthy udders, one having clinical and 5 having subclinical mastitis in one or more of their udder-quarters. The applications of an effective mastitis control program in South African herds is therefore essential. The application of a basic control program in commercial herds in the U.K. and the U.S.A. have reduced infection levels by 50 % within one year and by 75 % to 85 % within 2 years[16]. Mastitis infection in the U.S.A. was reduced to a level of less than 8 % of all udder-quarters[14]. The somatic cell count of bulk milk was concurrently reduced from 730 000 to 320 000 within 3 years[16]. A similar control program has been successfully employed with Natal fresh milk herds[1].

The following basic control program of Dodd and Neave[5] is therefore strongly recommended for application in Bloemfontein and South African dairy herds:

TABLE 4: KIRBY-BAUER SUSCEPTIBILITY FIGURES (PERCENTAGE FOR COMMONLY USED ANTIBIOTICS)

ORGANISM	ANTIBIOTIC				
	Ampicillin	Methicillin	Penicillin G	Steptomycin	Tetracycline
S. aureus	68	100	67	76	97
Coagulase negative staphylococci	71	100	71	59	88
Streptococci	100	100	100	25	67
C. bovis	75	100	75	63	88
P. aeruginosa	—	—	—	0	0
Enterobacteria	0	0	—	0	25

1. Regular teat dipping using an effective teat dip immediately after the milking machine has been removed.
2. Cloxacillin therapy under veterinary supervision to *all* cows at drying off and to clinically affected quarters.
3. Culling cows with recurrent clinical mastitis and cows with treatment-resistant infections, particularly Gram-negative infections.
4. Good general husbandry and milking practices. This includes the regular maintenance of the milking machine and reduction of vacuum fluctuations in the teat cups.

The routine use of cloxacillin-containing lactating and dry cow preparations is strongly recommended, as the dominant group of Gram-positive bacteria was 100 % susceptible to cloxacillin. The objection that repeated dry cow therapy could cause a build-up of antibiotic resistance, is probably not valid because staphylococcal infections are most efficiently eliminated in the dry udder[22]. Furthermore, cloxacillin has been used in the U.K. for the past 9 years and no resistance has so far been determined[16].

A policy of dry cow therapy for *all* cows in a herd has been shown to be the most practical and economical method of reducing the level of infecton in the dry udder[13]. Although selective dry cow therapy for older cows and cows with a mastitis record will reduce the cost of antibiotics considerably, it appears to be uneconomical in the long run[4]. As facilities for the diagnosis of cows with subclinical mastitis in South Africa are limited, selective therapy will often exclude cows with subclinical infections. This may cause mastitis related problems in the future. Dry cow treatment is also a most effective method of treating subclinical and *S. aureus* infections[23].

The culling of cows with chronic or recurrent mastitis infections is a component of the recommended mastitis control program that is not always readily accepted by dairy farmers. Culling would remove a primary source of mastitis pathogens from the premises and reduce a substantial percentage of the clinical cases. It has also been mooted[16] that only 7 % of the cows in a herd account for 40 % of all clinical mastitis cases. This level of culling should be economically acceptable.

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A REPORT OF SWINE ERYSIPELAS IN A LITTER OF PIGLETS

STELLA S. BASTIANELLO* and B.T. SPENCER**

ABSTRACT: Bastianello S.S.; Spencer B.T. **A report of swine erysipelas in a litter of piglets.** *Journal of the South African Veterinary Association* (1984) **55** No. 4, 195-198 (En). Department of Pathology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

Out of a litter of 7 two-week old Landrace piglets, 6 developed cutaneous haemorrhages especially on the limbs and ears. Two of these piglets died within 24 hours of the haemorrhages appearing whilst the other 4 recovered following penicillin therapy.

The histopathological lesions were centred around the smaller vessels of the dermis and hypodermis. These included hyperaemia, leukostasis and intravascular fibrin coagulation or thrombosis. Bacterial emboli were present within the vessels of the skin, spleen, liver and kidney and loose in the areolar tissue of the dermis and hypodermis. Other lesions included scattered but extensive dermal and hypodermal haemorrhages and a mild cellular infiltration of the dermis and hypodermis.

INTRODUCTION

Swine erysipelas is a porcine disease caused by the bacterium *Erysipelothrix rhusiopathiae (insidiosa)*. It usually manifests as one of 3 forms, namely, the acute form, the subacute form and the chronic form. The acute form is septicaemic in nature and is often referred to as "diamond skin disease" due to the development of raised, urticarial, cutaneous, haemorrhagic lesions which are generally rhomboidal in shape. The subacute form is similar to the acute form but less severe whilst the chronic form develops following on the acute/subacute form or consequent to a subclinial infection. The latter form is characterised by chronic arthritis and valvular endocarditis[2]. All forms can precipitate abortion in the pregnant sow.

The disease usually affects pigs between 3 weeks and 3 months of age and often occurs shortly after weaning or other stressful conditions. Piglets less than 3 weeks of age are generally immune to the disease due to the presence of antibodies which develop in the gilt or sow following subclinical infection or vaccination[2].

The outbreak, as dealt with in this report, describes the clinical symptoms and pathology of acute erysipelas in a litter of piglets two weeks of age.

HISTORY AND CLINICAL SIGNS

Out of a litter of 7 two-week old Landrace piglets born of a gilt, 2 piglets died suddenly, 4 became sick whilst the 7th piglet was not visibly affected. The 2 piglets that died initially developed a blue haemorrhagic discolouration of the claws and limb extremities. The discoloured areas spread to involve the skin of the entire length of the limbs, the sides of the thorax and abdomen and the ears. The piglets had appeared dejected, feverish and anorexic and died within 24 hours of the discolouration having first been noticed. The 4 clinically affected piglets also developed haemorrhagic lesions of the limb extremities and non-specific signs such as anorexia and dejection. The haemorrhagic lesions did not, however, spread to involve other parts of the body but instead they disappeared and the piglets recovered following long-acting penicillin treatment (Compropen V, Glaxo).

No other litters were affected but within 2 weeks of the piglet mortalities, 3 gilts developed typical rhomboidal haemorrhagic skin lesions which subsequently disappeared. Another gilt aborted one month post-service. The dam of the affected litter was not clinically affected in any way.

All gilts on the farm received 2 vaccinations 3 weeks apart before their first mating and then again 3 weeks before farrowing. The sows are vaccinated 3 weeks before farrowing and the piglets at 7 weeks of age when they are weaned and put on to a growth ration. The vaccine used is the Onderstepoort Swine Erysipelas vaccine which is an alumprecipitated bacterin containing the immunogenic serotypes 1 and 2 of *E. rhusiopathiae.*

MATERIALS AND METHODS

Pathological examination

Post-mortem examinations were performed on the 2 piglets that died. Specimens of affected areas of the skin as well as of the spleen, liver and kidney were taken in 10 % buffered formalin for histopathological examination. Sections of these specimens were routinely cut and stained with the haematoxylin and eosin (HE), Giemsa and Gram stains.

Bacteriological examination

Specimens of the skin, liver, kidney, spleen and brain from both piglets were submitted to the Bacteriology section of the Veterinary Research Institute for routine aerobic bacterial isolations and specifically for *E. rhusiopathiae* isolation.

RESULTS

Bacteriology

E. rhusiopathiae was isolated from the brain, spleen, liver and kidney of both piglets but not from the skin due to overgrowth by contaminants.

Pathology

The post-mortem findings in the 2 piglets were similar and the description set out below applies to both piglets.

Macroscopical findings

Haemorrhages of varying shapes and sizes from pinpoint up to a few centimetres across occurred on the skin of the limbs, the ventral abdomen, the lateral thorax and abdomen and the face and ears. The limbs below the level of the tarsus showed a mild oedema and blue discolouration due to cutaneous hyperaemia and

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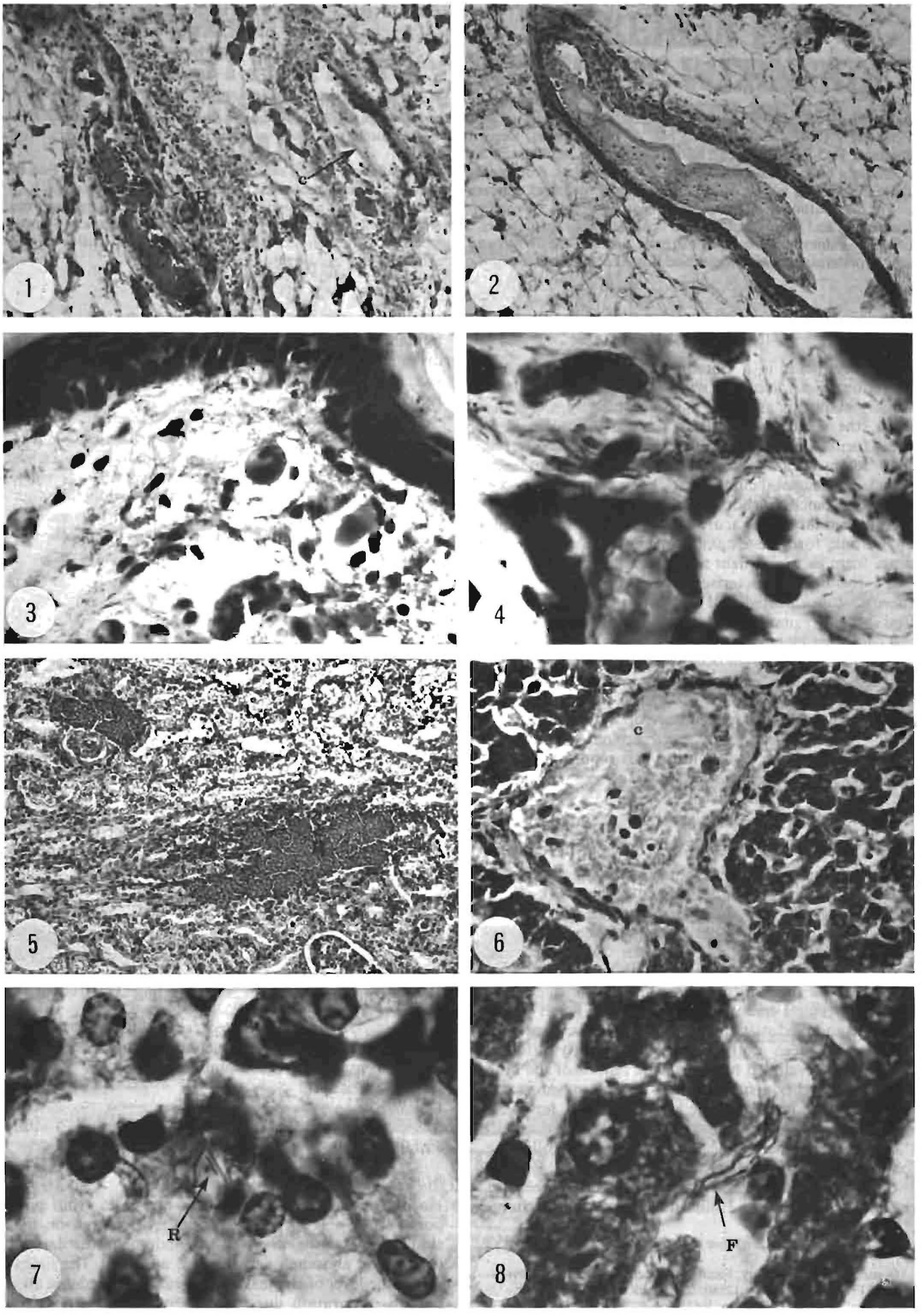

confluence of the haemorrhagic areas.

There was also a generalized congestion of the skeletal musculature and internal organs, mild splenomegaly and hepatomegaly and a few petechiae in the epicardium and renal cortices.

Microscopical findings

Skin: The cutaneous lesions were centred in and around the dermal and hypodermal vessels. The epidermis did not show any specific lesions, merely a mild focal vacuolar degeneration of the stratum spinosum probably as a result of the vascular lesions.

The principal vessels affected were the dermal capillaries and arterioles and the small to medium hypodermal venules. The vascular changes included hypervascularisation, hyperaemia, leukostasis, intravascular coagulation and thrombosis (Figs. 1 & 2). Hyperaemia was seen in all the dermal and hypodermal vessels whilst hypervascularisation was most pronounced in the dermal papillae and involved the dermal capillaries. Leukostasis occurred in the majority of cutaneous vessels. The cells involved were predominantly monocytes, neutrophils to a lesser extent and occasional lymphocytes. Intravascular fibrin coagulation occurred in several dermal capillaries and arterioles whilst, predominantly fibrin thrombi occurred in several hypodermal venules (Figs. 1 & 2). Scattered but extensive haemorrhages stretching from the hypodermis to just below the epidermis were also present.

A moderate inflammatory reaction occurred in the dermis and to a lesser extent in the hypodermis. This reaction was particularly prominent perivascularly. The inflammatory cells were predominantly monocytes, neutrophils, the latter occasionally forming small aggregates, and scattered lymphocytes. A few dermal arterioles showed fibrinoid necrosis and neutrophil infiltration of their walls.

Bacteria were faintly evident in the HE-stained sections within the lumens of vessels especially those showing leukostasis, intravascular coagulation or thrombosis, or loose in the areolar tissue of the dermis and hypodermis. These bacteria were clearly evident with the Gram stain. They occurred either as curved or straight rods, a few μm in length and occurring singly or in short chains, or as long thin filaments often clumped together and estimated to be 15-20 μm in length (Figs. 3 & 4). The Giemsa stain did not show up the organisms as clearly as the Gram stain.

Kidney: The kidney showed severe hyperaemia and scattered cortical haemorrhages (Fig. 5). Many vessels showed leukostasis occurring either on its own or together with intravascular fibrin coagulation and bacterial emboli. The appearance of the bacteria within the vessels was as described under the skin. The tubular cells showed mild degenerative changes and dilatation of a few tubules.

Liver: The hepatic lesions included mild degeneration of the hepatocytes and vascular lesions as described in the kidney. The vessels affected included the sinusoids and the larger veins (Figs. 6, 7 & 8).

Spleen: The red pulp was contracted but hyperaemic. There was hyperplasia of the reticulo-endothelial cells. The principal cells present within the sinusoids were monocytes and neutrophils. Lymphoid cells were scarce.

The white pulp was reduced to a narrow rim of lymphoid tissue around the central arteries and lymphoid follicles were absent. Neutrophils were present in the marginal zone and amongst the lymphoid cells.

DISCUSSION

The symptoms and pathology seen in the affected piglets fits in with the acute form of erysipelas. The acute disease begins with a bacteraemia which may result in the development of cutaneous lesions and a septicaemia or transient cutaneous lesions followed by clinical recovery[2]. The 2 piglets that died developed a septicaemia as shown by the isolation of *E. rhusiopathiae* from the brain, spleen, liver and kidney. They died within 24 hours of developing cutaneous haemorrhages. The 4 clinically affected piglets probably developed a bacteraemia followed by a subclinical infection and recovery following antibiotic therapy. It is probable that the 7th piglet in the litter had also been subclinically infected without having developed any cutaneous lesions. The non-specific signs of listlessness, anorexia and fever were probably attributable either to a septicaemia in the 2 piglets that died or a bacteraemia in those that recovered.

Fig. 1: Hypervascularisation, congestion and leukostasis of the dermal capillaries on the lefthand side. (H) represents an area of haemorrhage. Intravascular coagulation (c) is present within one of the vessels. HE orginal magnification x10

Fig. 2: A hypodermal venule containing a fibrin thrombus. HE original magnification x10

Fig. 3: Numerous bacteria diffusely scattered in the dermal tissue. Gram original magnification x40

Fig. 4: Rod-shaped bacteria in the dermal tissue. Gram original magnification x100

Fig. 5: A haemorrhage (H) in the renal cortex. HE original magnification x10

Fig. 6: A hepatic central vein showing congestion, leukostasis, bacterial emboli and intravascular coagulation (c). Gram original magnification x40

Fig. 7: Bacteria appearing as rods (R) amongst the leukocytes in a hepatic sinusoid. Gram original magnification x100

Fig. 8: A clump of filamentous bacteria (F) within a hepatic sinusoid. Gram original magnification x100

The exact source of infection for this outbreak could not be determined. An infected or subclinically infected pig is the usual source of infection shedding numerous organisms which contaminate the environment and spread to other pigs on the farm[2]. The infection can then be picked up per os or per cutaneously through skin wounds[1 2]. The pigs affected in this report were young, namely 2 week-old piglets, or gilts. The gilts were probably partially immune and so developed only a mild clinical form of the disease. The piglets on the other hand were more susceptible to the infection resulting in fatalities amongst some of the affected piglets. The older sows in the herd remained unaffected probably due to a high level of immunity to the infection. The reason for a gilt aborting was not determined. It is possible that she developed a transient bacteraemia and fever and that the latter led to the abortion. Pregnant sows have been known to abort due to infection by *E. rhusopathiae* during pregnancy and the bacteria have been isolated from aborted or stillborn foetuses[1].

The Onderstepoort Swine Erysipelas vaccine is a bacterin containing serotypes 1 and 2. These 2 serotypes are responsible for the majority of erysipelas outbreaks in this country and as they are highly immunogenic strains, the immunity they produce is usually good (dr C Cameron, Veterinary Research Institute, Onderstepoort, Personal communication). In our experience the vaccine is indeed effective in the field, but occasionally vaccinated pigs may develop transient anorexia, listlessness and cutaneous lesions shortly after being vaccinated. Piglets from gilts that have been vaccinated before mating are generally immune to percutaneous infection by *E. rhusiopathiae* up to 6 weeks of age[5]. Wellman & Heuner[7] found that the duration of colostral passive immunity in the piglets depends on the level of antibodies which develop in the sow following on vaccination. This varies from one sow to the next and each piglet in the litter does not get the same protective titre[7]. It is probable therefore that the dam of the affected piglets in this report developed a low protective titre which waned sufficiently for the piglets to pick up the infection. That 2 piglets died whilst 4 developed only mild clinical signs indicates the variation in immunity that exists amongst piglets from the same litter. Other possibilities are that the infection was caused by a strain not present in the vaccine or that the gilt was inadvertently not vaccinated.

The exact pathogenesis of the lesions in cases of acute erysipelas is not known[2]. An acute bacterial septicaemia as occurred in these 2 piglets induces stasis of blood cells seen as congestion and leukostasis with consequent changes in the blood flow rate and hypoxia. The latter 2 factors can activate the intrinsic clotting factors leading to intravascular coagulation, seen histologically as fibrin thrombi within the vascular lumens[3 4 5]. The histopathological lesions observed were centred around the vascular system and included congestion and leukostasis, with bacterial emboli and fibrin coagulation in the capillaries of the skin, liver, spleen and kidney and fibrin thrombi in some of the hypodermal venules. According to Schulz et al. experimental infection with *E. rhusiopathiae* leads to the development of hyaline or fibrin thrombi in the terminal vascular beds of various organs especially the capillaries of the myocardium[5].

The cellular response to *E. rhusiopathiae* consists mainly of monocytes and to a lesser extent neutrophils[2]. A moderate inflammatory response was present in the dermis and hypodermis. This was characterised by the infiltration especially around the vessels of monocytes in particular and to a lesser extent neutrophils. Neutrophils are more prevalent when typical rhomboidal lesions develop. In these cases, the reaction is centred around the arterioles, viz., fibrinoid necrosis and neutrophil infiltration of the arteriolar walls. The latter reaction probably represents a hypersensitive reaction of the Arthus type and only a few dermal arterioles of the 2 piglets in this report developed the latter lesions. The arteriolar lesions are apparently the basis for the development of the rhomboidal skin lesions[1]. This explains the lack of these lesions in the piglets reported here. The arteriolar lesions probably also take longer to develop and these piglets died within 24 hours of clinical symptoms having developed.

The bluish discolouration of the limbs and ears could probably be ascribed to the intense congestion of the dermal capillaries at these sites. The colour of the skin lesions has a direct correlation with the outcome of the disease. Light pink to red lesions generally resolve and the pigs recover whilst dark blue-purple lesions as observed in this report usually precede the death of the animal.

In smears made from cultures of *E. rhusiopathiae,* the bacteria appear as slender straight or curved Gram-positive rods occurring singly or in short chains. After several subcultures, a filamentous form up to 20 μm in length is also observed[2]. Bacteria were visible within the vessels of the dermis, hypodermis, kidney, spleen and liver as well as loose in the areolar tissue of the skin. They occurred either as rods or filaments as described above. The bacteria with the filamentous form were often clumped together. It seems strange that the filamentous form should have occurred as this form generally occurs later after subculture of the bacteria. Its occurrence in the tissues of the 2 piglets discussed here may be indicative of rapid multiplication of the bacteria.

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CASE REPORT **GEVALSVERSLAG**

SUSPECTED FACIAL ECZEMA IN SHEEP IN THE CENTRAL ORANGE FREE STATE

J.A.L. DE WET* and J.A. ERASMUS**

ABSTRACT: De Wet J.A.L.; Erasmus J.A. **Suspected facial eczema in sheep in the central Orange Free State.** *Journal of the South African Veterinary Association* (1984) 55 No. 4, 199-200 (En). Veterinary Laboratory, P.O. Box 502, 9300 Bloemfontein, Republic of South Africa.

Facial eczema is known to occur in the Humansdorp district of the Cape Province. During June 1982-January 1984, 5 outbreaks of hepatogenous photosensitivity occurred in sheep in central districts of the Orange Free State. In one of these outbreaks a diagnosis of suspected facial eczema was made histologically.

Key words: Facial eczema, *Pithomyces chartarum, Panicum* photosensitivity, icterus, sheep.

INTRODUCTION

Geeldikkop is a photosensitivity disease of sheep grazing on predominantly *Tribulus terrestris* ("dubbeltjies") in the Karoo and certain parts in South Africa. The disease has been experimentally produced by the simultaneous ingestion of *T. terrestris* plants and dosing of toxic cultures of *Pithomyces chartarum*[2] as well as by the administration of extracts or feeding of whole *T. terrestris* plants collected during outbreaks of geeldikkop (G.F. Bath, Regional Veterinary Laboratory, Allerton, Pietermaritzburg, unpublished work). A disease indistinguishable from geeldikkop, locally known as dikoor or *Panicum* photosensitivity, occurs sporadically in the central and northern Orange Free State (OFS). Although the aetiology of this disease has not been completely elucidated, a similar pathogenesis to geeldikkop has been proposed[2].

P. chartarum has been demonstrated on pastures in the cool, relatively moist climate of the coastal belt, in the hot, semi-arid climate of the Karoo, the warm temperate grasslands of the Highveld and in the savannah of north-eastern South West Africa[2].

Facial eczema, which is caused by *P. chartarum,* was diagnosed for the first time in South Africa in the Humansdorp districts of the Cape Province[3] but has not been reported in the OFS.

RESULTS AND DISCUSSION

During the period June 1982-January 1984, 5 outbreaks of photosensitivity with icterus in sheep were investigated in the Brandfort, Dealsville, Dewetsdorp and Tweespruit disctricts (Table 1). On Farm 2 no formalin-fixed tissue were available for histopathological diagnosis. Dikoor and geeldikkop were diagnosed on Farms 3 and 5 respectively while microscopical liver lesions compatible with facial eczema were evident in a sheep that died on Farm 1. A liver sample taken from an effected sheep on Farm 4 was without any specific lesions.

Samples of the wheat pasture on Farm 1 were collec-

Table 1: DETAILS OF OUTBREAKS OF DIKOOR, GEELDIKKOP AND FACIAL ECZEMA IN SHEEP IN SOME CENTRAL DISTRICT OF THE ORANGE FREE STATE. JUNE 1982-JANUARY 1984.

Farm	District	Month and year of outbreak	Number of sheep in flock	Age	Number of sheep		Grazing during outbreak	Histopathological diagnosis
					affected	dead		
1	Brandfort	July 1982	125	Adult Lambs (5 months)	0 23	0 8	Green wheat without "dubbeltjies" and *Panicum* grass	Facial eczema
2	Dealsville	July 1982	400	Adult	32	12	Green wheat without "dubbeltjies" and *Panicum* grass	Not done
3	Dewetsdorp	December 1982	240	Lambs (4-5 months)	38	27	Dry wheat land with wild oats and *Panicum* grass	Dikoor
4	Tweespruit	January 1983	500	Adult	30	0	Dry land lucerne with "dubbeltjies" and *Panicum* grass	Negative
5	Dealsville	January 1984	600	Lambs	68	8	Wheat stubbles with "dubbeltjies", but without *Panicum* grass	Geeldikkop

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Table 2: METEOROLOGICAL DATA PRIOR TO AND DURING THE OUTBREAK OF FACIAL ECZEMA IN THE BRANDFORT DISTRICT*

Month	Rainfall (mm)		Day Temperature (°C)	
	Actual downpour	Long term average	Monthly average	Long term average
March	73,4	76,7	19,5	19,5
April	97,6	53,7	16,3	15,4
May	0	23,5	13,1	10,9
June	16,0	7,0	8,8	7,5
July	33,2	10,4	8,7	7,0

* This data was kindly made available by the Department of Agrometeorology, University of the Orange Free State, Bloemfontein.

ted during the outbreak of photosensitivity (Table 1), and were submitted for mycological examination. *P. chartarum* was isolated but toxicological examinations on the isolates were not performed.

Weather conditions in the vicinity of Farm 1, where the confirmed outbreak of facial eczema was diagnosed, are summarised in Table 2. Compared with the long term averages, the particular season could be described as warmer and more humid than normally expected. Such conditions might have favoured outbreaks of facial eczema[1].

Evidently dikoor, geeldikkop and possibly facial eczema may be found in certain parts of the OFS.

ACKNOWLEDGEMENTS

We wish to thank the staff of the Section of Pathology, Veterinary Research Institute, Onderstepoort for the histological examination of the liver samples and the Section of Mycology, Plant Protection Research Institute for identifying the fungi on the plant material.

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BOOK REVIEW — BOEKRESENSIE

COLOUR ATLAS OF VETERINARY ANATOMY VOLUME ONE: THE RUMINANTS

R.R. ASHDOWN and S. DONE

1st Edn. Baillière Tindal, 1 St. Anne's Road, Eastbourne, East Sussex, BN21 3 UN, England. 1983. pp. 230, illustrations 600. Price £35,00. ISBN 0 7020 0980 6

This is the first of a three volume series of colour atlasses on the anatomy of domestic animals. The 600 illustrations consist of full colour photographs of detailed dissections showing important topographical features. Coloured drawings accompany the photographs to identify the important structures as well as the clarify the relationship of the relevant structures.

Each section begins with photographs of regional surface features before dissection. Photographs of a bovine skeleton illustrate the important palpable landmarks of these regions. The dissections and photographs reveal the topography of the structures as it would be presented to the student or veterinary surgeon during clinical examination or surgery. The sequence of the photographs present the progression of the dissections as they occur from superficial to deep. The dissections are mainly of the mature bovine animal. Where necessary, calves are used to show age differences, while dissections of sheep and goats are used to illustrate species differences. The nomenclature of the muscles, arteries, veins, nerves and lymphatics is based on that of Nomina Anatomic Veterinaria (1973), while anglicized terms are used for all other structures.

This is not a complete atlas of applied Veterinary Anatomy, but considerable emphasis is given to those regions and structures that are important to the student and veterinarian in practice. In the reviewer's opinion this book will be most useful to veterinary students and practicing veterinarians who work with ruminants.

A.J. Bezuidenhout

SUID-AFRIKAANSE BEESBILTONG – WEEREENS ONDER DIE SOEKLIG

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ABSTRACT: Osterhoff D R; Leistner L. **South African beef biltong – another close look.** *Journal of the South African Veterinary Association* (1984) 55 No. 4, 201-202 (Afr). Department of Zootechnology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

Twenty beef biltong samples from various commercial sources were investigated chemically and microbiologically. The biltong contained 5-10 % NaCl, little sugar and nitrite but 10-860 ppm nitrate. The addition of nitrate apparently did not ensure stability, since spoiled biltong often contained much residual nitrate. Biltong was stable at the barrier combination of $a_w \leq 0.77$ and $pH \leq 5.5$ because such samples did not spontaneously become mouldy. A high degree of contamination with moulds and yeasts was found indicating a real health hazard to which consumers might be exposed. Better control measures must be imposed to ensure a better product to the consumer.

Key words: beef biltong, dried salted meat.

INLEIDING

Die konservering van vleisprodukte was vir baie jare toegespits op lae temperature (vries en koel) óf op hoë temperature (stoom en inmaak). Hierdie konserveringstegnieke is ook in ontwikkelende lande gepropageer, maar groot finansiële verliese is gelei deur die implementering van hierdie duur tegnieke in daardie lande[1].

Europese vleisnavorsingslaboratoria het begin om na alternatiewe moontlikhede te soek en dit blyk dat tradisionele tegnieke van vleisberging nou op groot skaal ondersoek word[1]. Daar word hoofsaaklik op 2 rigtings gekonsentreer: op die rakstabiele produkte (Eng. shelf stable products – SSP) soos byvoorbeeld die Italiaanse mortadella of die Duitse dauerwurst en op die intermediêre vogtigheidsvoedsel (Eng. intermediate moisture foods – IMF), soos byvoorbeeld die tsusougan of sousong van China of die biltong of khundi van Afrika of die beef jerky of charque van Amerika[1].

In Suid-Afrika word omtrent 100 000 kg beesbiltong per jaar geproduseer[3] en Suid-Afrikaners het die kennis van biltongmaak, wat miskien wyer na ontwikkelende lande oorgedra kan word.

MATERIAAL EN METODES

Die senior outeur het 20 monsters van kommersieël-beskikbare beesbiltong verskaf, wat in die Federale Navorsingsinstituut vir Vleiskunde te Kulmbach in Duitsland deur die 2e outeur ondersoek is.

Die metodes is die gebruiklikes wat deur Van den Heever[2] in alle besonderhede beskryf is.

Tabel 1: RESULTATE VAN ANALIESE VAN SUID-AFRIKAANSE BEESBILTONG

Nr.	Produk	Tellings per gram biltong: Totale kiemgetal	Lakto-basille	Fungi	Giste	a_w-waarde	pH-waarde	NaCl %	Water %	Nitriet ppm	Nitraat ppm
1*	Biltong-skyfies	$5{,}1 \times 10^{6}$**	$<10^{2}$	$<10^{2}$	$1{,}5 \times 10^{4}$	0,874	5,48	4,1	35,8	1	20
2*	" "	$5{,}0 \times 10^{6}$	$<10^{2}$	$3{,}3 \times 10^{5}$	$4{,}8 \times 10^{6}$	0,813	5,72	4,7	29,4	2	485
3*	" "	$1{,}1 \times 10^{8}$**	$<10^{2}$	$2{,}0 \times 10^{4}$	$3{,}0 \times 10^{7}$	0,807	5,71	6,4	31,8	0	956
4	" "	$1{,}6 \times 10^{5}$	$<10^{2}$	$<10^{2}$	$6{,}1 \times 10^{4}$	0,778	5,39	6,4	31,1	1	11
5	" "	$4{,}5 \times 10^{3}$	$<10^{2}$	$<10^{2}$	$<10^{2}$	0,775	5,55	5,7	29,4	1	21
6	" "	$2{,}0 \times 10^{5}$	$<10^{2}$	$1{,}0 \times 10^{3}$	$<10^{2}$	0,769	5,37	8,2	29,9	0	11
7	" "	$5{,}8 \times 10^{3}$	$<10^{2}$	$<10^{2}$	$<10^{2}$	0,676	5,59	7,5	24,9	26	293
8	" "	$4{,}1 \times 10^{3}$	$3{,}0 \times 10^{2}$	$<10^{2}$	$<10^{2}$	0,664	5,24	5,2	16,4	2	70
9*	Biltong-stukke (oop verpak)	$4{,}7 \times 10^{3}$**	$<10^{2}$	$3{,}0 \times 10^{2}$	$<10^{2}$	0,797	5,73	17,0	24,9	0	33
10	" "	$7{,}0 \times 10^{3}$	$5{,}0 \times 10^{2}$	$<10^{2}$	$<10^{2}$	0,726	5,55	5,4	26,7	0	304
11	" "	$2{,}6 \times 10^{6}$**	$<10^{2}$	$1{,}7 \times 10^{3}$	$3{,}4 \times 10^{4}$	0,708	5,47	7,6	24,8	2	12
12	" "	$<10^{2}$	$<10^{2}$	$<10^{2}$	$<10^{2}$	0,654	5,49	10,5	20,4	0	11
13	" "	$4{,}1 \times 10^{4}$	$6{,}0 \times 10^{4}$	$<10^{2}$	$<10^{2}$	0,625	5,83	7,2	10,6	1	361
14	" "	$1{,}7 \times 10^{3}$	$3{,}6 \times 10^{3}$	$<10^{2}$	$<10^{2}$	0,586	5,84	8,5	14,4	0	22
15	" "	$1{,}8 \times 10^{4}$	$8{,}0 \times 10^{2}$	$<10^{2}$	$<10^{2}$	0,537	5,58	7,5	13,0	2	129
16	Biltong-poeier	$4{,}2 \times 10^{5}$	$3{,}9 \times 10^{5}$	$<10^{2}$	$<10^{2}$	0,637	5,35	7,7	17,8	0	152
17	" "	$<10^{2}$	$<10^{2}$	$<10^{2}$	$<10^{2}$	0,555	5,26	6,9	10,8	2	586
18	" "	$1{,}4 \times 10^{6}$	$1{,}2 \times 10^{6}$	$5{,}0 \times 10^{2}$	$3{,}5 \times 10^{4}$	0,362	5,56	6,1	3,6	3	860
19	" "	$6{,}4 \times 10^{5}$**	10^{2}	$6{,}6 \times 10^{3}$	$<10^{2}$	0,360	5,49	9,1	6,2	2	11
20*	Biltong-stuk (vakuum verpak)	$1{,}7 \times 10^{7}$	$2{,}6 \times 10^{7}$	$<10^{2}$	$4{,}0 \times 10^{3}$	0,923	4,81	4,4	48,8	1	12

* Voor die ondersoek deur skimmel-fungi en/of giste bederf
** Hoofsaaklik *Micrococcaceae*
0 Nie bewys nie

RESULTATE EN BESPREKING

Die volledige resultate van die ondersoek te Kulmbach word in Tabel 1 gegee.

Die gemiddelde waardes deur hierdie ondersoek gelewer is vergelyk met waardes wat deur Van den Heever[2]

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Table 2: VERGELYKING VAN DRIE VERSKILLENDE ONDERSOEKE OP BEESBILTONG

	Gemiddelde water-aktiwiteit (a_w)	Gemiddelde water-gehalte (%)	Gemiddelde NaCl-gehalte (%)	Gemiddelde pH-waarde	Totale kiemgetal
Van den Heever[2] 60 monsters	0,74	25,2	6,6	5,9	7,0 x 10^6/g
Van der Riet[3] 20 monsters	0,70	22,9	5,6	5,7	4,4 x 10^5/g
Leistner[1] 20 monsters	0,68	22,5	7,3	5,5	7,1 x 10^6/g

en Van der Riet[3] gepubliseer is (Tabel 2).

Die gemiddelde waardes is redelik goed vergelykbaar maar wat werklik kommer wek is die groot variasie in die waardes in Tabel 1. Vyf monsters was onstabiel en tot 'n groot mate bederf. Wat verder problematies is, is die byvoeging van 10 to 860 dpm kaliumnitraat. Dit is duidelik dat selfs die byvoeging van groot hoeveelhede nitraat nie bederf kon teenwerk nie. Die geweldige hoë kiemgetal is ook reeds deur Van den Heever[2] beskryf en dit wek werklik kommer. In agt van die monsters was fungi en giste met 'n kiemgetal van meer as 10^2/g aanwesig en Suid-Afrikaanse biltongentoesiaste weet dat fungi en giste die grootste oorsaak van biltongbederf is. So ook in hierdie ondersoek het vyf monsters op die manier bederf net deurdat die monsters vir vier dae by kamertemperatuur laat lê is.

Interessant is dat ook die laasgenoemde vakuumverpakte monster van Tabel 1 'n geweldige hoë laktobasilgehalte en 'n besonder lae pH-waarde getoon het. Met 'n a_w-waarde van 0.923 was koelbewaring nodig en dit is natuurlik nie die werklike idee van biltong-maak nie.

Die tyd het gekom dat behoorlike aandag aan hierdie Suid-Afrikaanse lekkerny gegee word, dat biltongprosessering verbeter word van huisresepte na wetenskaplik gefundeerde fabrieksprosesse. Wetenskaplikes in verskillende Europese vleisnavorsingsinrigtings wag op behoorlik-gefundeerde biltongresepte wat dan vir die ontwikkelende lande aanbeveel kan word[1]. Suid-Afrika kan met die beskikbare kennis 'n groot wetenskaplike bydrae maak. As daar gestandaardiseerde fabriksprosesse is, kan biltong 'n goeie mark oorsee verower maar dan moet die produk tot 'n groot mate gestandaardiseer en van beste kwaliteit wees. 'n Produk wat wissel soos die monsters in Tabel 1, wat volgens boereresepte in agterplaas-"fabrieke" gemaak word, is nie net vir uitvoer maar ook vir die Suid-Afrikaanse verbruiker onaanvaarbaar.

VERWYSINGS

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BOOK REVIEW — BOEKRESENSIE

THE VETERINARY TECHNICIAN'S GUIDE TO MEDICAL TERMINOLOGY

BARBARA HANDY-MARCHELLO

1st Edn. Reston Publishing Company, Inc. (A Prentice-Hall Company), Reston, Virginia 22090, USA. 1984 pp x and 285, several illustrations. Price not indicated. Paperback. (ISBN 0-8359-8313-7)

This handy little dictionary lists, alphabetically, in clear, concise, no-nonsence style, the commonly accepted definitions for most of the technical veterinary-medical terminology in current use. In addition some technical "jargon" and abbreviations are also included.

I can commend this book to any non-veterinarian working in close contact with veterinarians or in areas closely allied to the veterinary field. Veterinary technicians, nurses and aides, in particular, would benefit from this book, as would voluntary workers in the veterinary field (e.g. SPCA) and prospective veterinary students. It may even fill a void in the vocabulary of 1st and 2nd year veterinary students.

F. Reyers

THE RETENTION OF VAGINAL PROLAPSE IN THE COW USING A PURSE-STRING SUTURE

G.H. RAUTENBACH*

ABSTRACT: Rautenbach G.H. **The retention of vaginal prolapse in the cow using a purse-string suture.** *Journal of the South African Veterinary Association* (1984) **55** No. 4, 203-204 (En). Department of Applied Veterinary Practice, Faculty of Veterinary Science, Medical University of Southern Africa, 0204 P.O. Medunsa, Republic of South Africa.

Seventeen cows with prolapsed vaginas were treated by placing a purse-string suture cranial to the external urethral opening to retain the vagina. It was found to be a simple procedure with a high rate of success. Fifteen of the patients responded well to the procedure with a minimum of straining being evident in the post operative period.

INTRODUCTION

Prolapse of the vagina is a fairly common condition in the pluriparous cows, especially seen in pre- but also sometimes in post-partum patients. Predisposing factors include perivaginal fat deposition, injury and inflammation of the vagina and the cervix, oestrogenic substances in the diet, poor conformation and a reported hereditary basis[2]. Incompetence of the constrictor vestibuli and constrictor vulvae muscles play a major role in the aetiology of the condition[2].

The condition occurs with varying degrees of severity, from an intermittent prolapse with very little secondary damage of the mucous membranes to a stage where thrombosis, ulceration and necrosis of the vaginal wall is present with ultimate toxaemia and death[5].

A myriad of techniques for replacement and retention of the vagina are described in the literature[1 2 3 5]. It is also true that not all are as successful as the attending clinician would wish them to be. A major complication with most of the methods is continual straining by the patient after replacement and retention with possible recurrence of the problem[5].

This study was done to evaluate an old technique that is well known to the older veterinary generation but has fallen into disuse.

MATERIALS AND METHODS

Seventeen cases of prolapse of the vagina in different stages of severity seen during practice rounds were treated by the method described below. Follow-up visits were done 3-10 days afterwards during which clinical and vaginal examinations were done on the patient. The owners were asked to relay information on any complications back to the practice. Approximately 30 additional cases were treated in the same way without follow-up procedures.

In every case the prolapsed vagina was carefully cleaned with an antiseptic solution before replacement. Epidural anaesthesia was administered in a minority of cases where straining of the patient made reduction difficult. The vagina and cervix were carefully reduced and replaced. After replacement, a purse-string suture was placed about 20 mm cranial to the external urethral opening. By avoiding the median line of the ventral floor of the vagina there is little danger of compromizing the urethra. The suture material used was umbilical tape with a reverse cutting half-circle 57 mm suture needle.

After placement the suture is tightened until an opening of about 20 mm diameter is left in the vagina, and then tied.

The owners were shown how to cut the sutures with an ordinary pair of scissors by following the tape left hanging out of the vulva into the vagina. This was to be done as soos as the patient showed symptoms of the first stage of parturition. Where post partum cases were treated, sutures were left in for approximately 10 days. Future breeding was discouraged.

RESULTS

Of the 17 cases where follow-up procedures were done, a prolapse recurred in only one case in the first 10 days. In this case the patient was already in labour and the procedure was repeated after parturition. In one case the vaginal wall became so necrotic that euthanazia was recommended.

The patients showed a minimum of straining after the procedure, decidedly less than in cases where the vagina was retained by the Halsted or modified quill technique[2].

In some cases the sutures were not cut before parturition. Although precise data is lacking this did not seem to cause problems during parturition.

DISCUSSION

Placing a purse-string suture in the vaginal wall cranial to the urethral opening is a simple and effective way of retaining the vagina and cervix after a prolapse. The patient strains less than with external fixation and it is felt that this aids in the regression of the oedema and inflammation of the vaginal and cervical tissue which is usually the trigger for persistant straining.

In a previous publication[4] it was advocated that the suture should be placed while the vagina was still prolapsed. This was found to be difficult since involvement of the bladder in the ventral sector of the prolapsed vagina causes an asymmetry that makes orientation in

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placing the sutures difficult.

As the vulva and vagina were greatly stretched, it was found that in most cases both hands of the clinician could be introduced into the vagina to place the sutures.

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CONCOMITANT FELINE INFECTIOUS PERITONITIS AND TOXOPLASMOSIS IN A CHEETAH (*ACINONYX JUBATUS*)

I.B.J. VAN RENSBURG* and M.A. SILKSTONE**

ABSTRACT: Van Rensburg I.B.J.; Silkstone M.A. **Concomitant feline infectious peritonitis and toxoplasmosis in a cheetah (*Acinonyx jubatus*).** *Journal of the South African Veterinary Association* (1984) **55** No. 4, 205-207 (En). Department of Pathology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

Three wild caught littermate cheetahs succumbed to feline infectious peritonitis (FIP) after being in captivity for approximately 9 weeks. A necropsy and histopathological examination on one revealed typical signs of FIP as well as histopathological lesions in the liver and brain of concomitant toxoplasmosis. Hypochromic anaemia, neutrophilia, lymphopaenia, eosinopaenia and elevations of α2-globulin and γ-globulin fractions of the blood were present in the one animal examined. These findings together with some clinical signs are reported.

Key words: Cheetah, feline infectious peritonitis, toxoplasmosis.

INTRODUCTION

Since the original description of feline infectious peritonitis (FIP) in 1966[10] this widespread disease of felidae, caused by a coronavirus, has been well documented in the domestic cat[2,3,5,6,7,9]. According to Barlough, Adsit & Scott[1] the disease has also been reported in wild felidae kept in zoos, namely: the lion, leopard, black leopard, jaguar, caracal and lynx. The disease was also diagnosed by Colly in serval, bobcat, puma and cheetah and suspected in a tiger during 1969 – 1979 when an outbreak of this was experienced amongst large carnivores in the Johannesburg Zoological Gardens (L P Colly 1984, Johannesburg Zoological Gardens, Johannesburg, personal communication). In 1979 Horzinek & Osterhaus found antibody titres against FIP in sera collected in South Africa from wild-caught cheetah but it is not clear whether these cheetahs had been in captivity for any length of time before collection of these sera[4]. Recently Pfeifer et al.[8] described a case of FIP in a captive cheetah. This report deals with the concomitant occurrence of FIP and toxoplasmosis in a captured cheetah as well as some findings in 2 littermates.

HISTORY AND CLINICAL SIGNS

A litter of 3 approximately 6 month old cheetahs were captured north-east of Windhoek, South West Africa/Namibia and sold to a game capture and export company. The latter company introduced these into one of their enclosures after they had been vaccinated against feline panleukopaenia and dewormed with mebendazole (Telmin, Janssen). The enclosure measured about 10 x 15 metres, had a sand floor and at the time housed another 7 cheetahs. The animals were fed on calf foetusses obtained from the local abattoir.

Approximately 9 weeks after introduction into the enclosures all 3 littermates became ill. The owner described the clinical signs as lethargy, variable loss of appetite and considerable progressive loss in body mass. Abdominal distension was a prominent sign while the stool contained undigested meat. One of the litter died, but veterinary advice was not sought until a second cheetah from the litter died. This was taken to the Central Veterinary Laboratory in Windhoek where a post mortem examination was done.

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HAEMATOLOGICAL FINDINGS

Haematological examination of the littermate which survived the longest revealed a moderate hypochromic anaemia and a marked neutrophilia, lymphopaenia and eosinopaenia. The total serum protein was normal but the α2-globulin and γ-globulin fractions were elevated.

This animal was treated with 1000 mg oxytetracycline administered per os 3 times daily. Therapy was unsuccessful and the patient died several days after treatment was initiated. A gross post mortem examination was carried out.

GROSS PATHOLOGY

Necropsy on the second animal to die revealed emaciation, general palor of the carcase and a marked ascites of approximately 2 ℓ of protein-rich, orange-yellow, viscous fluid which clotted rapidly upon exposure to air. There was a severe, diffuse fibrinous peritonitis characterised by a ground glass opacity of the serosa which showed the presence of diffusely scattered, pin-head sized white granulomatous lesions and was covered with thick yellow fibrinous coagulae in many areas. Extensive fibrinous adhesions were present between the stomach and liver, and the liver and diaphragm. Hepatomegaly was present and the hepatic parenchyma showed an orange discolouration. There was marked atrophy of the spleen and peripheral lymph nodes. The stomach contained a brown "coffee granule-like" substance indicating the presence of occult blood, but the mucosa of the stomach showed no gross change. The costal parietal pleura adjacent to the diaphragm exhibited early fibrinous pleuritis while the lungs were markedly oedematous.

Specimens of intestine, liver, mesentery, a mesenteric lymph node, spleen, kidney, brain and spinal cord were fixed in 10 % formalin for histopathological examination.

Autopsy of the last member of the litter grossly revealed lesions typical for FIP, but no specimens were collected for histopathological confirmation thereof (U Schreiber-Sigwart 1983, Central Veterinary Laboratory, Windhoek, personal communication).

HISTOPATHOLOGY

There was a severe diffuse peritonitis characterised by swelling of the mesothelial cells with underlying

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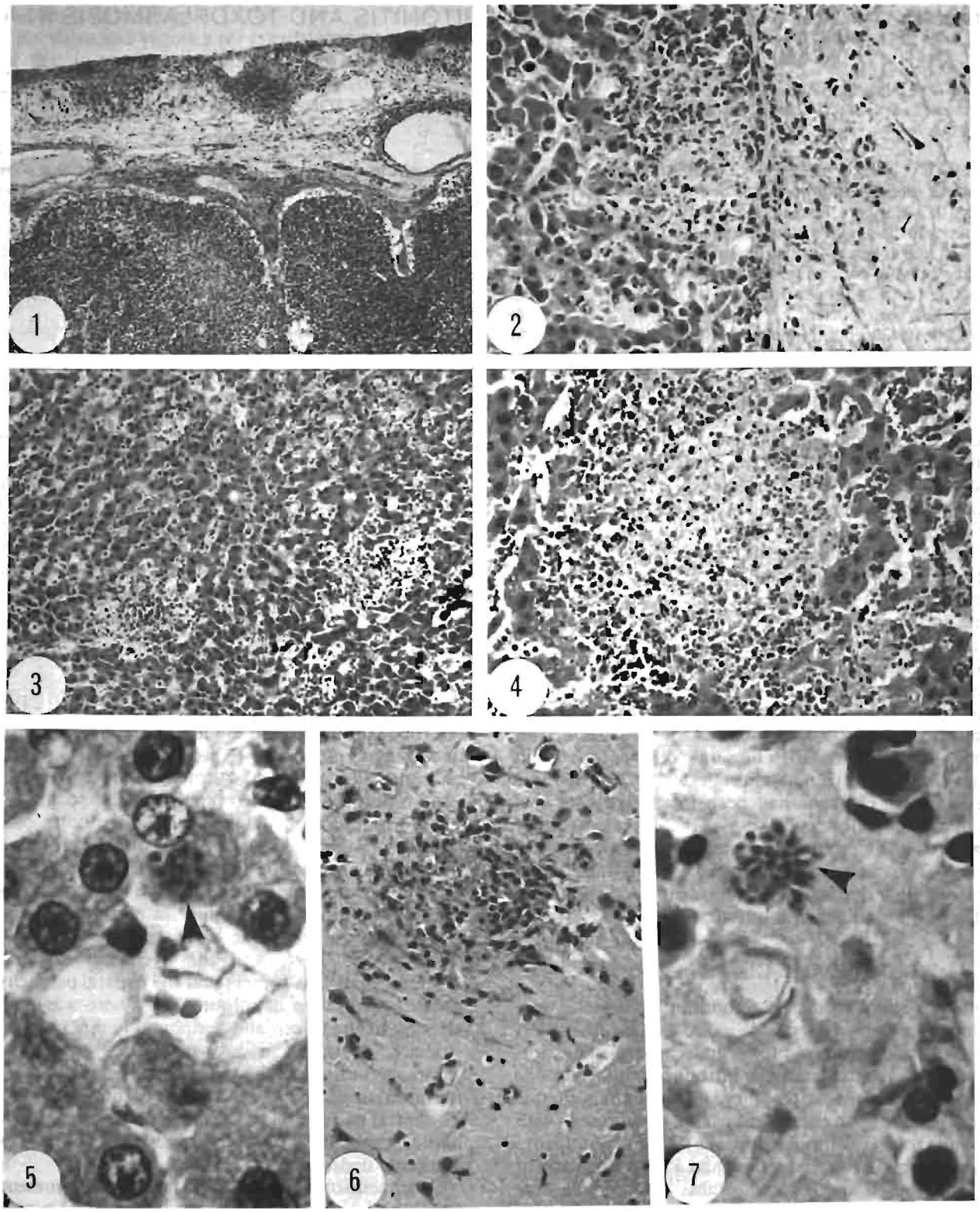

Fig. 1: Peritonitis characterised by fibrin deposition, cellular infiltration and vasculitis surrounding a mesenteric lymph node.

Fig. 2: Perihepatitis and subcapsular hepatic necrosis.

Fig. 3: Focal disseminate hepatic necrosis.

Fig. 4: Higher magnification of hepatic necrosis showing round cell infiltration.

Fig. 5: Toxoplasma parasites in a hepatocyte (arrow).

Fig. 6: Micrograniuloma in the brain.

Fig. 7: Toxoplasma parasites in the above lesion (arrow).

necrosis, fibrin deposition and infiltration of numerous plasma cells and macrophages which were densely aggregated in focal areas, often near blood vessels, to form fibrinocellular plaques. In some instances most of the infiltrated cells exhibited necrosis. Many blood vessels, especially those of the mesentery revealed a prominent vasculitis. Below the hepatic capsule was an almost continuous layer of plasma cells and, in addition, numerous foci of coagulative necrosis were disseminated through the liver parenchyma. In a few of these foci *Toxoplasma gondii* pseudocysts were present in some of the viable neighbouring hepatocytes. The spleen revealed fairly prominent lymphoid follicles and small accumulations of plasma cells in the sinusoids but the lymphoid follicles in the mesenteric lymph node were atrophic while the sinuses and medullary cords were packed with macrophages and plasma cells. The renal capsule manifested similar signs of peritonitis and the glomeruli were so swollen that they completely filled Bowman's spaces in most instances. The glomerular basement membranes were thickened and with haematoxylin and eosin staining were a pale eosinophilic colour.

Sections of the brain revealed a few microgranulomata characterised by glial proliferation and infiltration of small numbers of plasma cells. In these foci *T. gondii* bradyzoites and tachyzoites were present. No signs of meningitis were noticed. The spinal cord was not affected.

DISCUSSION

FIP was first suspected in cheetahs by Colly when he saw cases of this in 1979 in the Johannesburg Zoological Gardens (L P Colly 1984, Johannesburg Zoological Gardens, Johannesburg, personal communication). In the same year Horzinek & Osterhaus[4] found antibodies against the corona virus causing FIP in sera collected in South Africa from wild-caught cheetahs. However, clinical reports of FIP in cheetah remain scarce, the only one we could trace being that of Pfeifer et al[8].

The lesions found both macro- and microscopically in the case necropsied by the authors were typical for FIP[2,3,5,6,7,9,10]. The granulomatous nature of the peritonitis and the accompanying vasculitis observed are sufficient to warrent a diagnosis of FIP even though viral isolations and fluorescent antibody studies were not done to support the diagnosis.

Of interest is the focal disseminated hepatic necrosis which occurred extensively throughout the hepatic parenchyma and not mostly subcapsularly as is more usual for FIP. It is difficult to ascertain the role played by toxoplasma in causing these lesions as relatively few of these parasites were found in association with these lesions. Cerebral lesions caused by toxoplasma were also found but no clinical signs of nervous involvement were reported.

We are of the opinion that the cheetahs' diet of calf foetusses was qualitatively inadequate and may have led to a hypoproteinaemic immunodeficiency which predisposed to the infections of both corona virus and toxoplasma. The absence of well developed lymphoid follicles in the lymph node examined as well as the simultaneous infection with toxoplasma is possibly further support that an immunodeficient state may have prevailed. In domestic cats it is well known that the immunosuppressive effect of feline leukaemia virus infection predisposes to FIP.

There is a certain controversy in the pathogenesis of FIP, where on the one hand immunodefeciency predispose to the infection, and on the other hand, antibodies are essential in the pathogenesis of the disease which is based on a Type III hypersensitivity reaction. The finding of numerous plasma cells in the lymph nodes as well as a hypergammaglobulinaemia in a littermate indicates that enough antibodies could be produced. Therefore exactly how immunodefeciency and a high level of antibodies are interrelated is not clearly understood at present.

ACKNOWLEDGEMENTS

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BEHANDELING VAN KWAADAARDIGE EPULIS IN DIE HOND

J.S.J. ODENDAAL* en J.D.E. CRONJE**

ABSTRACT: Odendaal J.S.J.; Cronje J.D.E. **Treatment of malignant epulis in the dog.** *Journal of the South African Veterinary Association* (1984) 55 No. 4, 209-210 (Afr). 152 Benade Drive, Fichardt Park, 9322 Bloemfontein, Republic of South Africa.

The successful radiotherapy of malignant epulis in a 12 year-old Dachshund is discussed. Regrowth of the tumour, which was first removed by surgery, was treated with radiotherapy over a period of 36 days in 3 fractions. The total dose was 1800 rads. The lesion healed uneventfully. After 600 days, there was still no new growth but only scar tissue to be seen.

Key words: malignant epulis, radiotherapy, dog.

INLEIDING

Periodontale fibromateuse epulis is gewoonlik 'n goedaardige groeisel van gemengde seloorsprong van die periodontale reste van die plavei-epiteelselle. Sommige patoloë meen dat epulides slegs hiperplasties van aard is. Hierdie reste bestaan uit die neerslag van die emalje struktuur of tandlamina. Epulides is 'n redelik algemene groeisel in honde van alle ouderdomme, maar is dalk meer algemeen in ouer honde. Alhoewel daar geen geslagsvoorkeure in die voorkoms van die epulis is nie, toon Boxer-honde moontlik 'n hoër voorkoms as ander rasse[1].

Epulides kom gewoonlik in die mondslymvlies voor, veral in die premolare en molare streek en meesal om die karnasiaaltand. Hulle kan enkel of veelvoudig voorkom en is gewoonlik klein (10-20 mm in grootte). Dit het meesal 'n steeltjie en kan 'n gladde of knopperige voorkoms hê. Dit is pienk van kleur en voel meesal fibreus.

Alhoewel die oorsaak van gewasse van die mondholte nog grootliks onbekend is[2], word vermoed dat 'n epulis moontlik die beginstadium van die ontwikkeling van 'n karsinoom of sarkoom kan wees.

Die kwaadaardige vorm van epulis kom baie selde in honde voor.

GESKIEDENIS, FISIESE ONDERSOEK, DIAGNOSE EN BEHANDELING

'n Twaalfjarige gesteriliseerde Dachshundteef is aangebied vir diagnose en behandeling met 'n geskiedenis van progressiewe halitose en periodieke effense bloeding vanuit die bek. Volgens die eienaar het die pasiënt nog 'n baie aktiewe lewe gelei en was andersins baie gesond.

Die belangrikste bevinding tydens die kliniese ondersoek was knopperige groeisels aan die rostrale kant van die harde verhemelte. Die groeisel se oppervlakte was effens bloederig, die asem het baie sleg geruik en daar was tekens van sekondêre bakteriese infeksie.

Die letsels is onder algemene narkose chirurgies verwyder en monsters vir histopatologie is in 10 % gebufferde formalien versamel.

Die knopperige letsel in die mond was 10 x 20 mm groot, het 'n pienk voorkoms aan die buite rande gehad, maar was donkerrooi in die middel. Die pienk en rooi dele was goed onderskeibaar. Die letsel het vanaf die pre- en molare tande gestrek tot by die middellyn van die harde verhemelte. Die weefsel was fibreus tot hard en was oor die hele oppervlakte van die groeisel vas aan die slymvlies. As gevolg van infiltrasie van die groeisel in die slymvlies, was volledige verwydering onmoontlik.

'n Voorlopige diagnose van 'n epulis is gemaak.

'n Finale diagnose van kwaadaardige epulis is gemaak op grond van die histopatologiese ondersoek: die weefsel was neoplasties. Daar was osteoïde differensiasie en epiteelreste teenwoordig met min mitotiese figure maar baie rondesel-inflammasie en granulasie weefsel. Die prognose is as versigtig gestel weens die moontlikheid van infiltrasie en verwagte hergroei.

Twee-en-twintig dae na chirurgiese verwydering is 'n opvolgondersoek gedoen en hergroei het begin plaasvind. 'n Verdere 21 dae later was die groei van die letsel sodanig dat dit 'n 2de keer chirurgies verwyder is.

Aangesien hergroei so aktief plaasvind het, is besluit om radioterapie toe te pas. Radioterapie het begin 23 dae na die 2de chirurgiese ingreep. Dit is onder algemene narkose gedoen, het oor 'n tydperk van 36 dae gestrek en is in 3 fraksies toegedien. Die totale dosis was 1800 rads. Die bestraling is van binne die mondholte gedoen, direk op die letsel. Daar is gepoog om die oë sover moontlik uit die bestralingslyn te hou.

'n Opvolgondersoek 370 dae na die eerste behandeling het volledige genesing getoon (Fig. 1).

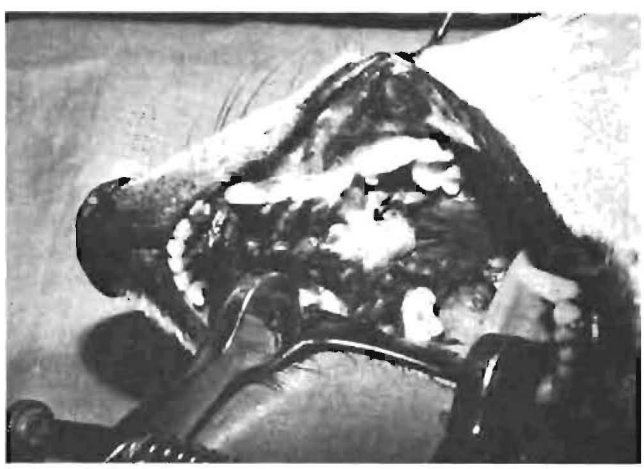

FIG. 1: Die letsel op die harde verhemelte toon litteken waar herstel plaasgevind het.

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RESULTATE EN BESPREKING

Alhoewel epulides redelik algemeen in honde voorkom moet die kwaardaardige vorm as seldsaam beskou word. Die volgende differensiële diagnose word kortliks bespreek.

Volgens Gorlin, aangehaal deur Theilen & Madewell[2], kom adamantinomas baie seldsaam by honde voor. Hy meen dat die meeste gevalle in die literatuur geklassifiseer behoort te word as periodontale fibromateuse epulides. Hierdie gewasse kom veral in volwasse honde voor en daar is geen geslagsvoorkeure nie. Adamantinomas is 'n kwaadaardige oorgroei van die epiteelreste en is van dieselfde oorsprong as periodontale fibromateuse epulis. Adamantinomas kan die aangrensende sagte en beenweefsel infiltreer, maar saai nie uit nie. Dit is ook geneig om weer na chirurgiese ingrepe te groei en lyk na 'n karsinoom met 'n lae graad van kwaadaardigheid[1].

Plaveiselkarnisoom van die epiteelselle kan ook 'n differensiële diagnose wees.

'n Volgende toestand wat ook soortgelyk aan kwaadaardige epulis is, is vratagtige karsinoom van die mondholte, wat ook selde in honde voorkom, en wat ook geneig is om weer te groei ná chirurgiese verwydering. Radioterapie is blykbaar in hierdie geval 'n kontra-indikasie omdat die gewas in mense geneig is om na bestraling te verander na 'n anaplastiese karsinoom. Die onderskeid tussen hierdie gewas en 'n epulis se pseudoepiteliomatiese hiperplasie, is dat vratagtige karsinoom van die mondholte se selle meer displasties is en die mitotiese indekse hoër. Vratagtige karsinoom is voorts ook meer infiltrerend as 'n epulis[3].

Theilen & Madewell[2] meen dat oro-faringeale karsinome redelik bestralingsgevoelig is. In mense is karsinome beperk tot die slymvlies van die mond egter hoogs geneesbaar met bestraling[2]. Die geval onder bespreking het baie goed gereageer op behandeling en leef na 600 dae nog 'n normale en gesonde lewe. Die radioterapie het waarskynlik die grootste bydrae gelewer tot die goeie resultate wat behaal is.

BEDANKINGS

Ons bedank die Superintendent van die Nasionale Hospitaal Bloemfontein vir toestemming om te publiseer en Dr Stella S. Bastianello van die Departement Patologie, Fakulteit Veeartsenykunde, Universiteit van Pretoria, vir die histopatologiese ondersoek.

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BOOK REVIEW — BOEKRESENSIE

NUTRITION AND BEHAVIOUR OF DOGS AND CATS

R.S. ANDERSON (Ed.)

Pergamon Press Ltd. 1984 pp IX and 241, Figures 23, Tables 59 (ISBN 0-08-029778-1) Price US dollars 30

This book is a report on the proceedings of the First Nordic Symposium on Small Animal Veterinary Medicine held in Oslo on 15-18 September 1982. As its title indicates, it covers two separate aspects of the dog and cat viz. nutrition and behaviour, which are presented in two parts.

Part 1 – Nutrition, is divided into four sections: Firstly an overview of dog and cat nutrition is given, in which objectives for general nutrition are stated. Secondly, feeding for reproduction and growth is covered. Feeding of the bitch during pregnancy and lactation and feeding of pups and kittens during the suckling period and after weaning are covered. Thirdly an insight into foods and their digestibility is given. A very welcome aspect here is various recipes that are presented for many conditions, e.g. cardiac conditions, uraemics, diabetes mellitus, etc., as well as for normal dogs. Fourthly, a section on nutrition and disease deals with various problems which may occur, ranging from the "overnutrition syndrome" in rapidly growing dogs, through problems in racing sled dogs, food allergies, dermatoses, and heart and kidney disease. Each part in this section should give the small animal practitioner valuable information and help him to learn how to treat and prevent many of our present day nutrition-related disease conditions.

Part 2 – Behaviour is also presented in four sections. Firstly an introduction deals with the methods used to describe normal and abnormal behaviour of dogs and cats. A whole new world opens itself to the uninitiated in this field. Secondly the neurophysiology of behaviour and human-animal relationships with emphasis on the Scandinavian pet environment is presented. The third section deals with behavioural development of the puppy in the home environment, social behaviour in free-ranging domestic and feral cats, inheritance of behaviour in the dog and also behaviour of the mature dog. Many new insights may be gained from this section. The last section explains how to communicate with the dog when training, it deals with behavioural problems in dogs and also in cats and terminates with an outlay of possible pharmacological approaches to control behavioural problems.

In general, this book "reads" easily, while at the same time it is filled with information that is relatively new to the general veterinary field.

This book should be of great value to practitioner, student and researcher in these fields.

P.D. Botha

GEVALVERSLAG CASE REPORT

CHIRURGIESE HERSTEL VAN OOP *DUCTUS ARTERIOSUS* IN 'N HOND

J.S.J. ODENDAAL* en D.B.R. WANDRAG*

ABSTRACT: Odendaal J.S.J.; Wandrag D.B.R. **Surgical repair of patent *ductus arteriosus* in a dog.** *Journal of the South African Veterinary Association* (1984) **55** No. 4, 211-213 (Afr.) 152 Benade Drive, Fichardt Park, 9322 Bloemfontein, Republic of South Africa.

The diagnosis, surgery and results of a case of patent *ductus arteriosus* in a dog are presented. The case was treated successfully in a private practice.

Key words: surgery, patent *ductus arteriosus*, dog.

INLEIDING

Oop *ductus arteriosus* is 'n baie bekende toestand in die hond, maar word nie so dikwels gediagnoseer nie.

Volgens Erickson kom hierdie toestand veral voor in die volgende honderasse: Dwerg poodles, Kollies, Pomeranians, Shetland Skaaphonde en Cocker Spaniëls. Die outeur meen dat daar in die Poodles 'n neiging tot poligenetiese drumpelwaarde oordraging van die afwyking is[2]. Buchanan & Lawson sê in een populasie ondersoek is selfs gevind dat een uit 'n duisend honde oop *ductus arteriosus* kan hê. Hulle stem saam dat die toestand meer algemeen voorkom in Poodles, Kollies en Pomeranians[1]. Tewe toon 'n hoër voorkoms as reunhonde[3].

Ductus arteriosus is 'n normale fetale arterie wat die dalende aorta en die *truncus pulmonalis* verbind, net bo die hartbasis waar die 2 groot slagare die hart verlaat. Normaalweg begin hierdie *ductus arteriosus* kort na geboorte sluit. Na 'n paar weke van degenerasie en fibrose word dit die *ligamentum arteriosus* genoem.

As gevolg van die biologiese onvermoë om sluiting van die *ductus arteriosus* te bewerkstellig, ontstaan die toestand van oop *ductus arteriosus* in die jong hond. Weens die hoër druk in die aorta vloei die bloed gewoonlik van die aorta na die *arteria pulmonalis*. Suurstofryke bloed word in hierdie proses met suurstofarme bloed gemeng.

Hierdie abnormale bloedvloei word 'n verskuiwing van links na regs genoem omdat bloed vanuit die linker ventrikel na die bloed vanuit die regter ventrikel vloei. Die gevolg van so 'n stap is voor die hand liggend. Omdat méér bloed na die *arteria pulmonalis* vloei word te veel bloed aan die longe voorsien wat longedeem tot gevolg kan hê. So ontstaan kongestiewe hartversaking wat weer aanleiding gee tot swakker suurstofvoorsiening aan die liggaam. Hierdie toestand veroorsaak in die jong hond swak groei, asook vinnige uitputting tydens oefening.

GESKIEDENIS

'n Ses-maande oue Duitse Herdershond-teef is ingebring met die klagte dat die hondje nie na behore groei nie. Die hond het ook gou moeg geword met oefening en was lusteloos. Verder kon die eienaar 'n "trilling" oor die hart-area voel.

KLINIESE ONDERSOEK

Die klagtes van die eienaar is tydens die kliniese ondersoek bevestig. Die geruis oor die hart-area was so uitgesproke dat dit met die hand gevoel kon word en omdat die hondjie so skraal was, kon mens die hart van buite teen die borswand sien bons. Met ouskultasie kon vasgestel word dat die geruis die sterkste tussen die derde en vierde ribspasie, naby die basis van die hart was.

Die hond was nie in 'n goeie kondisie nie en die swak groei van die hond kon duidelik waargeneem word, as sy met die res van die werpsel vergelyk is.

Die hond het met gedwonge oefening gou moeg geword en het lusteloos vertoon. Die asemhaling was kort en vinnig. Die slymvliese was ietwat sianoties en die pols ongeveer 80.

Die eetlus was redelik goed en geen koors was teenwoordig nie. Die hond was reeds geënt teen die algemene siektes van jong honde en was ook ontwurm.

DIAGNOSE

Daar is reeds op die geskiedenis en kliniese ondersoek 'n oop *ductus arteriosus* gediagnoseer. Om die diagnose te ondersteun is röntgenfotos van die borskas geneem van 'n laterale posisie. Die hond het op die regter sy gelê.

Kontrasmedium is toegedien met 'n naald en kateter wat deur die borswand in die hart geplaas is. Omdat die kontrasmedium so vinnig uit die hart beweeg is dit direk voor die foto geneem is in die hart gespuit (Fig. 1)

Die hart was groot en tekens van ligte longedeem was teenwoordig.

Volgens Buchanan & Lawson kan 'n aanduiding van oop *ductus arteriosus* van so 'n foto verkry word as die *arteria pulmonalis* en die *vena pulmonalis* van die regter apikale long ewe groot in deursnit vertoon, en hulle deursnit groter is as die vierde rib se dunste deel[1]. Die begin van die dalende aorta en die *arteria pulmonalis* is ook vergroot. Die oop *ductus arteriosus* tussen die 2 slagare kan nie gesien word nie, weens die aard en posisie daarvan.

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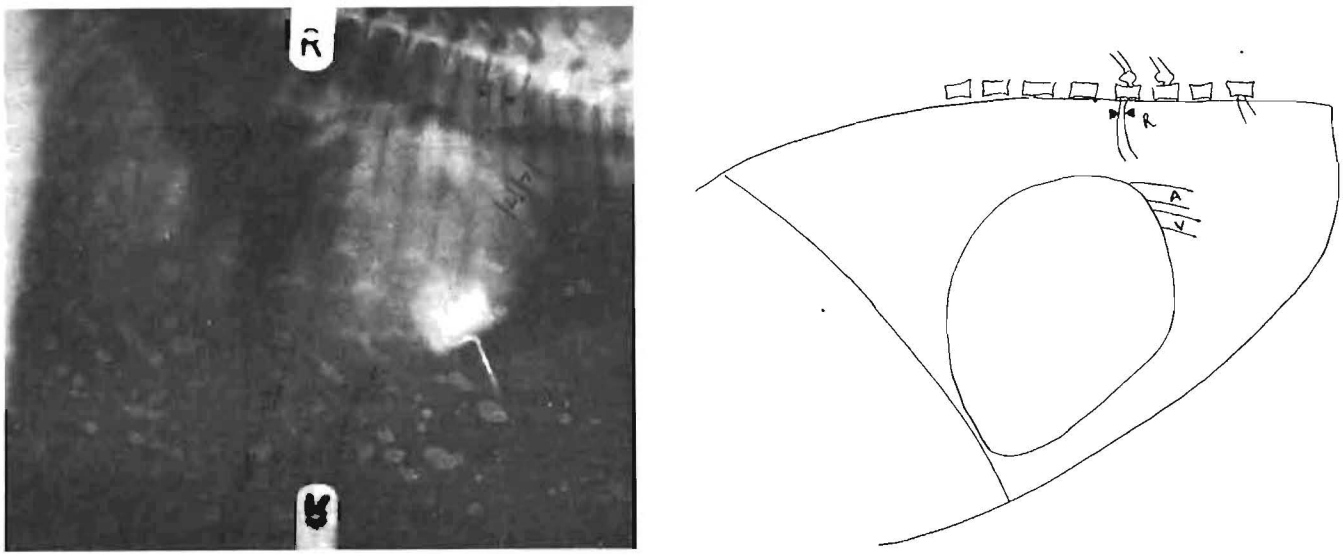

Fig. 1: Foto en verklarende skets. A: *ateria pulmonalis* en V: *vena pulmonalis* van regter apikale long is ewe groot. R: Dunste deel van die vierde rib; dunner as A en V.

BEHANDELING

Die volgende chirurgiese prosedure is gevolg om die toestand reg te stel.

Nadat die hond onder algemene gasnarkose geplaas is, is sy op haar regter sy geplaas en die linker borswand is voorberei vir 'n steriele operasie.

Nadat 'n snit van ongeveer 180 mm in die vel gemaak is, is 'n torakotomie uitgevoer en die vierde rib verwyder ten einde meer spasie te verkry vir die operasie. 'n Selfhou-wondstrekker is in posisie geplaas om die operasie veld bloot te lê. Die assistent chirurg het die linker long so in posisie gehou dat die basis van die hart en die slagare wat die hart verlaat, sigbaar is. Die hond is kunsmatig met die hand geventileer tydens die operasie.

Die mediastinum is oopgesny net bo die *nervus vagus*, parallel met die aorta. Die mediastinum is ventraal getrek om die area van die oop *ductus arteriosus* bloot te lê. Die aorta en *arteria pulmonalis* het beide vergroot vertoon. Daar was 'n baie nou verbinding tussen die 2 slagare. Deur palpasie met die vinger, kon die presiese posisie van die ductus vas gestel word. Daar is toe van stompdisseksie gebruik gemaak om die *ductus arteriosus* te isoleer. 'n Gekromde aartangetjie is onder die *ductus arteriosus* geplaas om die hegmateriaal in posisie te plaas vir die afbind van die *ductus arteriosus*. 'n Tweede lengte hegmateriaal is op dieselfde wyse in posisie geplaas.

No. 1/0 monofilament nylon is gebruik om die afbinding te doen. Die eerste knoop is met 'n dubbele slag om die naaldvoerder begin en daarna is daar nog 5 knope gemaak om die eerste afbinding te voltooi. Die vrypunte van die hegmateriaal is kort teen die finale knoop afgesny. Die tweede ligatuur is op dieselfde wyse langs die eerste een aangebring. Die eerste afbinding was aan die aorta se kant en die tweede aan die *truncus pulmonalis* se kant.

Weens die gevaar van skok wat kan intree as gevolg van die skielike veranderde bloedvloei wat die afbinding van die *ductus arteriosus* meebring, is die afbindproses geleidelik toegepas. Die totale afsluiting van die *ductus arteriosus* het ongeveer tussen 5 en 10 minute geneem. Skok behandeling is in elk geval tydens die operasie toegedien. Die mediastinum is met No. 3/0 chroom dermsnaar geheg. Die borswand is weer met monofilament nylon geheg. Die gaping wat ontstaan het as gevolg van die verwydering van die vierde rib is geheg deur eers drie spanningsteke in posisie te plaas, voordat die res van die weefsels met enkel onderbroke steke geheg is.

Net voordat die borswand finaal geheg is, is daar negatiewe druk in die borsholte verkry deur die longe só met suurstof te vul, dat dit die res van die borsholte beslaan. Die vel is met enkel onderbroke steke geheg.

Na die operasie is geen verdere behandeling toegedien nie en die velsteke is na 14 dae verwyder.

RESULTATE

Die hond het die operasie sonder komplikasies oorleef en is na 2 dae observasie, huis toe gestuur. Die eerste opvolg kontrole was toe die steke verwyder is.

Die hond het vinnig van die operasie herstel, sonder enige terugslae. Die kliniese beeld het geleidelik verbeter. Die erge geruis het verdwyn en die asemhaling, pols en kleur van die slymvliese was normaal. Die eetlus was baie goed.

Met die verloop van tyd het die hond weer gewig aangesit en gegroei. Die hond het meer lewenslus getoon en die aktiwiteit het toegeneem.

Die enigste klagte was dat die linker voorbeen, aan die kant waar die rib verwyder is, aanvanklik ongemaklik en styf voorgekom het met beweging. Na ongeveer 3 weke het die probleem egter feitlik verdwyn. Daarna het die hond baie aktief geword en was die bewegings weer sonder probleme. Die hond leef op die oomblik 'n normale lewe, 8 maande na die operasie.

BESPREKING

In 'n privaat praktyk word patente *ductus arteriosus* nie dikwels gediagnoseer en suksesvol behandel nie. Weens die positiewe resultate wat behaal is, kan 'n mens tevrede wees met die behandeling. Dit is wel moontlik om die operasie te doen sonder om die vierde rib te verwyder. So 'n stap sal bydra om die ongemak wat die hond met beweging gehad het na die operasie, uit te skakel.

Daar sal gepoog word om hierdie geval op te volg tot-

dat 'n nadoodse ondersoek uitgevoer kan word, ten einde die finale effek van nylon in die borsholte vas te stel.

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ABSTRACTS

ABSTRACT: Button, C., Reyers, F., Meltzer, D.G.A., Mülders, Maria S.G. & Killeen Valerie M., 1983. **Some physiopathological features of experimental *Homeria glauca* (Wood & Evans) N.E. Br. poisoning in Merino sheep.** *Onderstepoort Journal of Veterinary Research,* **50,** 191-196 (1983).

Five Merino sheep were dosed 3g/kg of dry, finely-milled *Homeria glauca* (Natal yellow tulp) plant material. An electrocardiogram was recorded and the arterial and central venous blood pressure, blood gases, haematological variables, plasma electrolytes (Na^+, K^+, Ca^{2+}, Mg^{2+}, Cl^- and PO_4^{2-}) and a variety of serum enzymes and chemical constituents were measured hourly until death (3 sheep) or until sheep were *in extremis* (2 sheep).

Heart rate rose progressively as a result of sinus and, later, ventricular tachycardia. Systolic blood pressure rose, but there was little change in the mean and diastolic arterial pressures and central venous pressure. There was progressive hypoxaemia, hypercarbia and acidaemia with depletion of plasma bicarbonate. Haemoconcentration, hyperkalaemia and hypochloraemia were found along with rising serum creatinin and plasma glucose. Rises in serum enzymes indicated widespread tissue damage. Electrocardiographic recordings were being made at the moment of death in 3 of the 5 sheep. In these 3 sheep the cause of death was ventricular fibrillation.

ABSTRACT: Spickett, A.M. & Nari Henrioud, A.J., 1983. The efficacy of the Drummond adult test on ***Boophilus microplus* females (Acarina: Ixodidae) subject to various periods of cold storage prior to organophosphate testing.** *Onderstepoort Journal of Veterinary Research,* **50,** 197-198 (1983).

Engorged females of *Boophilus microplus,* stored at 4 °C for up to 5 days, and females, kept at room temperature for 1 day en then at 4 °C for 1 day, showed no significant differences in their response to an organophospate as determined by the Drummond adult test.

ABSTRACT: Hoogstraal, H. & Wassef, Hilda Y., 1983. **Notes on African *Haemaphysalis* ticks. XV.H. *(Rhipistoma) norvali* sp. n., a hedgehog parasite of the *H. (R,) spinulosa* group in Zimbabwe (Acarina: Ixodidae).** *Onderstepoort Journal of Veterinary Research* **,50,** 183-189 (1983).

The male, female, nymph, and larva of *Haemaphysalis (Rhipistoma) norvali* sp. n., are described and compared with other members of the *H. (R.) spinulosa* group. Adult *H. (R.) norvali* parasitize in the southern African hedgehog, *Erinaceus frontalis* Smith, in Matabeleland, Zimbabwe. Immatures were reared on a laboratory rabbit.

ABSTRACT: Bastianello, Stella S., 1983. **A survey on neoplasia in domestic species over a 40-year period from 1935 to 1974 in the Republic of South Africa. VI. Tumours occurring in dogs.** *Onderstepoort Journal of Veterinary Research,* **50,** 199-220 (1983).

A survey was carried out on all canine neoplasms recorded in the registration files of the Section of Pathology of the Veterinary Research Institute at Onderstepoort over a 40-year period from 1935 to 1974. The neoplasms were divided and tabulated into 14 groups according to body systems or tissue types.

A total of 3 388 neoplasms were recorded. The 5 most frequently affected body systems were the mesenchymal tissues (33,7 %) the skin and adnexa (20,8 %) the female genital tract (10,2 %), the lymphohaemopoietic tissues (8,9 %) and the male genital tract (5,8 %). Mastocytomas, the most frequently encountered type of tumour, accounted for 12,7 % of all the neoplasms, followed by lymphosarcomas, malenomas, squamous cell carcinomas, basal cell tumours, haemangiosarcomas and histiocytomas.

A variety of mesenchymal tumours were encountered, the most common types being mastocytomas and histiocytomas as well as tumours of vascular, fibrous and adipose tissue origin. The principal cutaneous tumours included basal cell tumours, squamous cell carcinomas, perianal gland tumours and melanomas. Eighty per cent of the neoplasms of the female genital tract were mammary tumours, 50 % of which were mixed mammary tumours, whilst the principal neoplasms of the male genital tract involved the testes, of which Sertoli cell tumours were the commonest type. The majority of the digestive tract neoplasms occurred in the oral cavity, the most frequently recorded types being imflammatory epulides and malanomas. Osteosarcomas, neurofibromas and thyroid carcinomas were, respectively, the most frequently encountered neoplasms of the skeletal, nervous and endocrine systems. Pulmonary adenocarcinomas, melanomas and cholangiocarcinomas were the commonest tumours of the lung, eye and liver.

ABSTRACT: Horak, I.G., Potgieter, F.T., Walker, Jane B., de Vos, V. & Boomker, J., 1983. **The ixodid tick burdens of various large ruminant species in South Africa nature reserves.** *Onderstepoort Journal of Veterinary Research,* **50,** 221-228 (1983).

The ixodid tick burdens of eland *(Taurotragus oryx),* greater kudu *(Tragelaphus strepsiceros),* nyala *(Tragelaphus angasi),* bushbuck *(Tragelaphus scriptus)* and giraffe *(Giraffa camelopardalis)* in the Kruger National Park, Transvaal; of African buffalo *(Syncerus caffer)* and nyala in the Hluhluwe Game Reserve, Natal; and of gemsbok *(Oryx gazella)* in the Mountain Zebra National Park, and eland in the Thomas Baines Nature Reserve and an eland and greater kudu in the Andries Vosloo Kudu Reserve, eastern Cape Province, were determined.

The tick burdens of animals shot at the same time and locality are compared, and the attachment sites of some tick species on some of the hosts are given.

ABSTRACT: Cameron, C.M., van Biljon, B.J., Botha, W.J.S. & Knoetze, P.C., 1983. **Comparison of oil adjuvant and aluminium phosphate-absorbed toxoid for the passive immunization of lambs against tetanus.** *Onderstepoort Journal of Veterinary Research,* **50,** 229-231 (1983).

Immunization of ewes with oil emulsion toxoid followed by an aluminium phosphate-absorbed toxoid both containing 10 Lf per dose, resulted in a very high antitoxin level in their lambs. Two injections of aluminium phosphate-absorbed toxoid also imparted a passive immunity to lambs which is considered to be adequate to protect them against tetanus for 4 weeks after birth.

THE IMMUNOLOGICAL BASIS OF HOST RESISTANCE TO TICKS – A REVIEW

P.T. OBEREM*

ABSTRACT: Oberem, P.T. **The immunological basis of host resistance to ticks – a review.** *Journal of the South African Veterinary Association* (1984) **55** No. 4, 215-217 (En). Section of Entomology, Veterinary Research Institute, P.O. Box 12502, 0110 Onderstepoort, Republic of South Africa.

The immunological mechanisms including humoral, cellular and hypersensitivity reactions, involved in host resistance to ticks, their effects on the ticks, the factor affecting the level of resistance and possible consequences are reviewed.

Key words: Host resistance, ticks, immunological mechanisms.

INTRODUCTION

The findings of Sutherst & Utech[24], Utech et al.[36], Wharton et al.[27] and others on host resistance to ticks were successfully implemented to control *Boophilus microplus* infestations of cattle in Queensland (R W Hewetson 1984, Rous Mill, 2477 Astonville, Australia, personal communication). The possibility of employing similar tick control methods in South Africa is now being investigated (A M Spickett 1984 Veterinary Research Institute, Onderstepoort, personal communication).

In this publication an attempt is made to review the research which has been done on the mechanisms, mainly immunological, involved in host resistance to ticks. It is intended as a foundation upon which local researchers, new in this field of research, can build. The review by Willadsen[33] on this subject has served as a basis for this review and his format has been followed.

Host resistance to ticks is a broad concept which includes what is today regarded as acquired immunity to ticks as well as other characteristics, inherent in an animal, that are sometimes referred to as innate resistance. There is, however, very little evidence of the latter except for the work of Bonsma[6] who found cattle with thick moveable hides and smooth glossy hair-coats more resistant than cattle with opposite hide and coat types.

The mechanisms responsible for the manifestation described as host resistance have been studied widely since the first reports of this phenomenon in the 1930's. No single host-parasite relationship has yet been fully understood. For this reason, instead of research findings being catalogued, the broader principles of various host-parasite systems have been outlined.

The mechanisms involved have been discussed under various headings. The divisions are, however, artificial and it must be borne in mind that immunological process are no longer strictly classifiable as unique either cellular or humoral.

THE NATURE OF THE IMMUNOLOGICAL RESPONSE

A wide variety of immune responses have been shown to be involved in the rejection, and/or destruction of different parasites in the various host-parasite relationships.

Humoral immunity (including antibody formation and complement activation) and cellular immunity, as well as both immediate and delayed hypersensitivity reactions, have all been described separately. Although the importance of each varies from one host-parasite system to the next, there is evidence that all these responses are involved to varying degrees in all systems.

Immediate hypersensitivity reactions

The only tick-host system where this phenomenon has been well studied and documented is that involving *B. microplus* on cattle[33].

Larvae made shorter, repeated attempts to attach on highly resistant animals compared with their attachment pattern on less resistant hosts. Koudstaal et al.[20] showed that grooming activity resulted in a greater number of larvae initially attaching successfully. This suggests a response which causes irritation to the host and is sufficiently unpleasant to the tick to make it move frequently.

Willadsen et al.[35] showed a two-fold increase in the average total histamine content of the skin of resistant as compared to less resistant cattle; injection of histamine under an attached tick will cause it to detach[18]. The immediate hypersensitivity seen after infestation with ticks or after intradermal injection of highly purified tick allergens[34] has also been shown with *Dermacentor variabilis* on guinea-pigs[2] and with *Ixodes holocyclus* on guinea pigs and cattle[3].

These reactions are not seen on initial infestation or injection of tick-naive animals. The histamine reaction, triggered by the host's immune reponse to larval attachment, is probably responsible for the vasodilation of skin capillaries described by Hales et al[16]. These responses change the microclimate against the skin making it unsuitable for the attachment of tick larvae.

Further proof of the important role played by histamine was given by Wikel[29], who combined histamine type 1 and histamine type 2 antagonists to inhibit the expression of immunity to ticks. Chinery & Ayitey-Smith[12] showed *Rhipicephalus sanguineus* to have a histamine-blocking substance in its salivary glands which may be a defence against this mode of immunity to ticks.

Humoral immunity

As early as 1939, Träger[26] transferred partial immunity against *D. variabilis* to a susceptible animal using serum from a resistant animal. These findings were repeated with *I. ricinus* on rabbits[8]. Earlier it[7] had been shown that the serum gamma globulin concentration is significantly increased following tick infestation of cattle with *B. microplus*. He also demonstrated specific and non-specific antibodies to the salivary glands of adult ticks in the sera. The rise in serum antibody titre co-incided with the acquisition of immunity. Roberts & Kerr[22] also

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transfered passive immunity agains *B. microplus* with serum from resistant cattle to a previously unexposed, susceptible calf.

The specific effects that these antibodies have on the ticks are undetermined. Antibodies are able to cross the digestive tract of the tick unharmed[1]. Any organ of the tick including its reproductive tract may therefore be affected. The inhibition of feeding enzymes (anticoagulants, etc) has often been suggested[33] but this is as yet unsubstantiated. Tracey-Patte[25] found that the activity of an esterase enzyme, secreted by *B. microplus* into the skin of a host within an hour of attachment, is removed by a host previously exposed to these ticks. A previously unexposed host is not able to counteract this enzyme activity.

The involvement of complement was indicated by Willadsen[33] when it was shown that *B. microplus* larvae secrete a protein, capable of inhibiting bovine complement, into the host.

Delayed hypersensitivity reaction

Wikel et al.[32] found that an intradermal injection with antigenic material from *D. andersoni* into tick resistant guinea pigs gave significant delayed hypersensitivity reactions with a maximum response at ± 48 hours. Allen & Kemp[4] expanded on this and described, after histological studies that this delayed hypersensitivity correlated time-wise with the basophil cell infiltration and degranulation at the bite site. Hence they described it as a delayed cutaneous basophilic hypersensitivity. The basophils of guinea pigs are a major source of histamine, causing a reaction similar to that described under immediate hypersensitivity. Unsensitized tick-naive guinea pigs respond, in contrast to the above, initially with a neutrophil and later with an eosinophil and erythrocyte infiltration at the bite site of *Amblyomma americanum*[11]. The primary basophil response, followed by an eosinophil infiltration, in resistant guinea pigs is, according to Wikel & Allen[31] a T-lymphocyte dependent response.

Brown et al.[10] used rabbit anti-guinea pig basophil serum given at tick challenge to abrogate the resistance in guinea pigs. Anti-eosinophil serum also reduced resistance significantly, although less completely than anti-basophil serum, suggesting basophil-eosinophil co-operation in the mediation of immunity to ticks.

Cellular immunity

Apart from the indications for the involvement of T-lymphocytes given above[31] clear evidence of their involvement has only recently been reported. Wikel[30 32] used the responsiveness of lymphocytes of guinea pigs to T-lymphocyte mitogens, concanavalin A and phytohaemmagglutinin successfully as a measure of immunity to ticks. He showed a significant cellular response to low doses of salivary gland antigen while larger doses significantly depressed the response. This indicates that the paucity of evidence for cellular involvement is due to the small number of systems investigated and the complexity of the techniques required.

THE EXPRESSION OF IMMUNITY

The expression of these immune responses varies greatly depending on the host and tick species involved. The effects on the parasite range from rejection with little or no damage to the tick, to interference with feeding, reduction in engorgement weights, inhibition of egg laying and decreased viability of eggs, to the death of the parasite on the host[33].

It seems from the many examples in the literature, as Willadsen[33] suggests, that the more finely adapted, more host-specific species of parasite, such as the one-host-tick, *B. microplus*, are rejected without any damage[21]. On the other hand parasites with more catholic tastes such as *I. holocyclus,* a 3-host-tick, are rejected in a more dramatic fashion. Doube & Kemp[13] describe the death of this parasite in situ on the host.

DISCUSSION

The fact that the immune system plays such a major role in host resistance to ticks raises a number of interesting considerations.

a. The influence of external factors on the immune status of the host animal.

The following factors have been shown to affect the level of tick resistance.

i. Stress, particularly nutrition, can seriously reduce the level of resistance[9 23].
ii Shortening photoperiodicity will also reduce resistance[23].
iii. The reproductive status has the expected effects; probably stress related (R.W. Hewetson 1984 Rous Mill, 2477 Astonville, Australia, personal communication).
iv. Immunosuppressive drugs and anti-histamines will also reduce immunity to ticks[9].
v. Haemoparasites such as *Babesia bovis* and *Trypanosoma congolense*[17] have been reported to increase the susceptibility of infected animals to ticks.
vi. It has also been shown that a challenge of *D. andersoni* adults induces a variable but significant degree of reduction of host immune responsiveness[32]. We can also speculate on the effects of a large challenge of *R. appendiculatus* on this immunity.

These factors, plus the fact that the responses vary so greatly from one host-parasite system to another, must be borne in mind when planning a trial or interpreting results of trials investigating host resistance to ticks.

Vaccination against ticks

With immunity playing such a major part in tick resistance the possibility of improving resistance to ticks by vaccination has been considered. Numerous attempts have already been made to artificially immunise host animals with tick extracts.

Whole tick extracts of *D. variabilis*[26] and *D. andersoni*[15] have been tested for antigenicity and the stimulation of resistance as have various tick organs. Salivary gland extracts of *Hyalomma anatolicum*[19]; *R. sanguineus*[14]; *B. microplus*[7]; and *D. andersoni*[28] have all been used.

Allen & Humphreys[3] used midgut and reproductive organs and in a second trial all the internal organs of *D. andersoni.*

The effect of vaccination with haemolymph has also been assessed[5].

All these methods produced a measure of resistance in previously tick-naive, susceptible animals. None

however showed any higher degree of resistance than was obtained after natural challenge. Whether vaccination could induce an enhanced immunity not elicited by natural tick challenge is still to be determined; if this is successful it could have enormous economic implications.

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ABSTRACTS

ABSTRACT: Cameron, C.M. & Bester, Faith J., 1983. **The inefficacy of polivalent *Pasteurella multocida* vaccines for sheep.** *Onderstepoort Journal of Veterinary Reasearch,* **50,** 101-104 (1983).

Immunity assays on sheep sera using passive mouse protection tests showed that vaccines containing more than 4 strains of *Pasteurella multocida* did not give a good immunity. The immune response was not enhanced by the use of an oil adjuvant, and high concentrations of bacteria had only a partial positive effect.

Attempts to extract selectively the protection-inducing antigen(s) from *P. multocida* by veronal, phenol or potassium thiocyanate extraction were unsuccessful. Furthermore, it was found that sheep antisera to the recognized type strains of *P. multocida* afforded only limited protection against a number of field strains.

We concluded from this that successful immunization against ovine pasteurellosis will depend on either the identification of a strain of *P. multocida* that gives a wide spectrum of immunity or the discovery of a live mutant suitable for vaccine production and the definition of cultural conditions that promote the expression of a common immunizing antigen.

ABSTRACT: Scialdo-Krecek, Rosina C., 1983. **Studies on the parasites of zebras. I. Nematodes of the Burchell's zebra in the Kruger National Park.** *Onderstepoort Journal of Veterinary Research,* **50,** 111-114 (1983).

Nineteen species of gastro-intestinal nematodes were recovered from 10 Burchell's zebra. These include: *Cyathostomum alveatum, C. montgomeryi* and *C. tetracanthum; Cylicocyclus auriculantus, C. gyalocephaloides, C. insigne* and *C. riramosus; Cylicodontophorus schürmanni* and *Cylicodontophorus* n. sp., *Cylicostephanus bidentatus, C. calicatus* and *C. minutus; Poteriostomum ratzii, Craterostomum acuticaudatum, Triodontophorys minor, Habronema majus, H. muscae, H. zebrae,* and *Draschia megastoma,* as well as *Cylindropharynx* spp. The highest burdens encountered were those of *Cylicocyclus triramosus* (159 491), *Cylindropharynx* (19 875), *Cylicocyclus auriculatus* (15 792), *Cylicostephanus calicatus* (16 658) and *Cyathostomum tetracanthum* (13 723). The nematodes consistently present in all zebras were: *Cylicostephanus calicatus, Cylindropharynx* spp. and *Draschia megastoma.*

ABSTRACT: Bessenger, R. & Schoeman, J.H., 1983. **Serological response of cattle to infection with *Babesia bigemina* and *Babesia bovis* in Southern Africa.** *Onderstepoort Journal of Veterinary Research,* **50,** 115-117 (1983).

The indirect fluorescent antibody test was used to measure the antibody response of cattle for 8 weeks after infection with either *Babesia bigemina, Babesia bovis,* or a combination of both species. Serological cross-reactions were observed between the 2 species, but these were most marked when *B. bigemina* antigen was used. In animals infected with both *Babesia* spp., the *B. bigemina* reaction appeared to suppress the *B. bovis* reaction.

ABSTRACT: Littlejohn, A., Button, C. & Bowles, Felicity, 1983. **Studies on the physiopathology of chronic obstructive pulmonary disease in the horse. VIII. Mean modal vectors of the P wave and the QRS complex.** *Onderstepoort Journal of Veterinary Research,* **50,** 119-124 (1983).

Mean modal vectors of P_1, P_2 and QRS were determined in the 3 planes of a semi-orthogonal EKG lead system in 17 horses and ponies with chronic obstructive pulmonary disease (COPD) and in 17 clinically normal horses and ponies. Subjects were paired so that the heart rates of each pair were not dissimilar by more than 2 cycles per minute.

Probably significant differences were observed between the mean angles of P_1 vectors in the transverse and sagittal planes (T plane, normal = 324° ± 24,6°, COPD = 342° ± 21,0°,t = 2,0,P < 0,05; S plane, normal = 331° ± 22,6°, COPD = 348° ± 16,2°,t = 2,52,P < 0,02).

There were no significant differences between the mean angles of planar modal QRS vectors of normal subjects and those of COPD subjects.

ABSTRACT: Jansen, B.C., Hayes, Marianna & Knoetze, P.C., 1983. **The reaction of ovine neutrophils to *Histophilus ovis* in relation to genital infection of rams.** *Onderstepoort Journal of Veterinary Research,* **50,** 125-132 (1983).

Histophilus ovis was shown to be phagocytized by neutrophils when the organisms enter the lumen of the reproductive tract of the ram. The phagocytosis and destruction of *H. ovis* by neutrophils was demonstrated *in vitro* by the viable count method and by electron microscopy. It was shown that immunoglobulins and complement had no influence on the phagocytosis and destruction of *H. ovis*. Phagocytosis and killing of *H. ovis* was accomplished equally well by neutrophils from immunized and non-immunized rams. Immunized rams showed a massive infiltration of neutrophils into the walls, epithelium and lumen of their ampullae when dead *H. ovis* were introduced into their lumen.

ABSTRACT: Du Plessis, J.L., Jansen, B.C. & Prozensky, L., 1983. **Heartwater in Angora goats. I. Immunity subsequent to artificial infection and treatment.** *Onderstepoort Journal of Veterinary Research,* **50,** 137-143 (1983).

This study confirmed reports that Angora goats are highly susceptible to *Cowdria ruminantium* and showed that immunization of this breed against heartwater may be difficult and hazardous. It was found that if goats were treated on the 2nd or 3rd day of the febrile reaction following the intravenous inoculation of the heartwater agent, few animals survived the infection. If, on the other hand, treatment was instituted on the 1st day of the reaction, the chances of survival were good, but the immunity of the goats to subsequent challenge was poor.

QUESTIONS & ANSWERS **VRAE EN ANTWOORDE**

TUBERCULOSIS IN MILCH GOATS

QUESTION

Although on a limited scale, there is a regular countrywide demand for goat's milk with which to feed infants who are intolerant of or allergic to cow's milk. Particularly where it is to be fed raw, such goat's milk should, of course, be safe and at least free from those infections transmissible to man. Apart from Q-fever and brucellosis, one inevitably thinks of tuberculosis. What, then, are the dangers of tubercle-contaminated goat's milk? To which species of *Mycobacterium* is the goat susceptible? Can the tuberculin test be used to screen milch goats for tuberculosis? Where and how should this be done?

ANSWER

The goat is rather susceptible to infection with *M. bovis*, less so to *M. avium* and even less to *M. tuberculosis*. The widely held belief that goat's milk is necessarily safe is not based on fact; rather is the opposite true. Goats maintained with infected cattle or on infected premises are more than likely to be tuberculous. As in cattle and pigs, tuberculosis in goats results in an exudative rather than a proliferative tissue reaction; aerogenic infection is usual and the respiratory organs commonly constitute the primary complex. Protracted nodular generalisation usually follows but instances of chronic organ tuberculosis are also encountered. The typical picture of chronic isolated tuberculosis of the udder is quite common: a gradually spreading form of lobular-infiltrative tuberculosis with decreasing lymphonodular change similar to that which occurs in cases of chronic tuberculous bovine mastitis. It usually presents as circumscribed caseous nodules with corresponding lymphonodular changes. Organisms are present in the secretion from the very beginning. The excretion of mycobacteria in the milk of tuberculin-positive goats showing no clinical evidence of tuberculous mastitis is, however, also on record. Goats having lived or living with infected cattle must be considered as a source of bovine tubercle infection for consumers of their milk as well as for cattle in general.

Outbreaks of caprine tuberculosis due to *M. avium*, including instances of tuberculous mastitis, are on record. Progress in methods of typing mycobacteria has led to sometimes controversial new types being identified. *M. africanum* causes human disease and possesses characteristics which place it somewhere between *M. tuberculosis* and *M. bovis*, strains being closer to either the former or the latter. Goats appear to be rather susceptible to *M. africanum*, all goats infected with 6 strains having developed severe miliary pulmonary tuberculosis while none of the strains produced disease in calves.

Regarding the tuberculin test, it has the same value as in cattle. Testing of goats (and sheep, for that matter) that are in contact with cattle should be part of a bovine eradication scheme. The intradermal injection of 5 000 i.u. of tuberculin with a reading following 72 h later, is recommended. (The Onderstepoort bovine tuberculin contains 7 000 iu/0,1 ml and the avian 2 500 iu/0,1 ml.) Because of the narrowness of the goat's neck it is recommended that both sides are used in the comparative avian/mammalian test. The caudal skin fold and the skin at the base of the ear have also been used, and in sheep an intrapalpebral injection is recommended.

In Britain, goats reacting positively to the standard intradermal mammalian tuberculin test have been found to be infected with mycobacteria which were identical to *M. paratuberculosis* in growth rate and mycobactin requirements but which failed to produce Johne's disease in calves and could therefore have been variants of *M. avium*. Natural infection in goats led to death after a long prepatent period and 6-12 months of disease typical of Johne's disease.

It is therefore concluded that milk for human consumption should be obtained only from goats that are free from tuberculosis; that the tuberculin test can be used as in cattle in ensuring and maintaining such a status; that although bovines are the most important source of tuberculous infection of goats, human tuberculotics infected with some strains of *M. africanum* may also constitute a danger because of the high degree of susceptibility of the goat to the latter.

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ABSTRACTS

ABSTRACT: Horak, I.G., Biggs, H.C., Hanssen, Tammy S.T., Hanssen, Rose E., 1983. **The prevalence of helminth and arthopod parasites of warthog, *Phacochoerus aethiopicus,* in South West Africa/Namibia.** *Onderstepoort Journal of Veterinary Research,* **50,** 145-148 (1983).

A total of 38 warthog, *Phacochoerus aethiopicus,* shot on a farm in northern South West Africa/Namibia, were examined for internal and external parasites at montly intervals over a period of 13 months. They harboured cestodes, 9 nematode species, 6 ixodid tick species and 1 species each of an argasid tick, a flea, a louse and larvae of a dipteran fly.

Clear patterns of seasonal abundance could be determined only for the spirurid stomach worm, *Physocephalus sexalatus,* and the sucking louse, *Haematopinus phachoeri.*

ABSTRACT: Bastianello, Stella S., 1983. **A survey of neoplasia in domestic species over a 40-year period from 1935 to 1974 in the Republic of South Africa. V. Tumours occurring in the cat.** *Onderstepoort Journal of Veterinary Research,* **50,** 105-110 (1983).

A total of 243 neoplasms were recorded in a survey of all the feline neoplasms which are reported in the registration files of the Veterinary Research Institute, Onderstepoort, Republic of South Africa, covering a 40-year period from 1935 to 1974.

The tissues most commonly neoplastic were the skin, followed by the lymphoid tissue, the digestive tract and the genital system, which together accounted for 76,6 % of the total tumours. Squamous cell carcinomas, the commonest type of tumour, accounted for 65 (26,7 %) of the 243 neoplasms, followed by lymphosarcomas with 50 (20,5 %). The majority of squamous cell carcinomas involved the skin, especially that of the ear and nose. A reasonably high proportion of these tumours also occurred on the tongue and eyelid. The commonest form of distribution for lymphosarcomas was the multicentric form, followed by the alimentary, the renal and thymic forms.

Squamous cell carcinomas were the most frequent type of skin tumours, followed by basal cell tumours, mastocytomas and melanomas. The digestive tract accounted for 33 (13,5 %) of the neoplasms, the 3 most commonly encountered being squamous cell carcinomas, lymphosarcomas and intestinal adenocarcinomas. The mammary gland tumours accounted for 23 (9,5 %) of the total, 61 % of which were carcinomas.

Other tumours encountered were fibromas, fibrosarcomas involving particularly the skin, melanomas of the skin or eye, osteosarcomas, hepatocellular carcinomas and haemangiosarcomas.

ABSTRACT: Bastianello, Stella S., 1983. **A survey on neoplasia in domestic species over a 40-year period from 1935 to 1974 in the Republic of South Africa. IV. Tumours occurring in Equidae. Onderstepoort Journal of Veterinary Research, 50,** 91-96 (1983).

A survey was carried out on the neoplasms of horses, donkeys and mules which are recorded in the registration files of the Section of Pathology of the Veterinary Research Institute. Onderstepoort, in the Republic of South Africa, over a 40-year period from 1935 to 1974.

A total of 378 tumours are recorded, 339 of which were in horses, 32 in mules and 7 in donkeys. Sarcoids (38 %), squamous cell carcinomas (23,5 %), fibromas (8,2 %), melanomas (8,0 %), papillomas (4,5 %), fibrosarcomas (3,4 %) and lymphosarcomas (3,0 %) accounted for 88,6 % of the total.

Of the 58 sarcoids for which the site or origin was determined, 46,5 % occurred on the head, 32,8 % on the chest and abdomen, 19 % on the limbs especially below the level of the carpus or hock and 1,7 % on the neck.

Fifty percent of the 89 squamous cell carcinomas occurred on or around the eyes, especially on the eyelids or nictitating membrane, 23 % involved in the penis and/or prepuce, while just over 20 % arose on the skin. The melanomas involved the skin and eye, whilst papillomas originated premarily on the skin and less frequently on the penis.

ABSTRACT: Erasmus, J.A., 1983. **The usefulness of the API 20 E classification system in the identification of *Actinobacillus actinomycetem comitans, Actinobacillus seminis* and *Pasteurella haemolytica.*** *Onderstepoort Journal of Veterinary Research,* **50,** 97-99 (1983).

The prepuces of lambs aged 6-8 months and semen of 2 adult rams were found to be infected with gram-negative, non-motile, non-haemolytic, pleomorphic bacilli. These organisms were compared with those of known strains of *Actinobacillus actinomycetem comitans, Actinobacillus seminis* and *Pasteurella haemolytica,* using the API 20 E classification system. Applying the principles of numerical taxonomy, the majority of suspected strains of *A. seminis* could be classified as *A. actinomycetem comitans* and 3 examples as *Histophilus ovis.* Although some of the suspected strains of *A. seminis* could be classified as *P. haemolytica,* obvious differences between the genera *Actinobacillus* and *Pasteurella* were evident.

J.H.R. BISSCHOP

20 December 1898 – 28 April 1984

John Henri Roosegaarde – of Baas Bisschop (omdat hy almal as Baas aangespreek het), of Jack vir sy vriende, of oom Jack vir my wat vier jaar lank as student by hulle ingewoon het op Onderstepoort – se heengaan laat 'n besondere leemte in ons professie en in dié van ons wat bevoorreg was om hom te ken.

Sy vader was 'n Hollander uit Batavië en sy moeder, Ada Veal, uit Engeland. Geen wonder nie dat Jack so 'n unieke mengsel van Viktoriaanse korrektheid en Hollandse uitgesprokenheid was.

As student aan die destydse Transvaalse Universiteitskollege, en lid van die eerste veeartsenykundegroep, was hy geen onbekende op sy vinnige motorfiets nie. Voor op die stuur het sy meisie gesit en agterop nog 'n paartjie. Wat die prentjie nóg interessanter maak is die feit dat die jonge dame op die handvatsels niemand anders as Helene, jongste dogter van die waardige Ds. Neethling van Lydenburg, is nie!

Al Jolson was sy gunsteling sanger – veral as hy die treffer van die twintigerjare, Sonny Boy, gesing het. Die uitbundigheid van Fred Astaire en Ginger Rogers se danspassies het hom bekoor – want hy en Helene het dit heerlik gevind om te dans, goèd te dans. Jack het graag klavier gespeel en het ook as student sy eie dansorkes gehad.

Jack en Helene se twee seuns, Philip en Gys, het albei veeartse geword. Philip en ek was klasmaats. In die vroeë vyftigerjare toe ek vier jaar lank deel was van die huishouding was dit reeds duidelik dat Helene 'n ernstige hartkwaal het. Op doktersvoorskrif moes sy een dag per week in die bed bly om te rus. Haar humor en spontaniteit was vir oom Jack 'n eindelose plesier. Trouens, die romantiese klank van hulle studentedae het dwarsdeur hulle huwelik geloop. Na so 'n dag in die bed as die res van ons sommer net nog 'n rukkie sit en ginnegaap om die eetkamertafel, het oom Jack gereeld vir tant Leen ingedra om te deel in die geselligheid. Nes 'n prinses, het ek altyd gedink.

Elke weeksaand het hy tot elfuur by sy lessenaar gesit en werk. Daar was verslae oor sy proefwerk op Armoedsvlakte (waaruit sy bekende publikasie "Feeding phosphates to cattle" voortgevloei het), sy intensiewe ondersoeke as lid van die Tomlinson-kommissie en, sonder feil, die dag se gebeure wat aangeteken word in sy dagboek in sy fyn, netjiese handskrif.

Hierdie dissipline was die merk van al sy aktiwiteite. Sy wêreld was ge-orden. Dinge was op hulle plek en iets was óf reg óf verkeerd. Dit het hom soms onbuigbaar laat voorkom, ook vir sy kinders. Maar soos Gys vir my sê, sy pa het ook geluister en wou graag verstaan. Gys en Philip was altyd bewus van sy diep liefde en empatie.

Geen wonder dat Helene se dood in 1960 hom so diep geraak het nie. En toe verongeluk Philip in 1973. Die rasionele Viktoriaan moes nou dieper soek vir die sin van soveel pyn. Toe ek hom jare later weer sien het hy my vertel dat hy nou seker is dat die lewe aangaan na die dood, en dat dit vir hom 'n enorme troos is.

Aan hierdie baanbreker en "gentleman" van ons professie, professor en hoof van die Departement Soötegnologie van 1936 tot 1962, ontvanger van die S.A.V.V. se goue medalje in 1977, self-kritiese navorser en beminlike mens – ons dankbare hulde.

Malie Smuts

AWARD **TOEKENNING**

GOUE MEDALJE VAN DIE S.A.V.V. VIR 1984

WILLIAM HENRY GERNEKE

Die Goue Medalje van die Suid-Afrikaanse Veterinêre Vereniging ter erkenning van voortreflike wetenskaplike prestasie en/of 'n betekenisvolle bydrae tot die vooruitgang van die veterinêre wetenskap is vanjaar, die eerste jaar wat 'n nie-veearts daarvoor kwalifiseer, aan 'n uiters verdienstelike ontvanger en ere-lid van die Vereniging, prof Bill Gerneke toegeken.

William Henry Gerneke is op 1 Julie 1924 in Johannesburg gebore. Hy slaag die matrikulasie-eksamen van die Oranje Vrystaat in 1941 en behaal die BSc-graad van UKOVS in 1944 met Skeikunde en Dierkunde as hoofvakke.

Hy begin sy loopbaan as Tegniese Assistent in die Patologielaboratorium van die Frére Hospitaal in Oos-Londen waar hy vir 2 jaar werksaam is.

In 1947 word hy aangestel as Assistent-vakkundige Beampte in die dept Anatomie van die Navorsingsinstituut vir Veeartsenykunde te Onderstepoort en vorder hy binne 12 jaar tot Eerste Vakkundige Beampte.

Tydens hierdie periode word hy geskool in elke faset van tegniese vaardigheid en insig in die voorbereiding van histologiese preparate. Sy leermeester is prof Cecil Jackson – bekend vir sy uitmuntende kennis en onverbiddelike perfeksionisme.

Hy word in 1948 Demonstrateur in Histologie en so begin sy akademiese loopbaan in die Fakulteit Veeartsenykunde, Universiteit van Pretoria, waar hy in 1957 aangestel word as deeltydse lektor in Histologie.

Dit was in hierdie periode dat hy met kenmerkende deursettingsvermoë ingeskryf het vir sy meestersgraad. Die titel van sy verhandeling is "The embryological development of the pharyngeal region of the sheep". Hy behaal die MSc-graad met lof aan die Universiteit van Pretoria in Sept 1983.

In Februarie 1963 word hy Lektor in die departement Anatomie met hoofrigtings Histologie, Embriologie, Sitologie en Sitogenetika.

Dit lui 'n periode van akademiese en wetenskaplike produktiwiteit in: In 1966 word die DSc-graad (Pret) aan hom toegeken vir sy proefskrif "Cytognetic investigations on normal and malformed animals with special reference to intersexes".

Hy lewer oor 'n periode van 16 jaar 30 referate op wetenskaplike kongresse en 36 wetenskaplike publikasies verskyn uit sy pen.

Enkele van sy uitstaande navorsingsbevindings wat wêreldwye belangstelling uitgelok het is: Die eerste beskrywing in wêreldliteratuur van steriliteit in 'n bul a.g.v. tweelingskap met 'n kween ("free martin") asook die eerste beskrywing van chromosomale bewys van die kween-kondisie by skape.

Die eerste beskrywing van die unieke ovulasie, partenogenetiese deling en blywende ova in die fallopiese buis by die merrie.

Die eerste beskrywing van die unieke Langerhanselgranules in makrofage van die rumenepiteel en die eerste aanvaarbare konsep vir hul betekenis in die liggaam.

Verder is hy verantwoordelik vir die daarstelling van chromosoomstudies aan die Instituut en Fakulteit Veeartsenykunde – 'n aksie wat baie interessante navorsing en verskeie publikasies tot gevolg het.

Hy publiseer in 1982 die heel eerste Embriologiehandboek in Afrikaans. Sy kunstenaarstalent word bewys deurdat hyself die illustrasies doen.

Hy word ere-lid van die S.A. Veterinêre-Mediese Vereniging in 1961, is stigters- en komiteelid van die Anatomiese Vereniging van Suider-Afrika en lid van die Wêreldvereniging van Anatome, die Biologiese Vereniging en die Elektronmikroskopiese Vereniging van Suidelike Afrika.

In 1975 word hy bevorder tot mede-professor en in 1983 tot volle professor en bereik hy die hoogste sport in 'n merkwaardige loopbaan, 37 jaar na hy as tegnikus

begin werk het. Buiten sy voortreflike wetenskaplike prestasies het hy ook 'n wesenlike bydrae tot die vooruitgang van die veterinêre wetenskap gelewer deur die hoogstaande kwaliteit van die opleiding wat hy voorgraads en nagraads aan studente en kollegas in sy vakgebied gegee het.

As mens is Bill vir almal wat hom ken 'n opregte, hulpvaardige vriend en lojale kollega. Hy is getroud met Orpa, 'n innemende en uiters bekwame vrou wat hom dwarsdeur sy loopbaan bygestaan en onderskraag het.

Hier het ons 'n wetenskaplike van formaat wat in Suid-Afrika en oorsee bekendheid verwerf het en respek afgedwing het vir sy wetenskaplike oorspronklikheid en integriteit en in Suider-Afrika is daar stellig geen groter kundige op die gebied van die vergelykende histologie as W.H. Gerneke nie – hy is inderdaad die doyen van veterinêre en mediese histologie in Suid-Afrika.

Die Veterinêre Professie eer hom en as blyk van dank en respek is die Goue Medalje vir 1984 aan hom toegeken.

AWARD TOEKENNING

SILWER MEDALJE VAN DIE S.A.V.A. VIR 1984

FREDERIK JOHANNES VELDMAN

Dit is besonder gepas dat die eerste Silwer Medalje van die Vereniging, die hoogste erkenning vir uitstaande, langdurige diens aan en bevordering van die veterinêre professie, aan dr. F.J. Veldman toegeken word.

Hy is op 24 Desember 1918 in die Piet Retiefdistrik gebore en matrikuleer in 1936 te Ermelo. In 1941 kwalifiseer hy met lof as veearts te Onderstepoort en is hy dan ook die ontvanger van die gesogte Sir Arnold Theiler-gedenkmedalje.

As een van die vroeë pioniers van privaat praktyk begin hy in Kaapstad saam met mnr P Robertson, een van die laaste "Registered Veterinary Surgeons", werk.

Hy begin kort daarna 'n takpraktyk in Stellenbosch en werk vir sowat 10 jaar hier. Hy het vroeg ontdek dat vir 'n veearts om suksesvol te wees, mens ook intelligent en met gesag oor diereproduksie in die algemeen moet kan praat. Sy ondervinding hier en sy sterk aanvoeling vir boerderybestuur, voeding en produksie kwalifiseer hom ideaal vir sy loopbaan as veearts in die nywerheid.

Hy sluit in 1950 by die firma Agricura as veearts aan. Toe dr Mönnig aftree word hy Besturende Direkteur en toe hy enkele maande gelede die tuig neerlê is hy Direkteur van Skakeling van Agrihold. Hy is huidig Direkteur en Deeltydse Konsultant van Agrihold, 'n divisie van Sentrachem. In die nywerheid was hy weer eens 'n pionier en het hy bewys dat 'n veearts in 'n professionele hoedanigheid 'n onmisbare rol het om hier te vervul. Deur sy ywer en toedoen beklee die professie steeds 'n ereposisie hier.

Hy was saam met ander kollegas instrumenteel in die ontstaan van twee verenigings waarby die belang van die veearts in industrie bevestig is naamlik die Veeartse-in-Industrie-groep van die S.A.V.V. en die Landbou en Veterinêre Chemikalieë-vereniging. Hy speel 'n aktiewe rol in hierdie verenigings en was o.a. vir etlike jare voorsitter van albei.

Sy hoogste prioriteit en lewenstaak is egter nog altyd die uitdra van kennis en hiermee het hy die beeld van die professie onder die boeregemeenskap geweldig uitgebou. Hy het die besondere gawe om die boer se taal te kan praat of dit Engels, Afrikaans, Zoeloe of selfs Duits is. Hy is 'n baie gesogte spreker by lesings waarvan hy tot 6 per maand lewer.

Hy is mede-outeur van "Handboek oor Veesiektes" (Mönnig en Veldman) wat in 1953 verskyn het en was verantwoordelik vir die vertaling hiervan in Engels. Hierdie welbekende, uiters praktiese boek staan in haas elke boer (en baie veeartse) se boekrak en is twee keer hersien en 12 Afrikaanse en 5 Engelse oplae het verskyn.

As gevolg van sy besondere prestasie om as professionele voorligter met die boer te kommunikeer is hy dan ook in 1979 gepas geëer deur die Landbouskrywersvereniging met die gesogte toekenning van "Landboukundige van die Jaar".

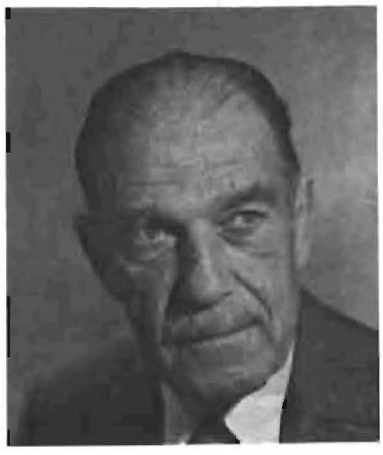
In die kunsmatige inseminasiebedryf het dr Veldman 'n reuse taak vervul. Hy is in 1962 gekies as voorsitter van die Transvaalse K.I. Koöperasie en later van Insemina en is vir baie jare al aktief betrokke by die bedryf in die hoogste uitvoerende kapasiteit. Sy grootste prestasie was egter dat grootliks deur sy ywer, bemiddeling en diplomasie die nasionale Suid-Afrikaanse K.I. Koöperasie (Taurus) in 1980 sy beslag gekry het. Die kardinale punt is dat die veterinêre professie vandag hier, in teenstelling met baie ander lande, 'n groot aandeel in die beheer van hierdie bedryf het en dus 'n wesenlike aandeel het in diereproduksie en die genetiese verbetering van die land se veestapel.

Dr Veldman is besiel met die idee dat die veearts in die landboukonteks 'n wesenlike rol moet speel en dat daar vir hom 'n werksveld gebied word wat hy moet betree. Sy ingewortelde oortuiging dat die landelike praktisyn ook 'n praktiese wetenskaplike op die gebied van veeboerdery moet wees, het grootliks daartoe gelei dat die Veterinêre Stigting gemotiveer is om die konsep van kuddebenadering na die professie uit te dra.

Hy is met Susan du Bruyn getroud en hul het drie seuns en 6 kleinkinders.

Hy het 'n rustige ongejaagde geaardheid en is 'n opreg nederige mens van onwrikbare beginselsoortuiging wat hy prakties uitleef. Die beleefdheid van hierdie ware "gentleman" is legendaries.

Dr Veldman is 'n uitstaande veearts wat deur sy hele lewe die professie uitmuntend gedien en bevorder het. Sy bydrae aan die boerderygemeenskap kan moeilik in waarde gemeet word. Hy het op professionele en wetenskaplike vlak 'n wesenlike bydrae tot die bevordering van dieregesondheid in ons land gemaak. Hy voldoen by uitstek aan die vereistes van die Vereniging se Silwer Medalje.



JACK BOSWELL AWARD FOR 1984

GEORGE CLIFFORD DENT

This award for 1984 in acknowledgement of dedicated service to the Veterinary Profession through the South African Veterinary Association was awarded to Dr Cliff Dent.

George Clifford Dent was born on 13 April 1923 and matriculated at Kingwood College, Grahamstown in 1940. He joined the South African Air Force and served with distinction as pilot in the Middle East throughout the Second World War and as instructor towards the end of this period.

In 1946 he resumed his studies and qualified as a veterinarian at Onderstepoort in 1950.

After 3 years in Government Service in the Transkei he was one of the pioneers of rural private practice in the Greytown and Ficksburg districts for a period of 10 years.

He joined the State Service again in 1963 and did sterling work at Queenstown where he initiated the investigational laboratory and took a particular interest in plant poisoning and internal parasites. Here he was involved in phenothiazine as a lick. In 1974 he was promoted to Assistant Director and transferred to Pretoria where he is now involved in import and export control, is the coordinator for tsetse fly control and is the liaison officer between the Division of Veterinary Services and the Self Governing Black States. He also handles veterinary matters between the Government and the Independant Black States and serves on the Bilateral Technical Committee in this regard.

He has always been very active in S.A.V.A. matters: He was a Founder member of the East Cape Branch and served on their executive for 9 years and as chairman for 3 years.

On his transfer to Pretoria he was elected to Federal Council in 1974 where he has served with distinction to date. His 10 years' personal experience of private practice as well as experience of State Service equipped him and gave him the insight and wisdom needed, and this, coupled with his inborn sense of fair play, made him the ideal person to be an outstanding Chairman of the Advisory Committee of the Association for 8 years. He took over from Ben le Grange and during his chairmanship of this important committee its name changed from the Disciplinary Committee to the Advisory and Ethical Committee.

He skillfully and impartially guided many arbitrations between the public and the profession and amongst practitioners during an exciting era of change in the profession where private practice in especially the small animal field became very competitive.

He is strong protagonist of the free-competition concept who does not believe in empire building, who propogated the absolute necessity of formal agreements between practitioners, locums and assistants and who believes that a satisfied client is the best advertisement for a veterinarian.

When the new Veterinary Council was established in 1983 the profession elected him as official representative of the S.A.V.A. – a very fitting tribute to a man who had put in so much effort in this field.

After the tragic death of his first wife Joan he is now married to Sybil and they have 4 children and 7 grandchildren.

Cliff Dent is a man who fully and richly deserves our in-house award for his unstinting service to the profession through the Association.

AWARD **TOEKENNING**

NAVORSINGSTOEKENNING VAN DIE S.A.V.V. VIR 1984

THEUNS STEPHANUS KELLERMAN

Die vierde ontvanger van hierdie toekenning is dr Fanie Kellerman van die Navorsingsinstituut vir Veeartsenykunde te Onderstepoort.

Hy behaal in 1957 die graad BSc.(Agric) met Plantpatologie as hoofvak aan die Universiteit van Natal en werk hierna vir 'n paar jaar as landbounavorser in Zimbabwe. Hy besluit egter om veearts te word en kwalifiseer in 1967 te Onderstepoort. Na 2 jaar as Staatsveearts te Bulawayo sluit hy in 1970 by die Navorsingsinstituut vir Veeartsenykunde, Onderstepoort aan waar hy hom in Toksikologie verdiep. Hy word Hoof van die Seksie Toksikologie en word in 1982 bevorder tot Assistent-Direkteur.

Hepatogene of sekondêre liggevoeligheid in herkouers in die RSA is van groot ekonomiese belang en is sedert die ontstaan van die NIV, Onderstepoort, bestudeer deur roemryke navorsers soos Theiler, Quin en Brown. Alhoewel dit vandag bekend is dat hepatogene liggevoeligheid in ons land deur verskeie plante, 2 fungi en 'n alg veroorsaak kan word het daar tot en met ongeveer 10 – 12 jaar gelede groot verwarring en onkunde bestaan t.o.v. die etiologie van liggevoeligheid.

Dr Kellerman se besondere kennis t.o.v. mikologie en in besonder mikotoksikologie en sy intense belangstelling in liggevoeligheid het sedertdien daartoe gelei dat die siektebeeld in geheel meer omskryf en gedefinieer is en dat die etiologie van baie van die liggevoeligheidtoestande gedeeltelik of ten volle opgelos is.

Hy het 'n groot aandeel gehad in die oorspronklike diagnosering en rapportering van twee mikotoksikoses nl. "Facial eczema" en lupinose in die RSA. Sy groot bydrae tot liggevoeligheid is egter die navorsing wat hy gedoen het ten opsigte van die gevreesde en ekonomies belangrike siekte, geeldikkop.

Sedert die dae van Theiler is dit bekend dat die dubbeltjie, *Tribulus terrestris,* geassosieer word met uitbrake van geeldikkop. As gevolg van die variërende toksisiteit van die plant is bespiegel dat die plant onder sekere klimatologiese toestande 'n labiele hepatotoksien(e) mag produseer en/of dat 'n mikotoksien moontlik hier betrokke mag wees. Na vele jare van navorsing het dr. Kellerman en medewerkers onder sy aansporing en dryfkrag in 'n pragtige spanpoging bewyse gevind dat die mikotoksien, sporidesmin wat geproduseer word deur *Pithomyces chartarum,* 'n sinergistiese aksie het met *Tribulus terrestris* in die verwekking van geeldikkop.

Verder is aangedui dat toksiese *Tribulus terrestris* 'n kristalloïede faktor(e) mag bevat wat spontaan mag presipiteer in veral die galbuise, maar dat sporidesmin as 'n snellermeganisme mag dien vir die proses. Die studies het ook lig gewerp op die moontlik patogenese van liggevoeligheid in geeldikkop nl. die blokkering van galbuise deur kristalloïede materiaal met gevolglike terugdamming van filloeritrien in die sirkulasie van die dier. Dit is 'n heel nuwe konsep wat nog nie voorheen in die literatuur geopper is nie.

Die Navorsingstoekenning het ten doel om navorsing van hoë gehalte aan te moedig en word toegeken vir die beste wetenskaplike artikel of reeks artikels wat onlangs in enige wetenskaplike joernaal gepubliseer is. Dr Kellerman se navorsing en reeks artikels oor geeldikkop en fotosensitisasie kwalifiseer uitmuntend hiervoor.

Kellerman T.S., van der Westhuizen G.C.A., Coetzer J.A.W., Roux Cecilia, Marasas W.F.O., Bath G.F., Basson P.A. 1980. Photosensitivity in South Africa. II. The experimental production of the ovine hepatogenous photosensitivity disease geeldikkop (*Tribulosis ovis*) by the simultaneous ingestion of *Tribulus terrestris* plants and cultures of *Pithomyces chartarum* containing the mycotoxin sporidesmin. Onderstepoort Journal of Veterinary Research 47: 231-261.

Coetzer J.A.W., Kellerman T.S., Sadler W., Bath G.F. 1983. Photosensitivity in South Africa. V. A comparative study of the pathology of the ovine hepatogenous photosensitivity diseases, facial eczema and geeldikkop (*Tribulosis ovis*), with special reference to their pathogenesis. Onderstepoort Journal of Veterinary Research 50: 59-71.

AWARD TOEKENNING

KLINIESE TOEKENNING VAN DIE S.A.V.V. VIR 1984

JOHANNES STEPHANUS JOUBERT ODENDAAL

Die vierde ontvanger van hierdie toekenning wat ten doel het om kliniese uitnemendheid te erken, is dr Johannes Odendaal van Bloemfontein.

Nadat hy in 1967 die graad BSc. in Chemie en Dierkunde behaal het kwalifiseer hy in 1971 as veearts aan U.P. Hy wend hom tot die privaatpraktyk en na twee jaar assistentskap begin hy sy eie praktyk te Bloemfontein waar hy die hoogste standaarde handhaaf wat hom dan ook 'n reputasie vêr buite die Vrystaat gee.

Behalwe vir sy aktiewe deelname in die aktiwiteite van die Vereniging is hy deeltydse veearts van die Proefdiereenheid van die Mediese Fakulteit aan die U.O.V.S., Bloemfontein, asook lid van die Etiese Komitee van hierdie fakulteit; het hy 'n besondere belangstelling in onkoterapie van huisdiere en doen hy aktiewe navorsing op hierdie gebied en is hy die dryfkrag agter die uiters belangrike nuwe studieveld en stigtingsvoorsitter van die Studiegroep van Mens/Dier-kontak, 'n groep wat studie van ons verhouding met troeteldiere ten doel het.

Oor die afgelope 7 jaar het hierdie praktisyn nie minder as 24 publikasies die lig laat sien nie – voorwaar 'n formidabele voorbeeld vir sy kollegas.

Hy voldoen voorwaar aan al die vereistes vir hierdie toekenning nl. om deur sy besondere voorbeeld die beeld van die professie te bevorder het.

HONORARY MEMBERSHIP OF THE S.A.V.A.

DR. W.F.O. MARASAS

At the 79th Annual General Meeting of the Association, Dr Walli Marasas was elected as an Honorary Member.

He obtained his BS.c.(Agric) in 1962 and MSc(Agric) in 1965 at the University of Pretoria and his Ph.D. at the University of Wisconsin in 1969.

From 1969 to 1975 he was a Mycologist at the Plant Protection Research Institute, Department of Agriculture and during this period he was closely associated with many veterinary research projects and investigations. Since 1975 he is Senior Chief Research Officer at the National Research Institute for Nutritional Diseases of the South African Medical Research Council.

Throughout his career he has directly or indirectly been involved in the investigation and diagnosis of all the known South African veterinary mycotoxicoses. This interest has been sustained despite the fact that he joined the Medical Research Council in 1975.

Over the years he has unfailingly supported the veterinary profession both nationally and internationally on the subject of veterinary mycotoxicology. At a more practical level he has contributed actively to pathology workshops and has always been more than willing to collaborate with our profession in investigating field problems.

He is an undisputed world authority and his international reputation is such that he is regarded as one of the major international figures in the field of veterinary and human mycotoxicology.

His Honorary Membership enhances the reputation of our Association.

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Vetgedrukte bladsy nommer dui enigste of senior outeur aan