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TYDSKRIF VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING

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TYDSKRIF VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING

Die TYDSKRIF is die offisiële mondstuk en eiendom en word gepubliseer deur die Suid-Afrikaanse Veterinêre Vereniging. Dit verskyn kwartaalliks en word aan sake van algemene veeartsenykundige belang gewy. Bydraers tot hierdie Tydskrif maak hul stellings en lug hul menings slegs op eie verantwoordelikheid; sodanige stellings word nie noodwendig deur die Redaksiekomitee onderskryf nie en die menings gee nie noodwendig die Komitee se menings weer nie. Kopiereg word op al die letterkundige inhoud van die Tydskrif voorbehou.

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DAMESVEEARTSE: 'N OPVOLGSTUDIE

J. NICOL*

ABSTRACT: Nicol J. **Female veterinary surgeons: a follow-up study.** *Journal of the South African Veterinary Association* (1983) 54 No. 1, 3-10 (Afrik) Counselling Section, Bureau for Student Services, University of Pretoria, Brooklyn, 0002 Pretoria, Republic of South Africa.

Questionaires were sent out to 68 registered female veterinary surgeons in South Africa to obtain career information essential to vocational guidance.

The 46 respondents represent 51,7 % of the 89 female BVSc graduates up to 1981; their average age was 33,3 years, 63 % were married, 30,4 % were from the Pretoria area, 60,9 % were living in the Transvaal, 52,2 % had had 6 and more years of veterinary experience. All the comparisons done indicated that the response group was representative of the women who had obtained the BVSc degree from the University of Pretoria.

Some of the more important findings were the following:

1. No less than 97,8 % of the response group are at present actively engaged in veterinary work – 73,9 % on a full-time basis.
2. Asked what they would choose now, 84,8 % of the respondents declared that they would choose veterinary science once again; 67,4 % would like to see their daughters enter the profession.
3. The most important reason for this positive attraction towards veterinary practice was given as "job satisfaction experienced".
4. Respondents indicated the things that they liked about veterinary practice: factors intrinsic to the job like surgery, pathology and research topped the list followed by the opportunity to work with and help animals and also the variety experienced in their work. The most important grievances were firstly the general attitude and actions of the public – callousness of animal-owners and inconsiderate behaviour towards practitioners. Long and irregular hours, administrative red tape and insufficient technical help were also complaints.
5. A general feeling was that the ratio between duration of study and income, as well as between hours worked and income, was unsatisfactory.
6. On a more popular level it was found that 50 % of the respondents had children and that 41 % of the married respondents were married to veterinary surgeons. Hobbies of a biological nature and sports involving horses were the most popular.
7. Asked whether there was any information they would like to pass on to prospective female veterinary surgeons, a number of themes emerged: A positive theme encouraging enrollment by interested girls; the warning that an interest in science is more important than merely the love of animals; the recommendation that gaining experience through holiday work with a practitioner is of utmost importance; a warning that the course is extremely demanding and requires great commitment; also that the BVSc qualification does not guarantee a very high income and, lastly, a totally negative theme whereby enrollment was strongly discouraged.

The findings of the study are extremely useful for prospective students and for persons involved in vocational guidance.

Key words: Female veterinary surgeons.

1. INLEIDING EN DOELSTELLINGS

Talle voornemende damestudente het 'n behoefte om buitelugwerk te doen, om met diere te werk en om by navorsing in die biologiese wetenskappe betrokke te wees. Een van die graadkursusse wat hulle dan oorweeg om toegang tot poste te kry wat aan hierdie vereistes voldoen, is BVSc. Aangesien veeartsenykunde egter nie tradisioneel 'n damesberoep is nie, bestaan daar by die voornemende studente heelwat vrae oor die kursus en oor die beroep self soos byvoorbeeld "sal ek die praktiese gedeelte van die kursus kan doen?", "is daar diskriminasie teen dames wat praktiseer?", "is dit die moeite werd om so 'n lang kursus aan te pak?", ensovoorts.

Een van die belangrike take van die Afdeling Konsultasie van die Studentediensburo aan die Universiteit van Pretoria is om beroepsvoorligting aan studente te verskaf. Om hierdie voorligting effektief te kan doen is die afdeling van beroepsinligting afhanklik wat op sy beste verkry kan word van persone wat binne bepaalde beroepsvelde staan.

Die verkryging van hierdie inligting is die primêre doelstelling van die opvolgstudie.

'n Sekondêre doelstelling is om meer te wete te kom oor die breëre kenmerke, beroeps- en lewensaktiwiteite van die byna 100 dames wat reeds die BVSc-graad verwerf het.

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2. METODE VAN ONDERSOEK

Die Registrateur van Veeartse kon die name van 68 damesveeartse verskaf. Aan hierdie dames is vraelyste in 1981 gestuur met 'n begeleidende brief waarin hulle gevra is om dit in te vul en terug te stuur. Die vertroulike aard van die ondersoek is beklemtoon en die versekering is gegee dat niemand se identiteit aan haar antwoorde gekoppel kon word nie.

3. BESKRYWING VAN DIE RESPONDENTEGROEP

Van die 68 vraelyste versend is 46 voltooides terugontvang. Hierdie terugsendingspersentasie van 67,6 % word onder die gegewe omstandighede as bevredigend beskou.

3.1 Biografiese besonderhede van die respondentegroep:

3.1.1 Ouderdom:

- Die gemiddelde ouderdom was 33,3 jaar, die mediaan 30,4 jaar en die modus 27 jaar.
- Die oorgrote meerderheid van die respondente val in die groep 26 tot 30 jaar oud (39,1 %).
- Van die totale groep is 71,7 % jonger as 36 jaar.

3.1.2 Huwelikstaat:

- Getroud, 29 (63,0 %)
- Nooit getroud, 15 (32,6 %)
- Geskei, 1 (2,2 %)
- Weduwee, 1 (2,2 %)

3.1.3 Die respondentegroep en groep aan wie vraelyste versend is volgens woonstreek.

In Tabel 1 word nie alleenlik die woonstreekverspreiding van die respondentegroep aangetoon nie maar ook die verspreiding van die adresse wat verkry is en waarheen vraelyste versend is. Die doel van hierdie gegewens is om te kyk hoe goed die verskillende streke verteenwoordig is. (Die streke is ingedeel aan die hand van die poskodestreke van die Departement Pos- en Kommunikasiewese).

- Die terugsending van vraelyste was vanuit al vier die provinsies, Suidwes Afrika en die buiteland bevredigend (50 % en meer is teruggestuur).
- Dit is opvallend dat 30,4 % van die respondentegroep in die Pretoria streek woonagtig is en dat 60,9 % van die totale respondentegroep in die Transvaal woon.

3.2 Enkele akademiese gegewens oor die respondentegroep:

3.2.1 Kwalifikasies:

Baccalaureus

- Uit die aard van die saak beskik al 46 van die dames oor die BVSc-graad.
- 7 dames (15,2 %) beskik ook oor 'n BSc-graad.
- Een dame het ook BSc Agric.
- Een verdere dame het ook 'n BA-graad.

Honneurs

- 3 van die dames het BVSc (Hons)
- Een het 'n BSc (Hons).
- Een het 'n BA (Hons).

Magister en Doktoraal

- Een van die dames in die groep besit 'n M Med Vet graad en 'n MS (uitgereik in die Verenigde State van Amerika) en een verdere dame besit die DVSc.

Ander

- Een van die respondente beskik oor 'n nagraadse diploma.

3.2.2 Jaar waarin die BVSc-graad behaal is: 'n vergelyking tussen die getalle BVSc-grade per jaar aan dames toegeken en die respondente se jaar van graduasie (Tabel 2).

- Dit is eerstens opvallend (en verblydend) dat meer as die helfte van alle dames aan wie BVSc-grade nog deur U.P. toegeken is, in die respondentegroep opgeneem is. (46 uit 89 = 51,7 %).
- Die jare tot 1970 is heelwat beter verteenwoordig as daarna – uit die 23 wat tot en met 1970 gegradueer het, is 16 in die respondentegroep opgeneem en dit is 69,6 %.

Tabel 1. WOONSTREEKVERSPREIDING VAN VERSENDINGS EN RESPONDENTEGROEP

Woonstreek	Vraelyste versend	Vraelyste terugontvang	% terug-ontvang	%van respon-dentegroep
Pretoria	18	14		30,4
Noord Transvaal	2	1		
Oos Transvaal	1	1		
Oos Rand	3	3		
Wes Rand	3	3		
Suid Transvaal	1	1		
Johannesburg	9	4		
Hoëveld	1	1		
Suid Oos Transvaal	1	0		
	39	28	71,8	60,9
Oos Londen	1	1		
Mid 2 Kaap	1	1		
Port Elizabeth	1	1		
Mid 1 Kaap	1	0		
Mid Wes Kaap	1	1		
Karoo	2	2		
WP 4	1	1		
WP 3	2	0		
Kaapstad	2	0		
	12	7	58,3	15,2
Pietermaritzburg	2	1		
Durban	1	1		
Suidkus	1	0		
Natal Kaap	1	1		
	5	3	60,0	6,5
Bloemfontein	1	1		
Drakensberg	1	1		
Oranje Vrystaat	1	1		
	3	3	100,0	6,5
Suidwes Afrika	3	2	66,6	4,3
Buiteland	6	3	50,0	6,5
	68	46	67,6	100,0

Tabel 2. 'N VERGELYKING TUSSEN DIE GETALLE BVSc-GRADE PER JAAR AAN DAMES TOEGEKEN EN RESPONDENTE SE JAAR VAN GRADUASIE

	a	b	c	d
Jaartal	BVSc-grade aan dames toegeken	Respondente gegradueer	Persentasie (b) van (a)	Persentasie van respondentegroep
Tot 1960	9	6	66,7	13,0
1961	1	0		
1962	1	0		
1963	1	0		
1964	1	0		
1965	0			
1966	1	1		
1967	0			
1968	3	2		
1969	3	2		
1970	2	2		
1961-1970	14	10	71,4	21,7
1971	4	1		
1972	1	1		
1973	8	4		
1974	5	2		
1975	4	2		
1976	3	3		
1977	7	4		
1978	2	2		
1979	11	5		
1980	7	4		
1971-1980	52	28	53,8	60,9
1981	14	2		4,3
	89	46	51,7	100,0

Ooreenstemmende syfers vir 1971 en daarna is 66 grade aan dames toegeken waarvan 30 opgeneem (45,5 %).

- Oor die algemeen wil dit voorkom asof 'n redelik verteenwoordigende respondentegroep bereik is in terme van die verspreiding van hul jare van graduasie.
- Die besonder lae persentasie vir die 1981 klas is waarskynlik daaraan te wyte dat hul adresse nog nie ten tye van die versending van vraelyste in die Registrateur van Veeartse se besit was nie.

3.2.3 Verdere studies

Agt van die dames in die respondetegroep (17,4 %) is tans vir verdere studies ingeskryf. Drie van hierdie 8 vir BVSc (Hons), 2 vir MMedVet en 3 vir ander kwalifikasies buite die veeartsenykundige rigting.

4. DIE BVSc-KURSUS

Enkele gegewens met betrekking tot die respondentegroep:

4.1 Ouderdom by aanvang van die BVSc studie

- 71,7 % van die dames was 19 jaar en jonger by inskrywing vir die BVSc-kursus. Die gemiddelde ouderdom by inskrywing was 19,2 jaar, die mediaanouderdom 18,1 jaar, die modus 18 jaar.

4.2 Inskrywing vir BVSc direk ná voltooiing van matriek al dan nie.

- 32 van die dames (69,6 %) het direk ná voltooiing van matriek vir die BVSc-kursus ingeskryf.

4.3 Redes waarom nie direk na matriek vir BVSc ingeskryf is nie:

Veertien van die dames het nie direk na matriek reeds vir BVSc ingeskryf nie.

- Helfte van hulle (7) omdat hulle nie gekeur is nie en toe eers vir iets anders ingeskryf het.
- 2 van hulle het eers gaan werk om geld vir die studie bymekaar te maak.
- 2 was direk ná matriek nog nie lus vir BVSc nie.
- Die oorblywende 3 het om ander redes nie direk ingeskryf nie (American Field Service bygewoon ens.).

4.4 Keuring: "Wat sou u gedoen het as u nie vir BVSc gekeur is nie?"

Op die vraag hierbo antwoord 60,9 % van die respondente dat hulle weer sou probeer om keuring te kry. Dit beteken dat hulle vir 'n ander kursus (bv BSc) sou inskryf om sodoende verdere kredietpunte en 'n beter kans op keuring te kry.

Sewe en dertig persent antwoord dat hulle van veeartsenykunde sou afsien en vir iets anders sou inskryf.

Twee persent het die vraag nie beantwoord nie.

4.5 Alternatiewe kursusse: "Indien u as gevolg daarvan dat u nie gekeur is nie van BVSc sou afsien: waarvoor sou u dan ingeskryf het?"

Vir 'n kursus in Wis- en Natuurkunde:	4 van die 17 (23,5 %)
Vir geneeskunde (MBCh B):	7 van die 17 (41,2 %)
Vir 'n ander natuurwetenskaplike rigting:	2 van die 17 (11,8 %)

Vir 'n kursus in geestes- en handelswetenskappe: 4 van die 17 (23,5 %)

4.6 Oordeel van die respondente aangaande die duur van die BVSc-kursus:

- Die kursusduur is te lank, 2 (4,3 %)
- Die kursusduur is te kort, 13 (28,3 %)
- Die kursusduur is net reg, 29 (63,0 %)
- Onbeantwoord, 2 (4,3 %)

4.7 Alternatiewe kursusse: "Het u naas BVSc enige ander kursusse redelik ernstig oorweeg?"

Hier antwoord 47,8 % van die respondentegroep dat hulle wel ander kursusse ernstig oorweeg het; 52,2 % rapporteer dat hulle eintlik net aan BVSc gedink het en nie ernstig aan enige ander kursusse nie.

4.8 Alternatiewe kursusse: "Indien daar sterk alternatiewe kursusse was, wat was hulle?"

Die 22 dames wat wel ander kursusse naas BVSc sterk oorweeg het, het aan die volgende gedink:

BSc	4 van die 22 (18,2 %)
Geneeskunde	10 van die 22 (45,5 %)
Landbou	2 van die 22 (9,1 %)
Geesteswetenskappe	6 van die 22 (22,3 %)

Uit 4.5 en 4.8 kom die feit na vore dat heelparty damesveeartse sterk tot geneeskunde aangetrokke was.

5. BEROEPSGEGEWENS VAN DIE RESPONDENTEGROEP

In hierdie afdeling word hoofsaaklik gekyk na die beroepsaktiwiteite en beroepsbelewenis van die groep.

5.1 Huidige beroep:

Tabel 3. DIE VERDELING VAN DIE RESPONDENTE VOLGENS HUIDIGE BEROEP

Beroep	Frekwensie	Persentasie
Algemene praktisyn	24	52,2
Staatsveearts	14	30,4
Dosent (Onderstepoort)	5	10,9
In diens van liefdadigheids-organisasies	2	4,3
Huisvrou	1	2,2
	46	100,0

- Die mees opvallende feit hieruit is dat so baie van die respondente aktief in beroepe staan – slegs 1 huisvrou werk tans nie in die breë veeartsenykundige veld nie.
- Die feit dat die adreslys van die Registrateur van Veeartse bekom is mag veroorsaak het dat die persone wat tans nie aktief is in veeartsenykunde nie, se adresse nie bekom is nie, en dat vraelyste dus nie aan hulle gestuur is nie.

5.2 Beroepsbeoefening voltyds of deeltyds:

- Voltyds: 34 dames (73,9 %)
- Deeltyds: 11 dames (24,0 %)
- Nie van toepassing: 1 dame (2,2 %)

5.3 Die aantal ure per week wat respondente aan hul veeartsenykundige werk bestee:

- Uit die 46 lede van die respondentegroep bestee 25 (54,3 %) meer as 40 uur per week aan hul werk. Nege en dertig komma een persent bestee 50 en meer ure terwyl 8,7 % meer as 61 ure bestee.

Tabel 4. VERDELING VAN RESPONDENTE VOLGENS URE PER WEEK GEWERK

Ure	Frekwensie	Persentasie
0 – 15	6	13,0
16 – 30	8	17,4
31 – 45	12	26,1
46 – 60	16	34,8
61 +	4	8,7
	46	100,0

5.4 Beroepservaring:

- Die respondentegroep is redelik jonk (71,7 % jonger as 36 jaar). As gevolg hiervan het 47,8 % minder as 5 jaar beroepservaring.

5.5 Toekomsplanne met betrekking tot beroep:

- Die oorgrote meerderheid (89,1 %) is van plan om met die beoefening van veeartsenykunde voort te gaan.
- Slegs 4,3 % beoog om die professie te verlaat terwyl 6,5 % onseker is oor hul toekomsplanne.
- Wat privaatpraktyk betref, beoog 60,9 % om hul professie so te beoefen terwyl 28,2 % van plan is om vir die staat te werk.

5.6 Beroepsevaluering:

In die volgende vrae is gepoog om vas te stel hoe die respondente tans oor veeartsenykunde as beroep voel en om sodoende meer oor die subjektiewe faktore uit te vind.

5.6.1 Kursuskeuse (self)

Die volgende vraag is gestel: "Indien u nou weer sou kon kies: sou u weer vir BVSc ingeskryf het?"

Die beantwoording was as volg:

- 39 van die respondente (84,8 %) sê ja, hulle sou nou weer vir BVSc inskryf.
- 6 van die respondente (13,0 %) sê nee.
- Een respondent is onseker en beantwoord nie die vraag nie.

Alhoewel verskeie faktore kontaminerend op hierdie verspreiding mag inwerk – dit is sekerlik vir niemand maklik om hier "nee" te antwoord nie – kan tog aanvaar word dat minstens 84 % van die respondente voel dat hulle wel die regte kursus- en beroepskeuse gedoen het en in die veeartsenykundige beroep gelukkiger is as in 'n ander beroep.

5.6.2 Kursuskeuse (dogter)

Om verder te bepaal hoe respondente veeartsenykunde as beroep vir 'n dame evalueer is gevra: "Gestel u sou 'n dogter hê: sou u graag sien dat sy haar in veeartsenykunde bekwaam?"

- 18 van die respondente (39,1 %) sê onvoorwaardelik "Ja".
- 13 van hulle (28,3 %) sê ook "Ja" maar met voorwaarde (bv. "mits sy absoluut wil", ens.).
- Dit beteken dat altesaam 67,4 % wel "Ja" sê.
- 7 dames (15,2 %) sê definitief "Nee".
- Laastens ontwyk 8 dames (17,4 %& die vraag deur bv. te antwoord dat hulle nie die dogter sal beïnvloed nie ens.

By hierdie verspreiding mag weereens faktore wees wat die beeld ietwat kleur. Een daarvan is sekerlik die tydsgees van individuele vryheid van keuse wat dit beklemtoon dat ouers hul kinders moet toelaat om hul eie beroepskeuses te doen. Ons moet ook hier onthou dat 50 % van die respondentegroep nie kinders het nie en dus met 'n suiwer hipotetiese "dogter" se keuse werk.

5.6.3 Kursuskeuse: Rede waarom respondente weer vir BVSc sou inskryf.

Uit 5.5.1 het geblyk dat 84,8 % van die respondente weer vir BVSc sou inskryf (m.a.w. hulself weer vir veeartsenykunde sou bekwaam) indien hulle nou weer sou kon kies. Die volgende redes is hiervoor verstrek:

- 52,8% gee *werksbevrediging* as hoofrede aan. Hieronder val bepaalde aktiwiteite in die werk, hoofsaaklik intrinsieke werksfaktore.
- 38,9 % noem *belangstelling:* hulle stel in veeartsenykunde belang en in niks anders nie.
- 8,3 % noem *praktiese oorwegings* soos die moontlikheid om deeltyds te praktiseer, die goeie inkomste ens.

5.6.4 Kursuskeuse: Rede waarom respondente nie weer vir BVSc sou inskryf nie.

Uit 5.5.1 het geblyk dat 6 van die respondente (13,0 %) nie weer vir BVSc sou inskryf nie. Hul redes was die volgende:

- 2 sê die inkomste is te laag, die verband tussen die aantal jare van studie en die inkomste van veeartse is nie gunstig nie.
- 2 sê werksgeleenthede is te min: daar is te veel veeartse en te min werk.
- 2 sê die werksure is te lank en te ongereeld, veral 'n getroude dame met kinders kan dit nie bybring nie.

5.6.5 Indien nie weer BVSc: Watter ander kursus?

Die 6 respondente wat voel dat hulle nie weer vir BVSc sou inskryf nie beantwoord hierdie vraag as volg:

- 2 van hulle sê hulle sou vir geneeskunde ingeskryf het.
- 2 vir kursusse in wis- en natuurkunde.
- Een vir 'n graad in geestewetenskappe.
- Een vir 'n diploma in sekretariële werk.

5.6.6 Wat geniet u die meeste in u werk?

Hierdie vraag is gestel om te bepaal watter beroepsaktiwiteite die dames voorkeur aan gee.

Die response was as volg:

- Intrinsieke faktore (43,5 %). Dit omsluit die beoefening van die veeartsenykundige wetenskap self met aktiwiteite soos sjirurgie, patologie, navorsing, ens.
- Die werk met, hulp aan en kontak met diere (19,6 %).
- Die afwisselende aard van die werk (17,41 %).
- Die kontak met mense (10,9 %).
- Die groot mate van outonomie (4,3 %).
- Die oorblywende 4,3 % het hierdie vraag nie beantwoord nie.

5.6.7 Wat hinder u die meeste in u werk?

Die griewe van die respondente kan in die volgende kategorieë verdeel word:

- Die optrede en houding van mense (30,4 %). Hier is hoofsaaklik na die onbedagsaamheid van diereeienaars en na die optrede teenoor veeartse deur die algemene publiek verwys.
- Die lang en ongereelde ure (24,0 %).
- Administratiewe rompslomp en onnodige voorskrifte wat nagekom moet word (8,7 %).
- Die gebrek aan tegniese hulp (6,5 %).
- Beperkte inkomste en beperkte werksgeleenthede (6,5 %).
- Gebrek aan ervaring (4,3 %).
- Ander dinge wat hinder (8,7 %).

5.6.8 Werkstevredenheid:

Vervolgens het die respondente uit ses stellings aangaande werkstevredenheid gekies. Die stellings en verspreiding van response was as volg:

	Frekwensie	Persentasie
1. Ek is volkome tevrede met my werk en stel in geen ander werk belang nie.	12	26,1
2. Hoewel enkele kleinighede my pla, is ek oor die algemeen tevrede.	27	58,7
3. Heelwat dinge pla my, maar dit wat my tevrede stel weeg effens swaarder.	7	15,2
4. Die dinge wat my ontevrede stel weeg effens swaarder as dit waarmee ek tevrede is.	0	0,0
5. Hoewel daar enkele dinge is waarmee ek tevrede is, voel ek oor die algemeen tevrede.	0	0,0
6. Ek meen dat ek in enige ander werk meer tevrede sal kan wees.	0	0,0
	46	100,0

Almal se response op hierdie vraag val dus aan die "positiewe" kant van die sespunt skaal. Altesaam 84,8 % val in die eerste twee stellings wat 'n hoë mate van werkstevredenheid aandui.

5.6.9 Werkstimulasie:

Op 'n vraag om te bepaal hoe stimulerend respondente hul werk vind, was die verspreiding as volg:

	Frekwensie	Persentasie
Baie stimulerend	12	26,1
Redelik stimulerend	33	71,8
Glad nie stimulerend	1	2,2
	46	100,0

5.7 Inkomste uit beroepsbeoefening:

Die verspreiding van respondente in terme van totale jaarlikse inkomste was as volg (Tabel 5):

Tabel 5. TOTALE JAARLIKSE INKOMSTE VAN RESPONDENTE

Inkomste	Frekwensie	Persentasie
R 0 (Werk nie tans nie)	1	2,2
R 1 – R 5 000	3	6,5
R 5 001 – R10 000	8	17,4
R10 001 – R15 000	18	39,1
R15 001 – R20 000	3	6,5
R20 001 – R25 000	7	15,2
R25 001 – R30 000	3	6,5
R30 001 – R35 000	1	2,2
R35 001 – R40 000	0	0,0
bo R40 000	2	4,3
	46	100,0

Wanneer die een respondent wat tans nie werk nie buite rekening gelaat word, dan blyk dit dat 29 uit die oorblywende 45 respondente minder as R15 000 per jaar inkomste het (64,4 %). Elf respondente werk deeltyds. Indien hulle ook weggelaat word, dan bly daar 18 uit 34 oor wat minder as R15 000 verdien (52,9 %).

5.8 Beskikbaarheid van deeltydse poste:

Hier rapporteer die respondente as volg:

Deeltydse poste vryelik bekombaar	: 23,9 %
Deeltydse poste van tyd tot tyd bekombaar	:34,8 %
Deeltydse poste geweldig skaars	: 39,1 %
Onbeantwoord	: 2,2 %

6. ENKELE ALGEMENE GEGEWENS OOR DIE RESPONDENTEGROEP

6.1 Kinders en kindertal:

Presies 50 % van die respondente het kinders. In terme van getalle is die verspreiding as volg:

	Frekwensie	Persentasie
1 kind	4	8,7
2 kinders	8	17,4
3 kinders	7	15,2
4 kinders	3	6,5
5 kinders	1	2,2
Geen kinders	23	50,0
	46	100,0

Altesaam het die 46 respondente dus 58 kinders.

6.2 Die beroepe van getroude respondente se eggenote:

Volgens 3.1.2 is 29 van die respondente (63,0 %) getroud. Die beroepe waarin hul mans staan is as volg:

- Veearts, 12 (41,4 %).
- Ander natuurwetenskaplike beroepe, 7 (24,1 %).
- Geestes- en handelswetenskaplike beroepe, 4 (13,8 %).
- Ander, 6 (20,7 %).

Dit is opvallend dat meer as 'n kwart van die totale respondentegroep en byna 'n helfte wanneer net na die getroudes gekyk word, met veeartse getroud is.

Vir interessantheid kan genoem word dat die totale jaarlikse *inkomste* van die mans kortliks die volgende eienskappe vertoon het:

- Die modusinterval was R15 000 – R20 000.
- 65,5 % van die mans verdien tussen R10 000 en R25 000.

6.3 Die gebruik van huisbediendes:

Op 'n vraag of hulle van die dienste van huisbediendes gebruik maak, antwoord 76,1 % van die respondente positief. Dit blyk verder dat 60 % van hierdie groep voltydse en 40 % deeltydse bediendes gebruik.

6.4 Stokperdjies:

71,7 % van die respondentegroep beoefen die een of ander stokperdjie.

Stokperdjies wat met die teel, kweek, versameling en versorging van lewende dinge (diere en plante) te make het is die mees algemene (40 %). Hierna volg "kunstige" stokperdjies soos skilder, naaldwerk ens. (31,4 %).

Die gemiddelde tyd wat aan stokperdjies bestee word, is 6,5 uur per week.

6.5 Sportdeelname:

Uit die 46 respondente neem 30 (65,2 %) aan sport deel.

Bo aan die gewildheidsleer is perdesport (spring en ry) gevolg deur balspele soos tennis en muurbal.

Die gemiddelde tyd wat per week aan sport bestee word, is 4,6 uur.

7. ADVIES AAN VOORNEMENDE STUDENTE IN VEEARTSENYKUNDE

Aan die einde van die vraelys is die volgende vraag ingesit om verder die mening van respondente te kry aangaande hul beroep:

"Is daar enige advies wat u aan beroepsvoorligters (skool of universiteit) wil gee om aan voornemende studente in die veeartsenykundige rigting oor te dra?"

Uit die aard van die saak was elkeen se respons hier verskillend, maar sekere breë temas het tog by herhaling voorgekom. 'n Opsomming van die belangrikste temas en 'n aantal tipiese response volg hier.

7.1 Belangrikste temas:

Tema 1: Positief

Die algemene advies wat hier voorkom is baie positief van aard, dames word aangemoedig om tot veeartsenykunde toe te tree en daarvan verseker dat hulle dit net so goed as mans kan doen.

Tema 2: 'n Belangsteling in die wetenskap belangriker as 'n liefde vir diere

Hier word beklemtoon dat 'n liefde vir diere nie genoeg is nie en dat dames werklik in die wetenskap moet belangstel.

Tema 3: Ondervinding soos vakansiewerk by 'n veearts is noodsaaklik

Die klem val daarop dat dames deeglik bewus moet wees van wat werklik op hulle wag in veeartsenykunde en ontslae moet wees van enige "romantiese idees" aangaande die beroep.

Tema 4: Die kursus is veeleisend en vereis groot toewyding

Die boodskap hier kom daarop neer dat die BVSc-graad nie maklik behaal word nie, mens moet hard studeer en baie sterk gemotiveer wees.

Tema 5: Die kwalifikasie bied nie 'n baie groot inkomste nie

Hier word beklemtoon dat die verhouding tussen studiejare en inkomste enersyds en werksure en inkomste andersyds, nie baie gunstig is nie. Die raad word gegee dat dames ook na ander kursusse sal kyk wat beter betaal.

Tema 6: Negatief

Teenoorgesteld van die positiewe advies van die eerste tema word die voornemende studente hier geadviseer teen veeartsenykunde. Klem word op negatiewe aspekte gelê sluit in dat daar wel teen dames gediskrimineer

word, dat die beroep met gesinslewe bots en dat werksgeleentheid en bevorderingsmoontlikhede min is.

7.2 Tiplese response:

- Veeartsenykunde is 'n lewenswyse, nie net 'n studierigting nie.
- "They must find a good equilibrium between their academic and social life to succeed. Woman must not be put off by people saying its not a woman's job".
- Baie Geluk. Studeer hard, maar bestee altyd genoegsame tyd aan sport en rekreasie.
- Poog dwarsdeur die kursus om jou vroulikheid te behou en dit sal goeie vrugte afwerp.
- "Prospective students should be scientific in attitude. They should accept that veterinarians are unlikely to earn similar salaries to the medical profession because human and animal lives are valued differently and therefore the responsibilities of the medical profession differ from those of vets".
- "Warn them that the salary: hours worked ratio is rather unfavourable".
- 'n Goeie en bevredigende beroep, maar iemand met intellektuele en ander eienskappe nodig vir veearts moet werksgeleentheid en vergoeding vergelyk met ander beroepe bv tandarts, medies, aptekerswese. Onthou dat geen man sterk genoeg is om 'n wilde bul te hanteer nie. Daarom is daar middels en drukgange om ons te help. My kliënte beskou nie my diens as minderwaardig t.o.v. my manlike kollegas nie.
- "Yes – tell the student advisers to get their facts straight and not to dissuade girls from studying vet science. I was told girls were discriminated against (untrue). Many girls are dissuaded by student advisers ignorant of the true facts".
- Studente moet bereid wees om hard te werk. Na voltooiing van die kursus is daar *verskeie* rigtings waarin hy/sy kan "spesialiseer" en is dus *nie* op 'n doodloopstraat nie. Meisies hoef geensins agteruit te staan vir mans nie – fisiese krag tel nie.
- As 'n meisie werklik belangstel en redelik tot goed presteer in haar werk, sou ek veeartseny *sterk* aanbeveel.
- Daar is geen rede om op grond van geslag teen dames te diskrimineer nie. Akademies en prakties presteer hulle bo-gemiddeld. As 'n dogter wil en akademies kan, moet sy aangemoedig word om BVSc te neem.
- Ek dink dit is belangrik om die groot verskeidenheid werksmoontlikhede uit te wys.
- Dis nie net 'n sentimentele werk, maar prakties en daar is baie rigtings wat die BVSc-graad 'n vrou toelaat om in te gaan. Nie net kleindier privaatpraktyk bv diagnostiese werk en navorsing by institute en universiteite.
- Probeer soveel as moontlik praktiese ervaring kry gedurende studies.
- Raai aan dat studente 'n dag by 'n praktisyn deurbring om 'n idee te kry wat werk vir hulle sal inhou.
- 'n Blote liefde vir diere is nie genoeg motivering (hoewel nodig), eerder 'n liefde vir die wetenskap.
- "Be absolutely (100 %) sure that it is what you want to do. There is much more to being a vet than just having a love for animals".
- Liefde vir diere is nie genoeg nie. Laat hulle 'n vakansie by 'n veearts help. Beklemtoon belangstelling in mense as 'n vereiste.
- "Only if you really "burn" to study vet science apply for the course – it is not mentally difficult but just such a great deal to learn and I had to study very hard – six nights a week.
- *Moet* gemotiveer wees anders mors hulle hulle eie tyd en dié van die persone wat hulle moet oplei.
- As die toekomstige student oortuig is dat dit die regte beroep is, laat sy daarmee aangaan.
- Mens moet *baie* ure insit en mens moet regtig motivering hê.
- Dis 'n strawwe kursus – deursettingsvermoë nodig. Beklemtoning dat dit nie 'n "glamour" beroep is nie – behels soms vuil/onaangename werk.
- 'n Vrou moet nie ligtelik (enige) professie opneem nie. Eis absolute dedikasie, uithouvermoë en belangstelling.
- "Get rid of any romantic notions you may have of saving innocent animal lives, etc. Be prepared for long and irregular hours. Lack of appreciation and consideration from the public".
- "Unless they are completely sure that this is the career for them, they should not set out on it".
- Vir dames wat getroud is en kinders het is dit moeilik om familiepligte asook die beroep te behartig omdat daar geen deeltydse poste beskikbaar is nie. Ek voel óf die een óf die ander word dan verwaarloos.
- Vir iemand met 'n groot jong gesin is die beroep en ure baie onprakties en veeleisend. Alleenloper – goeie beroep.
- Sy moet sterk wees en hardwerkend, want daar is baie diskriminasie teen vrouens op hierdie gebied.
- Kinders dink dit is 'n maklike beroep – hulle besef nie hoe veeleisend (veral fisies) en gevaarlik die werk is nie. (Min kinders (voornemende veeartse) wat in my praktyk kom werk het, het met veeartsenykunde aangegaan na hulle gesien het wat werklik gebeur).
- Moenie te romanties voel t.o.v. groot diere nie. Meeste boere in die platteland vertrou 'n vroue veearts nie naastenby soveel soos 'n man nie (ek het dit self eers nie geglo nie).
- Hulle moet *baie* seker wees dat hulle dit wil doen. Jou kliënte en diere kom altyd voor jou man en kinders. Daar is nog baie mense wat teen vroulike veeartse is en bevorderingskanse is baie min.

8. SLOT EN AANBEVELINGS

Die studie het in sy doel geslaag. Die gewensde inligting oor beroepsbeoefening en beroepsbelewenis van dames met die BVSc kwalifikasie is bekom en kan aan voornemende studente oorgedra word.

Dit is duidelik dat die vroulike veeartse 'n bydrae tot die professie lewer en verder lyk dit asof die professie beslis oor ruimte vir belangstellende dames beskik.

'n Hele aantal stereotipiese opvattings bestaan oor dames in veeartsenykunde. Die opvolgstudie kon geen bewyse vind vir stellings dat dames ongelukkig is in veeartsenykunde nie, dat daar alleenlik op die platteland (of alleenlik in stedelike gebiede) vir hierdie dames werksgeleentheid is nie, dat die dames net klaar studeer en dan voltyds huisvroue word en so vir die professie verlore is of laastens dat hierdie dames nie trou nie. Inteendeel dui alle bevindings daarop dat hierdie studierigting wel vir die regte dame geskik is en aan haar die geleentheid tot volle selfverwesenliking bied.

In die lig van die noodsaaklikheid van omvattende beroepskennis kan universiteitsowerhede dit oorweeg

om aandag te gee aan die aanbevelings van die respondente dat voornemende studente vakansiewerk by 'n geregistreerde veearts behoort te doen voor inskrywing. Die insluiting van so 'n vereiste by die keuringsreglement kan alleenlik meer realistiese kursuskeuses en beroepsverwagtings tot gevolg hê.

ABSTRACT: Littlejohn, A. & Bowles, Felicity, 1982 **Studies on the physiopathology of chronic obstructive pulmonary disease in the horse. VI. The alveolar dead space.** *Onderstepoort Journal of Veterinary Research*, 49, 71-72 (1982).

The alveolar dead space (VDalv) as a percentage of tidal volume was calculated in horses by substituting the values for partial pressure of carbon dioxide in end-expiratory gas ($PE'CO_2$) and for partial pressure of carbon dioxide in arterial blood ($PaCO_2$) in the equation:

$$\% \text{ VDalv} = \frac{PaCO_2 - PE'CO_2}{PaCO_2} \times 100$$

The mean % VDalv of 12 chronic obstructive pulmonary disease (COPD) subjects was 3 times greater than that of 22 normal subjects. Since the large % VDalv of COPD subjects was due to an elevated $PaCO_2$, is was considered that maldistribution of ventilation was the principal cause of their increased % VDalv compared with that of clinically normal subjects.

ABSTRACT: Biggs, H.C. & Anthonissen, M., 1982 **The seasonal incidence of helminth parasites and of *Oestrus ovis* in Karakul sheep in the Kalahari region of South West Africa/Namibia.** *Onderstepoort Journal of Veterinary Research*, 49, 73-77 (1982).

The seasonal incidence of gastro-intestinal helminths and of *Oestrus ovis* was determined by slaughter of successive groups of 4 tracer lambs, each exposed on pasture for 33 days.

Haemonchus contortus was present from March 1979 to early July 1979, with a generally increasing percentage of 4th stage larvae in each successive month. A "spring rise" in the egg count was seen in flock sheep in October 1978. *Oesophagostomum columbianum* was recovered from tracers slaughtered in March 1979. Apart from December and March, *Moniezia* spp. were present from November 1978 to early July 1979.

Oestrus ovis was active from September 1978 to early June 1979, with peak larval burdens recorded from October to December and from April to early June.

ABSTRACT: Herr, S., Roux, D. & Pieterson, P.M., 1982. **The reproducibility of results in bovine brucellosis serology and their correlation with the isolation of *Brucella abortus*.** *Onderstepoort Journal of Veterinary Research*, 49, 79-83 (1982).

In both the complement fixation test (CFT) and the serum agglutination test (SAT) titres were reproducible for the most part within a twofold range. They seldom exceeded these limits and never a fourfold range. *Brucella abortus* was successfully isolated in 86% of serologically positive cases and evidence is presented to confirm the use of the 30 International Units/mℓ level in the CFT as being diagnostically significant. The SAT, when done in microtitration plates, is even more reproducible than when done in tubes. The incidence of infected animals aborting or calving down with negative titres was found to be low.

ABSTRACT: Horak, I.G., 1982 **Parasites of domestic and wild animals in South Africa. XV. The seasonal prevalence of ectoparasites on impala and cattle in the northern Transvaal.** *Onderstepoort Journal of Veterinary Research*, 49, 85-93 (1982).

The prevalence of ectoparasites on a total of 36 impala (*Aepyceros melampus*) slaughtered monthly from February 1975 to February 1976 and a total of 24 cattle slaughtered monthly from March 1976 to March 1977 in the Nylsvley Provincial Nature Reserve was determined. Six species of ixodid ticks were collected from the impala and these, in order of abundance, were: *Rhipicephalus evertsi evertsi*, *Rhipicephalus appendiculatus*, *Amblyomma hebraeum*, *Boophilus decoloratus*, *Ixodes cavipalpus* and *Hyalomma marginatum rufipes*. Only 340 (2,7%) of the 12 757 ticks collected from the impala were adult. The 4 species of lice present on the impala were, in order of abundance: *Damalinia aepycerus*, *Linognathus aepycerus*, *Damalinia elongata* and *Linognathus nevilli*. The cattle harboured 8 species of ixodid ticks. In order of ubandance, these were. *R. appendiculatus*, *R. evertsi evertsi*, *A. hebraeum*, *Hyalomma truncatum*, *H. marginatum rufipes*, *B. decoloratus*, *Rhipicephalus simus* and *I. cavipalpus*. A total of 14 186 ticks was collected from the cattle and of these 4 660 (32,9 %) were adults.

Clear seasonal prevalences could be determined for certain ticks only. Adult *A. hebraeum* reached peak numbers on cattle from November to March, adult *H. Marginatum rufipes* from December to February and adult *H. truncatum* during January and February. Larvae of *R. appendiculatus* reached peak numbers on cattle and impala from March or April to July, nymphae from June to October and adults from December to March. Peak numbers of larvae of *R. evertsi evertsi* were recovered from impala from May to July and nymphae during July, while adults were present on cattle throughout the survey period, with peaks being recorded during December and February.

ABSTRACT: Boomker, J., 1982. ***Cooperia acutispiculum* n. sp. (Nematoda: Trichostrongylidae) from the kudu, *Tragelaphus strepsiceros* (Pallas, 1766).** *Onderstepoort Journal of Veterinary Research*, 49, 95-97 (1982).

A new species of nematode, *Cooperia acutispiculum*, was collected from the small intestine of 3 out of 4 kudu *Tragelaphus strepsiceros* (Pallas, 1766) and 2 out of 3 grey duiker, *Sylvicapra grimmia* (Linnaeus, 1758), culled in the Kruger National Park, Transvaal.

The males of this species are unique amongst members of the genus *Cooperia* in that the spicules are acutely pointed and the lateral branches of the distal part of the dorsal ray are wavy in appearance. The females could not be identified because of simultaneous infection of the type host with *Cooperia neitzi* (Mönnig, 1932).

GOODWILL – FACT OR FICTION

J.W.E. ADAMS*

ABSTRACT: Adams J.W.E. **Goodwill – fact or fiction.** *Journal of the South African Veterinary Association* (1983) **54** No. 1, 11-13 (En) Durban Veterinary Clinic, 12 Currie Road, 4001 Durban, Republic of South Africa.

The validity of goodwill payments when partners enter or leave practice is doubtful. The present situation in veterinary private practice in the Republic is described and compared with other professions. This profession is urged to review its attitute.

Key words: Goodwill, veterinary practice.

INTRODUCTION

At some time or another, practising veterinarians are faced with the purchase or sale of a practice or a share thereof. This involves placing a cash value on the practice.

In broad terms:

Value = Assets less Liabilities.

For the purposes of this paper, the premises are not considered under assets. The cost of accommodating the business is represented by rent paid to landlords who may or may not be members of the practice.

The security of tenure of the practice and its location are very important. This relationship that premises has to goodwill will be examined later.

To return to price: If the price of the practice is set higher than Assets less Liabilities, that excess is called Goodwill.

Value = Assets less Liabilities, becomes

Asking price = Assets less Liabilities plus Goodwill.

Assets and liabilities can be determined by examining the audited balance sheet of the practice. Apart from a word of caution about accepting certain valuations, e.g. debtors (may be bad), stocks (may be old), these matters will not be discussed. Attention will be directed towards the relevance of goodwill.

Conventional wisdom has it that veterinary practices have goodwill. In a profitable going concern, goodwill would be greater than in an unprofitable poorly managed practice although the latter usually has greater potential for increasing profit.

To assess profitability, one examines the Income and Expenditure (Profit & Loss) Account. Here practice income (turnover) is stated, expenses deducted, and the balance if any, described as profit or excess of income over expenditure.

INCOME & EXPENDITURE ACCOUNT

Fees charged (Income or Turnover)	R000 00
Expenses	
Accountancy	
Bad Debts	
etc.	
. . . .	
Rent	
Veterinary assistant	
Less	R000 00
Principals' (Partners') Profit . .	R000 00
(Also called excess of income over expenditure.)	

Some adjustment to the arrangement of the above figures is necessary in order to make a meaningful assessment.

The professional assistant's salary should be moved out of expenses. This leaves an amount after the deduction of expenses from turnover, which is called "balance for distribution to veterinarians".

After the deduction of realistic salaries for the principals and assistant any balance is the true profit available for the owner(s).

i.e. Turnover
Less *Expenses*
Balance (for distribution to veterinarians)
Less Principal's salary
Less *Assistant's salary*
True Profit. (= Dividend).

The derivation of true profit is imperative if a prospective purchaser, who is usually the assistant, wishes to evaluate the return on his investment. In other words, would he be better off as a partner drawing a partners salary plus profits (if any), or would it be better to remain as an assistant and to invest the goodwill figure in some other investment opportunity. At least some of the money not spent on goodwill should be invested in a retirement annuity or pension fund. This is to provide the funds on retirement that might (or might not) otherwise have been realised by the sale of goodwill.

If the practice value is set at assets less liabilities, i.e. without goodwill, then becoming a partner is usually a worthwhile investment. If value includes a share of goodwill then critical evaluation may reveal an unattractive investment. It must always be borne in mind that profit is that amount remaining after realistic salaries have been paid to all working professional staff.

The generally accepted valuation of Goodwill in veterinary practice in R.S.A. is one year's profit. This is taken to mean "balance for distribution to professional staff" and not "true" profit. This figure may be the average of the 3 years immediately preceding the arrival of the assistant. In the case of a partner leaving one can use the last full year prior to departure. There are of course as many ways to compute goodwill as there are ways to operate ruptured cruciates. Whatever method is used though true profit is the crux of the matter and is often not taken into account.

After a satisfactory assistantship the parties may wish to have a more permanent relationship. The principal can share the administration, financing and overall responsibility in the practice and the assistant can enjoy increased income, permanence and pride of ownership.

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The formation of a partnership between principal and assistant achieves the following:

For the principal

1. His capital investment is reduced to 50 %, as is the so-called "risk" of business.
2. His former assistant's fortune is positively linked to the practice's success. If any motivation is necessary here it is.
3. Equal division of duties, time off and holidays.

For the assistant

1. He obtains part ownership of the practice by capital investment. Ownership suggests permanence and pride.
2. His income is linked to endeavour. The capitalist motivator again.
3. Equal division of duties, time off and holidays.

The advantages of partnership can be seen to be evenly divided between the principal and his former assistant. For every advantage one party may enjoy there is a reciprocal equal advantage for the other.

Over and above contributing his pro rata share of capital towards fixed and current assets (his share of value before goodwill), can additional or goodwill payment be justified?

The author believes only with difficulty, except if the new partner can expect a substantial increase in income (which itself infers under-payment when the person was employed as the assistant). It must be stressed that goodwill is not being discussed in the context of a "walk in walk out" situation such as the sale of a one-man practice to another. What is being considered here are goodwill payments made when a partner joins (or leaves) a practice.

Basic salary cannot be subject to a goodwill consideration. As an assistant or a partner a salary for working is a right. If it is not earned in one practice it may be earned in another or in some other avenue of veterinary endeavour.

This may raise the cry: "But what about all the years I spent building up the practice?" So what! He earned an income during that time or he would have quit. The practice has work for more than one veterinarian or an assistant would not have been employed. Who needs who?

How can goodwill payments exist today if they are without foundation?

The answer is that goodwill is an anachronism persisting only in the medical, dental, and veterinary professions. It is generally absent in financially enlightened professions and is rapidly losing favour amongst doctors and dentists.

Veterinarians must be alerted to the impending disappearance of goodwill from our profession and members must re-appraise their retirement provisions without it.

At best goodwill is subjected to many variables:

Type of practice. Small animal clients are quite changeable while equine clients are less so. Either way, in our free society clients go where they wish and cannot be relied upon to stay with a new partner.

Future developments. Roads, zoning, industrial or residential developments. Property size, high rise projects, etc.

Size of the practice. The bigger the practice the less the goodwill.

Continuity. Security of tenure. Are the premises leased or owned?

Competition. Other practices in the area and the likelihood of new arrivals.

6. Employment agreements. Will these effectively prevent partners or assistants opening in competition.
7. Artificial inflation of practice figures by long hours of work or outbreaks of disease.
8. Profitability. Ironically an unprofitable practice may have the greater potential for growth. This may also be true of a practice which charges low fees.
9. Market forces. Relative keenness of the assistant to become a partner and the practice's enthusiasm to have him.

If having considered the factors mentioned above the intending partner decides that goodwill does exist then he will agree to pay the existing partner(s). Often the payment is by way of overdraft guaranteed by the recipient.

Our new partner starts to make payments with money he does not have, upon which he must pay tax (as earned income) to partners in whose hands it is capital and is thus tax free.

At the time in his life when he may be saddled with a mortgage and have a wife suffering from that occupational hazard of marriage, pregnancy, the assistant decides to share what is left of his income. He shares it with his partner(s) for the privilege of doing his part of the practice workload, plus his share of after hours work and accepting part of the responsibility of the practice expenses as well.

What drives young veterinarians to accept this lemming-like fate casting themselves into the North Sea of debt?

And if the purchase of goodwill is such a questionable deal what other possibilities are open to young practitioners?

Firstly it must be asked what financial training a new graduate might have had. Has his professional course included such everyday essentials as practice management, tax, insurance, mortgages, marketing professional skills and so forth?

The author's recent enquiries revealed no such training, formal or informal. Veterinarians are unique in this deficiency. Other professions polled had at least some lectures on these subjects.

How then does a fledgling practitioner obtain his financial knowledge? Usually from the practitioner for whom he works; the very person(s) (NOTE WELL) who will ultimately sell or attempt to sell the new partner goodwill. This is analagous to Pavlov and his dogs. If the vendor of goodwill has done his job efficiently when he rings the bell the new partner will salivate the required amount of goodwill finance.

The senior practitioners are not entirely to blame either. Their knowledge too has been handed down by their seniors, so tradition and superstition have kept goodwill enshrined in veterinary folk-lore. Veterinarians have never enjoyed any basic financial instruction at university neither has such training featured in continuing education.

Having taken the position that goodwill doesn't exist except in "True Profit" as described earlier where does the saleable worth of a practice lie apart from the capital accounts (fixed and current assets)?

If one imagines moving a partner from his hospital or clinic and the practice continuing in the same premises does the business collapse? Observations are that it continues quite satisfactorily and may confound disbelievers by expanding, even in the face of competition

from a previous partner.

If this is so then goodwill attaches itself more to premises than persons. Providing clients receive empathy, consideration and minimal competence, the personal reputation of the veterinarian plays far less of a role than the profession likes to believe.

Security of tenure of the practice is paramount. Ideally the practice premises should be owned by the partners but in a separate company. There should be a formal agreement of lease which clearly links rental to an independent index so that inflation/escalation is taken care of automatically without argument. Also, rights and obligations about maintenance must be clearly stipulated.

In order to set a fair rental at the outset of a partnership the premises (assuming they are owned by practice members) should be valued by:

1. An accountant using a "return" basis.
2. A sworn appraiser on a "sale" basis.
3. A builder on a "replacement" basis.

The three are averaged and a suitable net return calculated. Expenses e.g. rates, company costs and maintenance are added back and this then is the opening rental. This figure is linked to say the Consumer Price Index or the official inflation rate and adjusted regularly.

The value of shares in the property company can be kept properly adjusted by regular valuation of the premises and incoming partners can take up or decline shares as they wish. Should shares not be taken up by incoming partners they do make a contribution to the premises via their share of rental.

It follows from the above that if there is any threat to the security of tenure of the practice, short or even long term, e.g. zoning regulations, landlord tenant disputes, political unrest or whatever, such a practice cannot pretend to have any value above its net assets.

What are the chances open to a veterinarian who is unwilling to pay for nebulous goodwill? Money is not a problem. A well known bank assures me that a veterinarian as a professional man can expect R25 000 more or less on request. This money is available to him to use as he sees fit although he would be cautioned by the bank against spending it on goodwill.

Given the ease of leasing this amount is more than enough for a reasonably well motivated young man to establish himself in his own practice. He has only to accept a lower income for a short period and he will have created his own business. His investments will have been for real tangible assets.

To decide the real value of something such as goodwill ask yourself: "For what price could my widow sell it?"

There certainly is such a thing as goodwill but its importance is far less than our profession currently believes. As mentioned earlier its value must be set in relation to True Profit, i.e. *after* the deduction of professional salaries. There is always the danger that if a practice is purchased and the seller barred from unreasonable competition that someone else will open opposite or around the corner from you. What price then the goodwill recently purchased?

There are many more veterinarians being graduated now than ever before. Whether those coming into private practice join existing practices or compete with them lies largely in the hands of the members of the profession already in practice. Think carefully of the advantages of having your assistant with you in practice rather than in the next town or suburb. There is strength in unity. Larger veterinary units can offer better services and facilities and members can enjoy regular leisure time albeit at the expense of some financial efficiency.

The money not invested in goodwill can be invested elsewhere to yield very good returns on retirement.

A goodwill policy of "nothing in and nothing out" will be far healthier for the profession and will also bring us into line with the other professions whose financial track records are better than ours.

To those members who have invested in goodwill sympathies must be conveyed but because they saw fit to pay these sums is hardly reason to perpetuate the myth.

The newcomers to private practice are urged to think deeply before paying for something one can not touch, eat and likely will not be able to resell.

To those in charge of veterinary education please reexamine curriculum. Is it right to continue producing financially cryptorchid veterinarians who have to rely on the questionable expertise of their senior colleagues in practice for the skill to exist in the economic hurly burly of today's and tomorrow's world?

ACKNOWLEDGEMENTS

My thanks to Mr A. Porter, Registrar of the Royal College of Veterinary Surgeons, who explained how the British Society of Practising Veterinary Surgeons have tackled the task of student education. This society has put together a practice management course which is given at the various veterinary schools. This ensures that a standard course is presented by the people most interested in the subject. The individual veterinary schools are thus relieved of the need to provide this education.

BOOK REVIEW — BOEKRESENSIE

MALARIA CONTROL AND NATIONAL HEALTH GOALS

REPORT of the 7th Asian Malaria Conference TRS 680, World Health Organization, 1982

This booklet of 68 pages is a report of the collective views of the participants of the 7th Asian Malaria Conference 1980. It starts with a general description of the world-wide situation with regard to malaria and then describes some of the main political and administrative problems encountered in countries in Asia. The spread of *Plasmodium falciparum* resistance to chloroquine in Asia is discussed as is the appearance of sulfonamide-pyrimethamine resistance along the Thailand-Kampuchean border. Recommendations are made for the control of malaria in order to achieve the WHO objective of health for all by the year 2000.

This booklet would mainly be of interest to persons, both technical and administrative, who are concerned with the control of malaria.

C.G. Stewart

PARTNERSHIP PROPHYLAXIS

J.W.E. ADAMS

ABSTRACT: Adams J.W.E. **Preservation of partnership in veterinary private practice.** *Journal of the South African Veterinary Association* (1983) 54 No. 1, 17-19 (En) Durban Veterinary Clinic, 12 Currie Road, 4001 Durban, Republic of South Africa.

The most common association of veterinarians in private practice is that of partnership. Some such relationships are not happy and may not endure. The concept of partnership is examined and suggestions are made which will minimise dissension.

Key words: Veterinary practice, partnership.

INTRODUCTION

For the purposes of this paper partnership is taken to mean the association of two or more veterinarians in private practice who share the expenses and income of their efforts EQUALLY.

In accounting terms this situation is shown in an Income and Expenditure (Profit and Loss) Account. All expenses pertaining to the practice are totalled, deducted from the fees charged, and the balance (Net Income or Profit) shared amongst the partners.

Unfortunately, straightforward as the foregoing may sound, there exists the potential for dispute. In practice many matters lend themselves to different interpretations and possible abuse. A partnership that begins under favourable portents may sooner or later degenerate.

The partnership may not necessarily dissolve but, having survived, the relationship of the parties may be altered unfavourably.

This paper will examine the common causes of dissent and list a number of guidelines to assist principals and assistants undergoing the transition to partnership.

Common sense and courtesy, fair-play, and above all an equal division of effort and reward motivate the principles postulated here.

It is vital to reduce all agreements to writing to provide a permanent reference. Memories fade and are fallible.

To fall back on the Guide to Professional Etiquette and expect the Veterinary Board to bail the parties out of a predicament is unrealistic. The Board may properly decline to do so. The written record provides the basis for any future discussion or dispute and it is recommended that all documents, e.g. contracts and formal letters be drawn up or at least approved by attorneys representing each party individually. Fair and impartial as our legal brothers are, they should not be asked to act for 2 masters. Each party should be represented by his own legal adviser.

ASSISTANTSHIP

To be as realistic as possible this paper will follow the progress of a veterinarian who joins an existing practice. His passage from assistant to partner will follow a well defined course that is charted contractually.

An advertisement similar to the following commonly appears in Vet News/Nuus.

"Assistant required in mixed practice. Salary *(stated)* per month. Transport provided. Partnership prospects after probationary period. Apply: Dr Old, etc."

After a successful interview Dr New agrees to join the practice. A letter from one party to the other should confirm the details. Dr Old would normally send such a letter to Dr New, but colleagues already in practice do not always understand this, so it may be necessary for Dr New to send the letter.

Letter of Appointment

At least the following should be specified:

1. Period.
2. Salary: Any incentive or bonus must be linked to gross turnover, never profit or nett. The latter are unworkable because they may be differently defined or even manipulated. In any event, one has usually to wait for year end accounts to determine profit and it is thus too remote in time to be of use.
3. Duties: Especially after hours, week-ends and public holidays.
4. Telephone: Provision, answering and private use.
5. Leave: Entitlement, or salary in lieu thereof.
6. Transport: Provided or rate per kilometer for use of own vehicle. (refer Automobile Association Tables).
7. Medical Aid: Whether provided or not; sick leave entitlement.
8. Practice bar (restriction): Must be reasonable in both duration and distance.
9. Breach.
10. Notice of termination.

Such a letter thus clearly defines what is agreed. If either party disagrees or queries a particular point, additional correspondence will record the progress to agreement.

The matters set down in the letter of appointment can be included in a formal principal/assistant agreement or the letter itself can serve as the contract.

PROGRESS TO PARTNERSHIP

At this point, Dr New is employed as professional assistant to Dr Old, but no mention has yet been made of the "partnership prospects" referred to in the advertisement.

It is not unrealistic to talk date and cost of partnership BEFORE the commencement of assistantship because these matters might well influence Dr New's decision whether or not to accept employment. The date when partnership is hoped to commence should be stated. This is not meant to bind the principal to offer partnership, it merely means that if partnership is desired by both parties there is a date proposed when this will begin. If for some reason partnership is no longer sought by either party, this should be advised as soon as possible, permitting each party to replan the future.

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COST OF PARTNERSHIP

The cost of partnership can be specified within quite narrow limits. The value of furniture and fittings, surgical instruments, drugs and vehicles can be derived from the latest accounts and the method for determining goodwill, if any, specified.

It is less easy to estimate a partner's potential income but an attempt should be made to permit Dr New to make a reasoned decision whether or not his prospects are satisfactory.

The failure to act decisively in regard to the date of a partnership offer and the terms thereof is most unfair. It is more likely to be a sin of omission rather than commission by practice principals, but it can be countered by Dr New requesting (quite legitimately) this information. If there is no prospect of partnership then Dr Old should make this clear at the outset.

PARTNERSHIP AGREEMENT

Assuming a satisfactory assistantship, the terms of a partnership agreement must next enjoy attention. Before doing so it must be stressed that the partners should share the expenses and the profit of their efforts equally, with common sense, courtesy and fair play foremost in mind.

Discussion is limited here to those matters that are frequently problematical. It is assumed that the practice's books will be brought to balance as at the day prior to the new partnership.

1. Capital Accounts: These must be kept as near equal as possible. Active steps must be taken to prevent inequality. This eliminates the need to raise an interest charge because all parties will have an equal stake in funding the business.

   Furniture, fittings, drugs, equipment, etc. are taken at book value. It can be argued that the depreciated value of say an X-ray machine may be unrealistically low but once one starts seeking separate valuations for individual items in order to cost them above or below book value, the whole exercise will degenerate. There are tax implications too. If the value of an item seems too low, it must be borne in mind that the practice will not only have enjoyed long term use but also depreciation through the accounts as well.

   The practice accounts are the legal financial record of the partnership and book value should therefore be the basis of all valuations.
2. Book debts: These must be realistically valued. If their value is in doubt the incoming partner can justifiably have them excluded from the new partnership. This would be particularly prudent if poor credit control had been exercised prior to partnership.
3. Interest charges: If monies are owed by one partner to the other(s) such outstanding amounts must attract interest at the prevailing overdraft rate. This is to ensure that it is not more profitable to owe your partner or practice than the bank.
4. Drawings/Salary: These should be kept equal. If one partner has to overdraw say to pay tax, the remaining partner(s) should draw an equal amount. This also keeps overdraft expenses equal. The concept of equal monthly salaries for each partner plus periodic dividends is preferable to a monthly draw of available funds. The latter method makes budgeting more difficult both in the business and at home.
5. Goodwill: If goodwill is relevant the formula for determining it must be stated clearly, related to the practice accounts, and capable of clear decisive application both at entry and exit of partners. A partner should not pay for goodwill created while he was an assistant.
6. Duties and responsibilities: Partners must devote their full time and attention to the practice unless otherwise agreed. Suretyship or guarantees must not be undertaken personally or in the name of the partnership unless authorised in writing. All extra-practice activities during practice time must be at the discretion of the other partner(s).
7. Rent: There should be a formal lease with the owner. It is wise not to include the premises as a practice asset because this causes much larger sums of money to be involved making the whole situation unwieldy. The premises are best owned by a separate company whose shareholders may well be partners of the practice. Rental escalations and maintenance obligations must be defined clearly.
8. Leave, after hours duties and time off: Strictly recorded and kept equal. Any time away from the practice by a partner must be matched by equivalent time off given to the other partner(s). Time away from the practice must count the same whether it is for veterinary committee work or for whatever outside activity a partner may choose. Outside activities should be limited to say 10 days per annum (so-called occasional leave) and thereafter time off would be regarded as part of annual leave. The remaining partner(s) must have absolute power of veto on outside activities.
9. Motor Vehicles: The vehicles should be owned privately by the partners and used in the practice. A fixed equal amount for running expenses should be paid. Repairs and maintenance can be charged through the practice but then added back to drawings. If one partner uses his car on practice business more than the others then a suitable adjustment must be made having in mind Automobile Association motoring costs.

Partner A		Partner B
Drawings		
(salary + dividend)		Drawings
+ Motoring costs		+ Motoring costs
+ Other perquisites		+ Other perquisites
TOTAL	Equal	TOTAL

   This leaves each partner free to choose his type of transport which he may abuse at no cost to his partner(s).

   Adding back motoring, telephone or any other practice necessities or perquisites offers tax advantages but does not penalize partners who absorb less in expenses.
10. Management: In a 2 man practice circumstances may arise when partners find themselves unable to agree on a course of action. An impasse must be capable of resolution and to do this a third voting member of the practice is necessary. This could be the practice lawyer, accountant or a mutually respected accessible colleague.

11. Dissolution: The precise mechanism must be spelled out. When one partner has to give notice to the other the remaining partner must be able to give reciprocal notice and not be stuck in a practice he was also contemplating leaving. The amounts, times of payment and interest rates must be agreed in advance, preferably in the original partnership agreement, so that what is often a very trying time is not made more difficult.
12. Additions and deletions: Additions and deletions to the partnership agreement can be made by properly minuted resolutions signed by the partners. This allows a flexibility to meet changing or unexpected circumstances.

OTHER POTENTIAL FLASHPOINTS

Before suggesting that wives should not assist as bookkeepers, receptionists, nurses, telephonists, full tribute must be paid to all veterinary spouses. Without their very often poorly or unpaid expert assistance, the majority of private practices would not function. However, when the time comes to afford an assistant or partner one must be able to afford to phase family out of the surgery, clinic or hospital. If the receptionist earns a reprimand, imagine the dilemma facing the new partner. Does he treat her as the boss's wife or as an employee?

Communication between partners is probably the crux of partnership prophylaxis. It enjoys least attention in practice but, because we all know how important it is, it will enjoy attention here. Regular weekly or monthly meetings are imperative to review management and professional matters. Keeping brief minutes of decisions is highly recommended.

Do not allow your professional relationships to degenerate to the furtive exchange of notes or messages via a third party. Meet face to face and say what is on your mind with due regard to your associate's feelings. A practice that talks together stays together.

DISCUSSION

The main thrust of this paper is equality in all things. Early on in a partnership wide latitude in give and take may be possible without engendering rancour. However, one permits inequality at one's peril. If any minor disagreement is allowed to become a major one, all the little irritations come tumbling out and bedevil the solution of the initial problem.

Successful veterinary practices exist even where the partners are not particularly friendly towards each other, provided that the principles of practice prophylaxis are observed. Practices, like our patients, do better on a regimen of prevention rather than cure.

ACKNOWLEDGEMENTS

My thanks go to the many private practitioners who fully and forthrightly discussed their partnerships with me. Their problems and the solutions thereto assisted me to form conclusions and to propose answers to some of partnerships' problms. For obvious reasons my colleagues' names are not mentioned.

BOOK REVIEW — BOEKRESENSIE

VETERINARY EPIDEMIOLOGY AND ECONOMICS

W.A. GEERING, R.T. ROE and L.A. CHAPMAN

Proceedings of the Second International Symposium on Veterinary Epidemiology and Economics
Published by Australian Government Publishing Service, Canberra 1980. pp XXV and 661 Figures 100 Tables 150 Price R19,80

By its nature, this collection of 83 papers is rather amorphous and varied both with regard to approach and quality. Few of the contributions can be considered as being of general epidemiological interest; their appeal is more likely to be determined by the field of interest of the reader. For this reason the publication cannot, as its title suggests, be considered definitive in the context of veterinary epidemiology or economics. Nevertheless because of its breadth of subject-matter this is a useful reference work.

The papers are divided into 8 categories, viz. information gathering, analysis and interpretation of data, definition of disease status of areas, education, evaluation of the production and economic effects of disease, the decision making process and planning and implementation of control programmes. Unfortunately, the most important category so far as epidemiology is concerned ie., analysis and interpretation of data, contains little to advance either insight or perspective and nothing inovative. This deficiency, however, merely reflects a general problem in veterinary epidemiology where (if one accepts the definition of H.V. Thursfield) the "why, how, when and where" are usually adequately covered but the "to what extent" is largely ignored.

Despite this criticism the book fulfills a major deficiency in the veterinary field, particularly in this country, by demonstrating the importance and wide applicability of epidemiological investigation and, as Dr R.S. Morris said in his closing address, refuting ". . . the dominant, but declining view, that no specific training is required (in epidemiology) since the requisite knowledge will be picked up in passing."

G.R. Thomson

FATAL SOFT TISSUE CALCIFICATION IN SUCKLING PUPPIES

ELIZABETH W. HOWERTH*

ABSTRACT: Howerth E.W. **Fatal soft tissue calcification in suckling puppies.** *Journal of the South African Veterinary Association* (1983) 54 No. 1, 21-24 (En) Pathology Section, Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa.

Renal, pulmonary and vascular calcification was observed in 2 suckling puppies. The lesions were similar to those associated with hypervitaminosis D and possibly hypervitaminosis A. It was suspected that high contents of vitamin D and A in the diet of the lactating bitch had elevated the activity of these vitamins in the milk to levels toxic for the suckling puppies.

Key words: Calcinosis, lactation, milk, vitamin D, vitamin A.

INTRODUCTION

Soft tissue calcification is clasically divided into dystrophic calcification, which is the deposition of calcium salts in degenerating tissues, and metastatic calcification, which is the precipitation of calcium salts as the result of persistent hypercalcaemia. In dogs, soft tissue calcification has been associated with a number of diseases that cause hyprecalcaemia. These include primary hyperparathyroidism[19], pseudohyperparathyroidism[12], chronic renal failure[3], and hypervitaminosis D[4 7 9 10 15].

This report describes soft tissue calcification in 2 suckling puppies which was apparently caused by high levels of vitamin D and, possibly, vitamin A in the diet of the lactating bitch.

CASE HISTORY

A breeder of Toy Pomeranians had 6 bitches whelp over a period of one month. Sixteen of the 24 puppies born from the 6 different litters died within a month of birth. Affected puppies stopped nursing and became lethargic at 2 – 3 weeks of age. Death occurred within 3 d. There were usually 1 or 2 survivors from each litter which were most often the weakest puppies at birth.

Postpartum bitches were fed commercial dry dog food which is used on a widespread basis in South Africa with apparently no deleterious effects. Their diet was supplemented with 1 teaspoon (approximately 5,0 g) of a vitamin/mineral compound (Calsuba Powder, Beecham), containing 90 IU vitamin D, 900 IU vitamin A, 667 mg calcium lactate and 167 mg calcium glycerophosphate. Bitches also received 1 cup (approximately 250 ml) of cow's milk daily. The bitches remained healthy throughout lactation.

MATERIALS AND METHODS

Two Pomeranian puppies (1 live and 1 dead) between 14 and 21 d of age were submitted for necropsy. Selected tissues were fixed in 10% buffered formalin and embedded in paraffin. Sections 5 – 7 μm were cut and stained with haematoxylin and eosin, von Kossa's, Alizarin Red S and Masson's trichrome.

PATHOLOGICAL RESULTS

In both puppies gross lesions were confined to the lungs. The lungs of the dead puppy failed to collapse and were dark purple and oedematous. The lungs of the live puppy were slightly emphysematous.

Histopathologically, multifocal mineral deposits were randomly distributed throughout the renal cortex but predominantly occurred at the corticomedullary junction in both puppies (Fig. 1). Calcium was deposited in the interstitium, along the tubular basement membranes, and sometimes in epithelial cells and lumens of tubules. Interstitial fibrosis was present in these areas and associated tubules were either atrophic or showed evidence of regeneration (Fig. 2). Langhans-type giant cells were seen in some of these foci (Fig. 2). Occasional tubules in the medulla had mineralized epithelial cells and contained calcified casts in the lumens. One kidney had a small number of necrotic glomeruli.

One puppy also had medial arterial calcification (Fig. 3) and pulmonary calcification (Fig. 4). In this puppy, there were multiple areas of elastic fibre mineralization in the wall of the aorta and pulmonary artery. Calcification of the lung was extensive and involved the walls of the bronchi and bronchioles and the alveolar septa. In addition to the calcification, the alveolar septa were often thickened by fibrous tissue. The alveolar spaces contained small amounts of pale, eosinophilic oedema fluid, small numbers of alveolar macrophages, and occasional Langhans-type giant cells (Fig. 4). Alveolar emphysema was also noted.

DISCUSSION

The D vitamins are sterols formed from provitamins derived from both plant and animal sources. Ergosterol, which is derived from plants, and 7-dehydrocholesterol, from animal sources, are converted to ergocalciferol (Vitamin D_2) and cholecalciferol (Vitamin D_3), respectively, by ultraviolet radiation. Vitamin D_2 and Vitamin D_3 are converted to their 25-hydroxyl derivatives (25-hydroxyvitamin D) in the liver[5]. The 25-hydroxyvitamin D constitutes the major fraction of circulating vitamin D but is normally relatively inactive[6]. In the kidney, 25-hydroxyvitamin D undergoes a second hydroxylation to form 1,25-dihydroxyvitamin D which is the biologically active form of the vitamin under normal circumstances.

Vitamin D plays an important role in calcium and phosphorus metabolism. The major target organ for 1,25-dihydroxyvitamin D is the mucosa of the small intestine where it increases the active transport of calcium and phosphorus. For mobilization of bone calcium 1,25-dihydroxyvitamin D is also required. In the kidney, active metabolites are thought to stimulate calcium and

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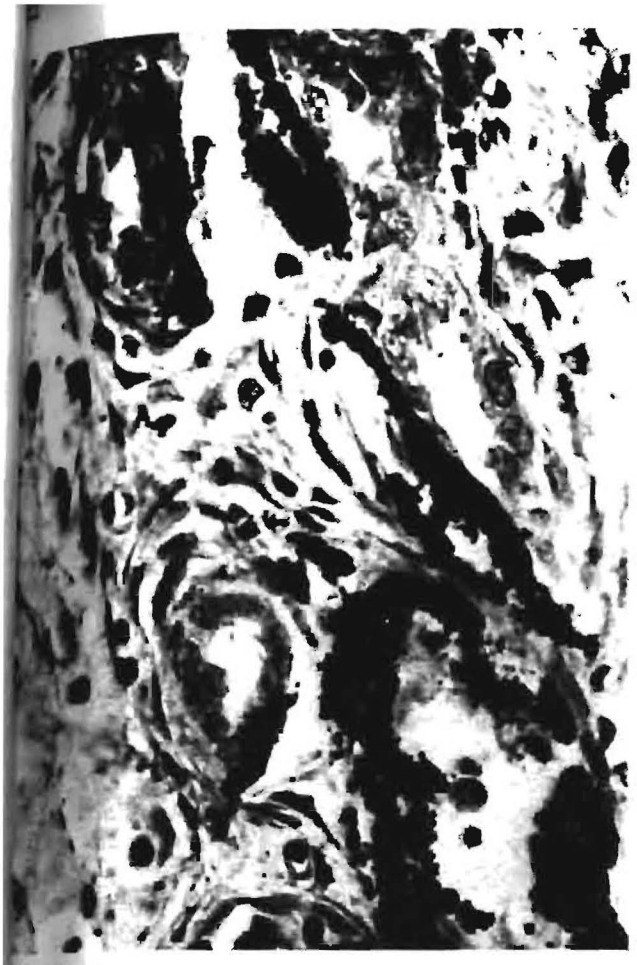

FIG. 1: Area in renal cortex with extensive calcium deposition. HE X 450

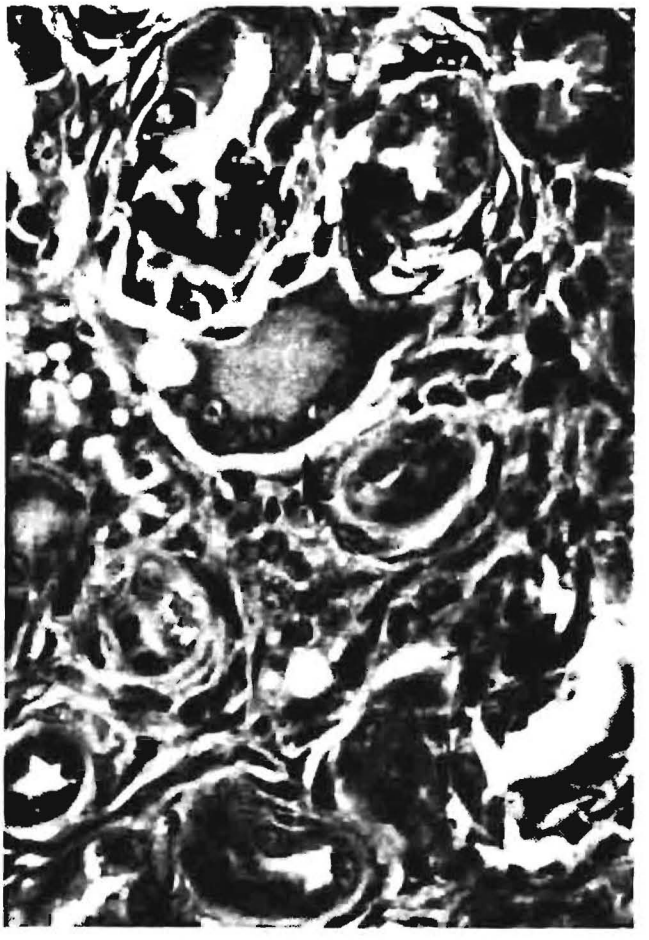

FIG. 2: Interstitial fibrosis and tubular regeneration in the renal cortex. Note Langhans-type giant cell (arrow) adjacent to a calcium deposit (c). HE X 450

phosphorus retention by increasing proximal tubular resorption[5].

The 25-hydroxyvitamin D can simulate 1,25-dihydroxyvitamin D at the intestinal receptor and is capable of mobilizing bone calcium when present in excessive amounts. Association of these facts with the relative absence of regulation over the production of 25-hydroxyvitamin D has led to the suggestion that 25-hydroxyvitamin D, rather than 1,25-dihydroxyvitamin D, is the causative agent in vitamin D intoxication. This hypothesis is supported by the observation that anephric patients, incapable of biosynthesizing 1,25-dihydroxyvitamin D, can become vitamin D-intoxicated[6].

In supraphysiological amounts, vitamin D causes hypercalcaemia and hyperphosphataemia[2]. Dogs given excessive quantities of vitamin D consistently show soft tissue calcification, including cardiovascular calcification, nephrocalcinosis[4 7 9 10 15], and pulmonary calcification[7 9 10 15]. The deposition of calcium in the soft tissues secondary to hypervitaminosis D is usually described as metastatic[14]. However, experimentally it has been shown that deposition of mineral in the kidney follows rather than precedes cell injury and should be considered dystrophic[13].

Although hypervitaminosis A reportedly causes hypercalcaemia by increasing bone resorption[20], there is conflicting evidence that this fat soluble vitamin is capable of eliciting soft tissue calcification. Experimentally, the administration of large doses of vitamin A to dogs has not resulted in soft tissue calcification[1 8]. Intraperitoneally administered vitamin A in rats induced renal-cardiovascular calcinosis similar to that caused by excessive vitamin D. Orally administered vitamin A did not induce renal-cardiovascular calcinosis[16]. Nephrocalcinosis has been reported in rabbits after excessive administration of Vitamin A intraperitoneally[17].

No data is available on the effect of diet on the calcium and vitamin D and A content of dog milk. Information from other species suggests that the calcium content of milk remains relatively constant except when the lactating female is subjected to multiple dietary deficiencies. On the other hand, the vitamin D and A activity in milk varies with the amounts present in the diet of the lactating female and feeding extra vitamin D or A will increase the content of the milk[18].

Suggested daily nutrient requirements of vitamin D and A for the lactating bitch are 22 IU/kg body weight and 220 IU/kg body weight, respectively, and most commercial dog foods in South Africa supply adequate amounts of these vitamins[11]. In addition to the vitamin D and A in the dog food, the bitches in this report were receiving approximately 73 IU vitamin D/bitch/d and 1025 IU vitamin A/bitch/d from the vitamin/mineral compound and the cows milk. Although these levels do not seem excessively high, the lesions in these 2 puppies are compatible with those observed in hyprevitaminosis D, and possibly hypervitaminosis A, suggesting that they were high enough to elevate the vitamin D and A activity of the milk to levels toxic for the suckling pup-

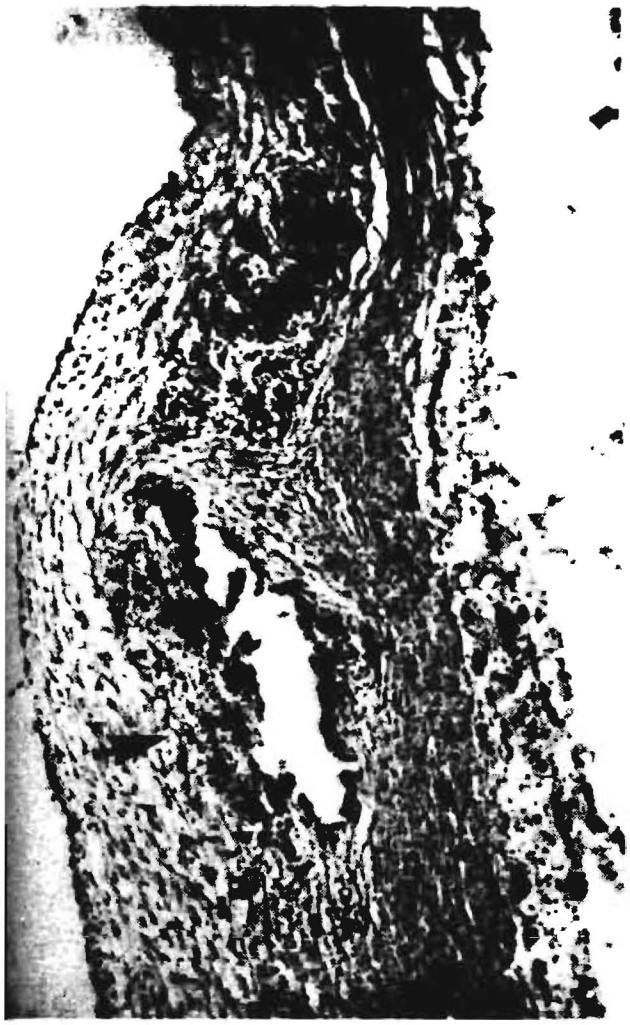

FIG. 3: Thoracic aorta with areas of mineralized elastic fibres (arrows). HE X 70

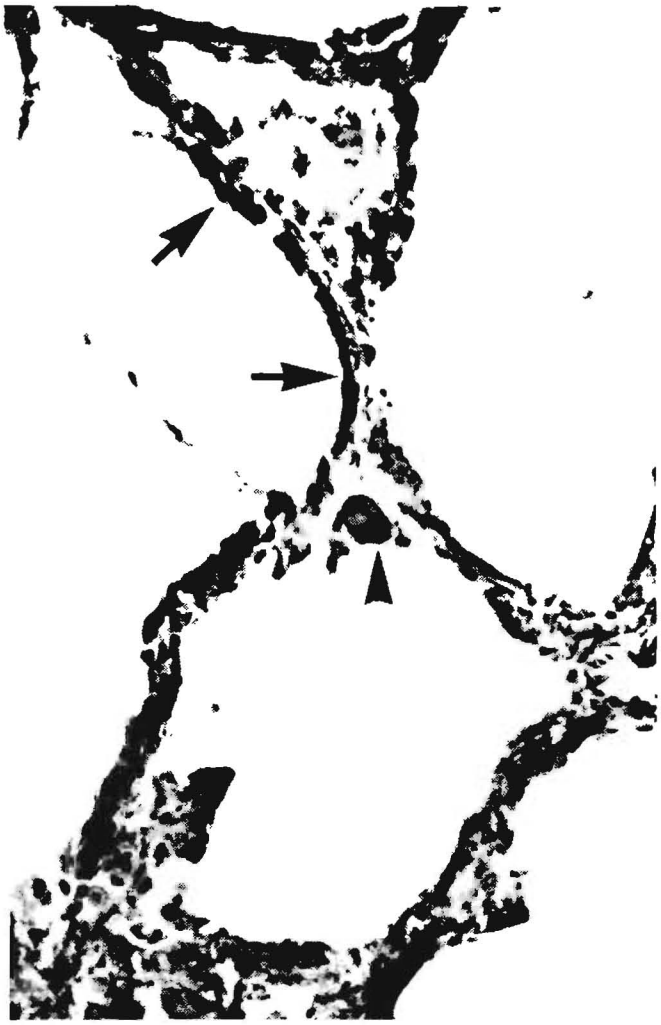

FIG. 4: Alveolar septa are thickened by calcification (arrows) and fibroplasia and alveolar spaces contain macrophages. Note Langhans-type giant cell (arrowhead). HE X 180

pies. In rats, consumption of a diet with a high content of vitamin D led to hypercalcaemia in the 12 and 17-day-old suckling offspring indicating that the vitamin D content of rat milk is influenced by diet and that suckling offspring can be affected by excessive vitamin D in their milk[18].

It is thought that the surviving puppies, identified by the breeder as the weaker puppies of the litters, probably consumed smaller quantities of milk and therefore, lesser amounts of vitamin D and A. Decalcification of the soft tissues takes place gradually during the recovery from hypervitaminosis D[9]. Although hypervitaminosis D in growing dogs can produce irreversible changes in the jaw and teeth, including malocclusion and pitting, irregular placing, and poor development of the teeth[7 9], the breeder claims that the surviving puppies developed into normal appearing adults.

During subsequent lactations, the breeder has not supplemented the diet of these bitches with any type of vitamin/mineral compound and has reduced the amount of cow milk offered by half. There have been no further problems with litters whelped by these bitches supporting the speculation that high levels of vitamin D, and possibly vitamin A, in the diet of the lactating bitches were responsible for the soft tissue calcification in these puppies. Experimental studies of hypervitaminosis D or A in suckling puppies have been neglected despite the widespread use of vitamin supplements in dogs. Further research is needed to define what levels of vitamin D and A in the diet of the lactating bitch can cause toxic levels of these vitamins in the milk.

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THE FREQUENCY AND SOME CHARACTERISTICS OF ANAEROBIC BACTERIA ISOLATED FROM VARIOUS FORMS OF BOVINE MASTITIS

A.S. GREEFF*, J.H. DU PREEZ** and MARIA DE BEER*

ABSTRACT: Greef A.S.; Du Preez J.H.; de Beer Maria. **The frequency and some characteristics of anaerobic bacteria isolated from various forms of bovine mastitis.** *Journal of the South African Veterinary Association* (1983) 53 No. 1, 25-28 (En), Department of Medical Microbiology, Faculty of Medicine, University of Pretoria, P.O. Box 2034, 0001 Pretoria, Republic of South Africa.

The prevalence of strictly anaerobic bacteria in the secretions from untreated cases of mastitis in lactating dairy cows was investigated. The study involved 147 Friesland cows in 12 highveld herds. All herds yielded cows with anaerobic udder infections. No anaerobic bacteria were recovered from cows with normal quarters or those with latent aerobic infections. Only anaerobes were present in 10 % of so-called 'aseptic' mastitis cases. A variety of anaerobic organisms was isolated concurrently with facultative bacteria from 5,3 % and 58,8 % of cases classified as subclinical and clinical respectively. *Peptococcus* spp. was associated with *Corynebacterium pyogenes* and *Bacteroides* spp. with *Staphylococcus aureus* and/or *Streptococcus agalactiae* in 80 % anaerobic udder infections. Gram positive anaerobic species were mostly sensitive to penicillin-G but all the Gram negative rods were resistant. In addition, all *B. fragilis* strains produced β-lactamase. The ability to produce heparinase was demonstrated in one strain of *Peptococcus indolicus* and a *Peptostreptococcus* sp.

Key words: Anaerobic bacteria, bovine mastitis, prevalence, antimicrobial sensitivity, heparinase production.

INTRODUCTION

Previous reports on the involvement of non-sporeforming anaerobic bacteria as causative agents of bovine mastitis were mostly concerned with *Peptococcus indolicus*[16 17 18 19]. Shinjo et al[15], however, isolated *Bacteroides* spp. and *Fusobacterium necrophorum* in addition to members of the Peptococcaceae from outbreaks of mastitis as well as the healthy udders of non-lactating heifers. Except for the communications by Greeff et al.[9] and Du Preez et al.[6] the literature is devoid of quantitative data on the prevalence of strict anaerobic bacteria in the secretions of quarters with bovine mastitis. Both of these studies reported on the isolation frequencies of a wide range of anaerobic species from such secretions in the lactating cow: occurring in 16,6 % of cases of subclinical mastitis and in 7,4 % of cases of clinical mastitis. The practical difficulties encountered in obtaining samples from untreated cows with clinical mastitis may have been responsible for the low isolation rate[6 9].

The purpose of the present study is to report on the rate of isolation of obligate anaerobic bacteria from mastitic cows in lactation which had not received recent antimicrobial treatment prior to sampling. We also report on the antimicrobial susceptibility profiles of anaerobic isolates to various commonly used antimicrobial agents.

MATERIALS AND METHODS

Animals

The experimental group of animals consisted of 147 lactating Friesland cows in 12 dairy herds on the Transvaal highveld. Udders were classified as either healthy or afflicted with one of the following: 'aseptic' (AS-) mastitis, latent udder infection, subclinical (SC-) mastitis or clinical (C-) mastitis (see Table 1). This was done by routine cytological and aerobic bacteriological methods according to the criteria of the International Dairy Federation[12].

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Anaerobic sampling

Sterile 10 ml disposable syringes equipped with 150 mm × 1,0 mm catheters were preflushed with an anaerobic gas-mixture consisting of N_2(80%): CO_2(10%): H_2(10%) on a volume basis. Such treatment removes all traces of atmospheric oxygen from the system. The teat canal orifices were disinfected before milk samples were aseptically aspirated from the gland cistern of the udder by insertion of the prepared catheters through the teat canal. The aspirated samples were immediately injected into 20 ml vaccine type bottles with crimped butyl rubber sealers. These transport bottles contained the anaerobic gas-mixture and a redox indicator[10] to ensure complete anaerobiosis. Bacteriological analysis of samples was started within 6 hours of sampling.

Bacteriology

The cultivation and identification of anaerobes and facultative organisms were done according to methods previously described[4 6 10].

Antimicrobial sensitivity

The broth-disc method of Wilkins & Thiel[20] was used to determine the susceptibility of anaerobes to penicillin-G(10 units/ml), chloramphenicol (12μg/ml), clindamycin (3,2μg/ml), metronidazole (6μg/ml) and cephalothin (6μg/ml). The chromogenic cephalosporin method of O'Callaghan et al.[13] was used to determine the presence of β-lactamase.

Heparinase production

This was determined by the method of Gesner & Jenkin[8].

RESULTS

Isolation frequency of anaerobic bacteria

It is evident from Table 1 that strict anaerobic bacteria were isolated from the quarters of cows in all 12 herds. Their absence from milk of healthy udders is, however, in sharp contrast with the varying isolation rates obtained from clinical mastitis (58,8 %),

Table 1: FREQUENCY OF ISOLATION OF ANAEROBIC BACTERIA FROM LACTATING UDDERS

Status of Experimental group	Number of Animals	Number of Quarters	Anaerobes present	Incidence of Anaerobes %
Herds	12	195	12	100,0
Healthy udders	30	44	0	0,0
Latent udder infection	15	26	0	0,0
C-mastitis	17	24	10	58,8
SC-mastitis	75	91	4	5,3
AS-mastitis	10	10	1	10,0
TOTAL	147	195	15	14,5

C-mastitis = clinical mastitis,
SC-mastitis = Subclinical mastitis,
AS-mastitis = 'aseptic' mastitis

Table 2: ANAEROBIC AND FACULTATIVE BACTERIA ISOLATED CONCURRENTLY FROM VARIOUS FORMS OF MASTITIS

Cow No	Herd No	Anaerobes	Facultatives
Clinical mastitis			
1	A	*Peptococcus saccharolyticus*	*C. pyogenes*
2	A	*Bacteroides melaninogenicus*	*S. aureus*
3	B	*Peptococcus indolicus**	*C. pyogenes*
		Eubacterium combesii	*S. aureus*
		Propionibacterium acnes	*S. agalactiae*
4	B	*Peptococcus indolicus*	*S. aureus*
		Eubacterium combesii	
5	C	*Veillonella parvula*	*E. coli*
		Megasphaera elsdeni	
6	D	*Peptococcus indolicus*	*C. pyogenes*
		Fusobacterium necrophorum	
7	E	*Peptococcus indolicus*	*C. pyogenes*
		Clostridium sporogenes	
8	F	*Bacteroides fragilis*	*S. aureus*
			S. agalactiae
9	G	*Bacteroides fragilis*	*S. aureus*
			S. agalactiae
10	H	*Bacteroides fragilis*	*S. agalactiae*
Subclinical mastitis			
11	C	*Bacteroides fragilis*	*S. aureus*
12	I	*Peptococcus indolicus*	*S. aureus*
13	J	*Eubacterium lentum*	*S. aureus*
14	K	*Peptostreptococcus* sp.*	*S. aureus*
'Aseptic' mastitis			
15	L	*Peptococcus indolicus*	absent

* Produced heparinase

subclinical mastitis (5,3 %) and 'aseptic' mastitis (10 %). Anaerobes were also absent in the milk of udders with latent infections from which facultative bacteria (usually *Staphylococcus aureus*) could be isolated from the teat canals.

A broad range of anaerobic species were isolated from mastitic udders (Table 2). Except for one case of 'aseptic' mastitis they were always accompanied by facultative bacteria. The subclinical forms of mastitis usually yielded a simpler bacterial population compared to that of clinical mastitis (Table 2). In this respect a pattern with regard to the average number of species present in the different forms of mastitis seems to establish itself as follows: C-mastitis (3) > SC-mastitis (2) > As-mastitis (1). From Table 2, two dominating associations of anaerobes with facultative organisms in cases of mastitis were apparent.

Firstly, *Peptococcus* spp. was present in 47,7 % of cases (7/15) while in 57,1 %(4/7) of such infections they were associated with *Corynebacterium pyogenes*. Similarly, *Bacteroides* spp. was always associated with either *S.aureus* or *Streptococcus agalactiae* or both. Together, these 2 types of associations occurred in 80 % (12/15) of cases of mastitis. The relative frequency of occurence of anaerobic species present in mastitic ud-

Table 3: RELATIVE FREQUENCY OF OCCURENCE OF ANAEROBIC SPECIES FROM MASTITIS

Organisms	Strains	Occurence %
P. indolicus	6	28,5
P. saccharolyticus	1	4,8
B. fragilis	4	19,0
B. melaninogenicus	1	4,8
E. combesii	2	9,5
E. lentum	1	4,8
C. sporogenes	1	4,8
F. necrophorum	1	4,8
M. elsdenii	1	4,8
Peptostreptococcus sp.	1	4,8
P. acnes	1	4,8
V. parvula	1	4,8

Table 4: ANTIMICROBIAL SENSITIVITY AND β-LACTAMASE PRODUCTION OF ANAEROBIC BACTERIAL STRAINS ISOLATED FROM CASES OF BOVINE MASTITIS

Anaerobe	Strain	Antimicrobial agents					
		Pen	Chl	Cli	Met	Cep	β-lactamase
P. indolicus	12/80	±	+	+	+	+	A
P. indolicus	E2/80	+	+	+	+	+	ND
P. indolicus	L1/80	+	+	+	+	+	ND
P. indolicus	M/81	+	+	+	+	+	ND
P. indolicus	G2/80	+	+	+	+	+	ND
P. indolicus	116/1	+	+	+	+	+	ND
B. fragilis	13/3	−	+	+	+	−	P
B. fragilis	N3/1	−	+	±	+	−	P
B. fragilis	M8/1	−	+	+	+	−	P
B. fragilis	3/1	−	+	+	±	−	P
E. combesii	I3/80	+	+	+	+	+	ND
E. combesii	L2/80	+	+	+	+	+	ND
E. lentum	15/1	+	+	+	+	+	ND
C. sporogenes	G1/80	−	+	−	+	+	ND
F. necrophorum	E3/80	−	+	−	+	+	A
Peptostreptococcus sp.	6/1	+	+	+	+	+	ND
P. acnes	I4/80	+	+	+	+	+	ND

Pen = penicillin – G, Cep = cephalothin. A = absent,
Chl = chloramphenicol, + = sensitive, P = present,
Cli = clindamycin, − = resistant, ND = not done.
Met = metronidazole, ± = partial resistance

ders is reported in Table 3. It is again evident that *Peptococcus* spp. (33,3 %) and *Bacteroides* spp. (23,8 %) as well as *Eubacterium* spp. (13,8%) are isolated far more frequently from various types of mastitis than are other anaerobic species (4,8 % each).

Antimicrobial sensitivity

The susceptibility of the different species of anaerobic bacteria to commonly used antimicrobial agents as well as their ability to produce β-lactamase is reported in Table 4.

DISCUSSION

The results obtained in this study differ in some important aspects from previous work in this field. In an earlier study Du Preez et al.[6] reported an isolation rate of 7,4 % for anaerobes from lactating cows with clinical mastitis. This is in significant contrast to an isolation rate of 58,8 % in the present study. The procedures employed for the sampling, isolation, cultivation and identification of anaerobes were essentially the same as those used in the previous study. However, in the majority of clinical mastitis cases in the first study[6], antibiotic treatment had already been initiated by the farmer before sampling could take place. We feel that the high isolation rates of anaerobes reported in the present study is due to the fact that milk from clinical and subclinical cases of mastitis was drawn before antimicrobial treatment had been initiated. Our finding of a 58,8 % involvement of anaerobes in clinical bovine mastitis is also in agreement with the higher isolation frequencies (often >60 %) of these organisms from a wide range of purulent human infections[2 7 21].

The polimicrobial nature of anaerobic infections is quite evident from both clinical and subclinical cases of mastitis, averaging respectively 3 and 2 species per cow. These infections always involved both anaerobic and facultative bacteria. Similar results are commonly reported from human infections[1 7].

Two dominant associations of anaerobes with facultative organisms were found in 80 % of mastitis cases. We found the most common combination to be *P. indolicus* with *C. pyogenes*. This confirms the findings in earlier studies[16 17 18 19]. Another important combination is that of *Bacteroides* species with *S. aureus* and *S. agalactiae*. These results suggest a possible synergistic action between these combinations of organisms during the course of establishing infection. It has, for example, been elegantly shown by the use of animal models that the combination *B. fragilis–Escherichia coli* exhibited interdependence and synergism in experimental peritonitis[1].

The ability of a *P. indolicus* strain and a *Peptostreptococcus* sp. to produce heparinase needs to be further investigated as a possible virulence factor. Since heparin has an important function as an inhibitor of the normal blood coagulation cascade[11], it follows that its inactivation by bacterial heparinases may have serious consequences. In man, septic thrombophlebitis is commonly associated with anaerobic infections involving heparinase producers[3 7]. This condition may lead to metastatic abscesses which are partially responsible for the difficulties encountered in treating anaerobic infections[3].

From their antimicrobial sensitivity profiles it is clear that the anaerobic Gram negative rods (*Bacteroides* and *Fusobacterium)* exhibited a wider pattern of resistance compared to the majority of Gram positive isolates. Since penicillin-based antibiotics are commonly used in the treatment of bovine mastitis, the reaction of anaerobes to penicillin-G is especially important. The Gram negative rods were all resistant to penicillin-G. In addition, all the *Bacteroides* strains produced β-lactamase. Most strains of *B. fragilis* reported from human clinical material behave similarly[14]. This would be important from a chemotherapeutic point of view. Due to the very frequent isolation of penicillin resistant

fragilis strains from cases of bovine mastitis, antibiotic treatment should be designed to overcome this problem. In a previous study[6], where antibiotic pretreatment of mastitic cows before sampling could not always be excluded, *B. fragilis* was the dominant species isolated. It was also noted that such cases each usually yielded only one anaerobe[5 6]. This may well mean that the more resistant strains survived treatment and led to a chronic condition refractive to antimicrobial treatment. Until more cases are studied, we cannot, however, conclude that cases of so-called 'aseptic' mastitis, involving only anaerobic bacteria, are the result of therapeutic failure or evidence of primary infection by anaerobic organisms alone. Perhaps both possibilities exist in nature.

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BOOK REVIEW — BOEKRESENSIE

RESEARCH ANIMALS AND CONCEPTS OF APPLICABILITY TO CLINICAL MEDICINE

K. GÄRTNER, H. HACKBARTH and H. STOLTE

Volume 7 in Experimental Biology and Medicine – Monographs on Interdisciplinary Topics. S. Karger, Basel. 1982. 46 figures and 35 tables. Price $88.75 (ISBN 3-8055-3492-2)

This book is the proceedings of the symposium entitled "Research Animals and Concepts of Applicability to Clinical Medicine" organised by the Deutsche Forschungsgemeinschaft at Hannover in 1981. The argument that certain results obtained from experiments on laboratory animals cannot be extrapolated to man, as put forward by certain people involved with the problems of animal welfare, is scientifically analysed and discussed. It is concluded that the structures and functions of the body do not differ randomly among the different mammalian species and if the general concepts of comparability are known, extrapolation of experimental results from animals to man is possible.

The book is divided into 2 main sections. Concepts of comparability between the mammalian species as pertaining to biological research is firstly discussed. This is followed by a section on selected fields of medical clinical research such as ageing, immunology, nephrology, rheumatology, reproduction and oncology. A paper on the uses and limitations of tissue culture models in clinical research is included. Finally a section on miscellaneous animal models for research is presented.

For the research scientist in the field of laboratory animal science, experimental medicine and experimental biology, this volume will serve a useful purpose.

W.S. Botha

FATAL CARDIOMYOPATHY IN FEEDLOT SHEEP ATTRIBUTED TO MONENSIN TOXICOSIS

S.J. NEWSHOLME*, ELIZABETH W. HOWERTH*, STELLA S. BASTIANELLO*, L. PROZESKY* and J.A. MINNÉ**

ABSTRACT: Newsholme S.J.; Howerth, E.W.; Bastianello, S.S.; Prozesky, L.; Minné, J.A. **Fatal cardiomyopathy in feedlot sheep attributed to monensin toxicosis.** *Journal of the South African Veterinary Association* (1983) **54** No. 1, 29-32 (En) Section of Pathology, Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa.

Three outbreaks of fatal cardiomyopathy in feedlot sheep are reported which were associated with the introduction of pelleted feed that contained greater than recommended concentrations of monensin. Gross and histopathological examination of some of these sheep revealed evidence of cardiomyopathy. Myocardial lesions which had a predominantly epicardial distribution are described. Lesions were also observed in skeletal muscle and in brain.

Key words: Cardiomyopathy, sheep, monensintoxicity, poisoning.

INTRODUCTION

Monensin, an ionophore antibiotic obtained from *Streptomyces cinnamonensis*, has been shown to increase feed efficiency in cattle[11 12] and sheep[7 10] when included in the ration at low levels. The increased feed efficiency has been ascribed to an effect of monensin upon the ruminal microflora which results in a substantial increase in the ratio of propionic to butyric and acetic acid production[13]. Low levels of monensin are currently included in commercial feeds for ruminants in many countries, including South Africa, to improve feed efficiency.

Cardiac failure associated with accidentally high levels of monensin in the feed has been reported in cattle[1 2 6]. Large single doses of monensin have also resulted in rapid deaths in lambs[3]. Monensin has positive inotropic actions in mammalian myocardium which are thought to result from the effect of this cationic ionophore upon sodium ion transport across cell membranes[14 15].

Herein are reported 3 outbreaks of deaths in sheep, which were caused by cardiomyopathy attributed to high levels of monensin in the feed. Pathological features of some affected sheep are described.

CASE HISTORIES

Outbreak 1

In a feedlot near Ermelo, Transvaal, 125 out of 600 feeder lambs and adult ewes died within a period of 10 days. These sheep were of mixed breeds which consisted of Merino, German Merino and Suffolk-crosses. They were fed *Eragrostis curvula* hay ad libitum and a commercially produced pelleted feed which contained monensin. A new batch of pelleted feed was introduced and was withdrawn after 6 days. The deaths commenced approximately 3 days after introduction of this batch of feed and continued during the time it was fed. Only 6 cases occured after the pelleted feed was withdrawn.

Deaths occurred suddenly and illness prior to death was rarely noted. The owner could not describe clinical signs. Approximately 75 % of the deaths were among the lambs and no breed appeared to be preferentially affected. Four dead lambs and formalin-fixed tissues from 3 others were submitted to the Veterinary Research Institute, Onderstepoort, for pathological examination. Samples of the pelleted feed were collected at the time of the outbreak for analysis.

Outbreak 2

In a feedlot near Bloemfontein, Orange Free State, 115 out of 1 200 Merino-cross feeder lambs died within a period of 2 weeks. Some of these lambs died suddenly while being moved for a head count. Others became dyspnoeic and recumbent and they died after several hours. In a few cases posterior ataxia and weakness were evident. All lambs that showed signs of illness died subsequently.

These lambs were being fed exclusively on a commercially produced pelleted feed containing monensin. Deaths commenced 2 days after the introduction of a new batch of pelleted feed and ceased 6 days after this feed was withdrawn.

Five lambs were submitted to Onderstepoort for necropsy 8 days after the deaths commenced. Four were dead and one was alive but recumbent and unable to stand. Three bags of this batch of pelleted feed were collected for analysis.

Outbreak 3

In a feedlot near Vrede, Orange Free State, 44 out of 360 5-month-old Merino feeder lambs died at a rate of 5-6 daily over a period of 1-2 weeks. Deaths commenced 5-6 weeks after the lambs were introduced to the feedlot. These lambs were fed *Eragrostis curvula* hay ad libitum and restricted amounts of a commercialy produced pelleted feed containing monensin for an adaptation period of 1 week following their introduction to the feedlot. Thereafter they were fed the pelleted feed exclusively. Most of the deaths were sudden. Some of the lambs showed hindleg weakness and "knuckling over" at the metatarsal joints, and some became recumbent.

A live lamb showing hindleg weakness and formalin-fixed tissues from a lamb which had died were submitted to Onderstepoort for examination. Samples of the pelleted feed were collected for analysis.

MATERIALS AND METHODS

Feed Analysis

The samples of pelleted feed were examined for monensin content according to the colorimetric method described by Golab at al.[4]

Pathological Examination

The sheep that were received alive were killed by intravenous injection of pentobarbitone sodium. Necropsies were performed on all the sheep received. Selected tissues were fixed in 10 % buffered formalin. Sections

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of the fixed tissues from these sheep and of the fixed tissues received were prepared and stained with haemotoxylin and eosin (HE).

RESULTS

Feed Analysis

The monensin concentrations measured in the feed samples are presented in Table 1.

Table 1: MONENSIN CONCENTRATIONS OF THE FEED SAMPLES

Sample		Monensin Concentration (parts per million)
Outbreak 1	A	83
	B	88
	C	93
Outbreak 2	A	382
	B	430
	C	650
Outbreak 3	A	164

Gross Pathology

The carcasses of the 7 sheep received dead were in good condition. They showed generalized congestion, marked pulmonary oedema with froth in the trachea, and mild to severe hydrothorax, hydropericardium and ascites. In 2 cases the epicardial surfaces had a mottled appearance. This was imparted by multiple, irregular pale areas of varying size which were distributed throughout the ventricular walls but which predominantly involved the epicardial myocardium (Fig. 1). Small epicardial haemorrhages were numerous in 3 cases. The small intestinal mucosa was diffusely congested and the contents were blood-stained and watery in 6 of these sheep. Congestion of the mucosa was evident in the caecum and colon of 1 case. The livers were markedly swollen and congested and centrilobular areas were pale in 2 of them. Kidneys were moderately swollen in all cases.

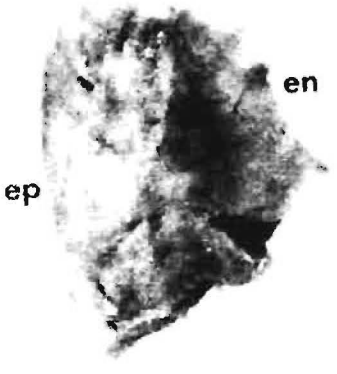

Fig. 1. Pallor of left ventricular wall involving the epicardial myocardium (ep) and sparing the endocardial myocardium (en). Formalin-fixed tissue.

Of the sheep that were received alive, dark brown urine was the only significant gross finding in the case from Outbreak 2. In the case from Outbreak 3, both semimembranosus muscles showed pronounced diffuse pallor and contained petechiae throughout. Petechiae were more numerous at the musculo-tendinous junctions. The urine was pale red.

Microscopical pathology

Heart: Cardiac lesions were present in all cases. The epicardial myocardium was consistently the predominant site of involvement although scattered lesions occurred throughout the myocardium.

Mild lesions were characterized by perivascular infiltrates consisting of lymphocytes and occasional neutrophils and macrophages which were frequently associated with small foci of myocardial necrosis (Fig. 2). Myocardial fibres in these foci were shrunken and irregular in transverse section and possessed strongly eosinophilic, hyalinized or vacuolated cytoplasm and pyknotic nuclear remnants. Individual mineralized myocardial fibres were seen both within the necrotic foci and scattered throughout the myocardium in 2 cases.

The more severely affected hearts contained multiple areas of extensive myocardial necrosis and attendant mild inflammatory cell infiltrates (Figs. 3 & 4). In 3 cases these changes involved the epicardial myocardium diffusely. Features of necrotic myocardial fibres were similar to those in the mild lesions. In some of the lesions, however, there was complete loss of myofibres, leaving only the stromal elements remaining (Fig. 4). The attendant cellular infiltrates consisted of lymphocytes, plasma cells, macrophages and a few neutrophils. These cells also infiltrated adjacent epicardium. Plump fusiform cells with weakly basophilic cytoplasm and large, ovoid vesicular nuclei, which were interpreted as immature fibroblasts, were observed amongst the necrotic myofibres and in the stromal tissue where myofibre loss had occurred. In 2 cases small groups of myofibres within or neighbouring the areas of myocardial necrosis were enlarged and distorted by pronounced cytoplasmic vacuolation (Fig. 5).

Lungs: In all the sheep received dead there was mild to severe congestion and patchy to diffuse alveolar oedema.

Intestine: Intense congestion of the mucosal and submucosal vessels was a consistent finding in all the sheep received dead. In one sheep from Outbreak 2 scattered cryptal abscesses and small superficial erosions were observed in the colon.

Kidneys: Moderate congestion and mild nephrosis were consistent findings. Nephrosis was characterized by slight dilatation of the lumens of the tubules in the cortex and Bowman's spaces which contained small amounts of weakly eosinophilic homogeneous material and occasional eosinophilic granular casts.

Liver: Mild to severe congestion affected mainly the centrilobular areas. Centrilobular hepatocellular necrosis was evident in 2 cases from Outbreak 1.

Spleen: Necrosis of the Malpighian bodies was observed in 5 cases.

Lymph nodes: Congestion was consistently present, and necrosis of the germinal centres was observed in 3 cases.

Brain: Mild vacuolation of the white matter was present in 2 cases. This was noted in the pons and corpora

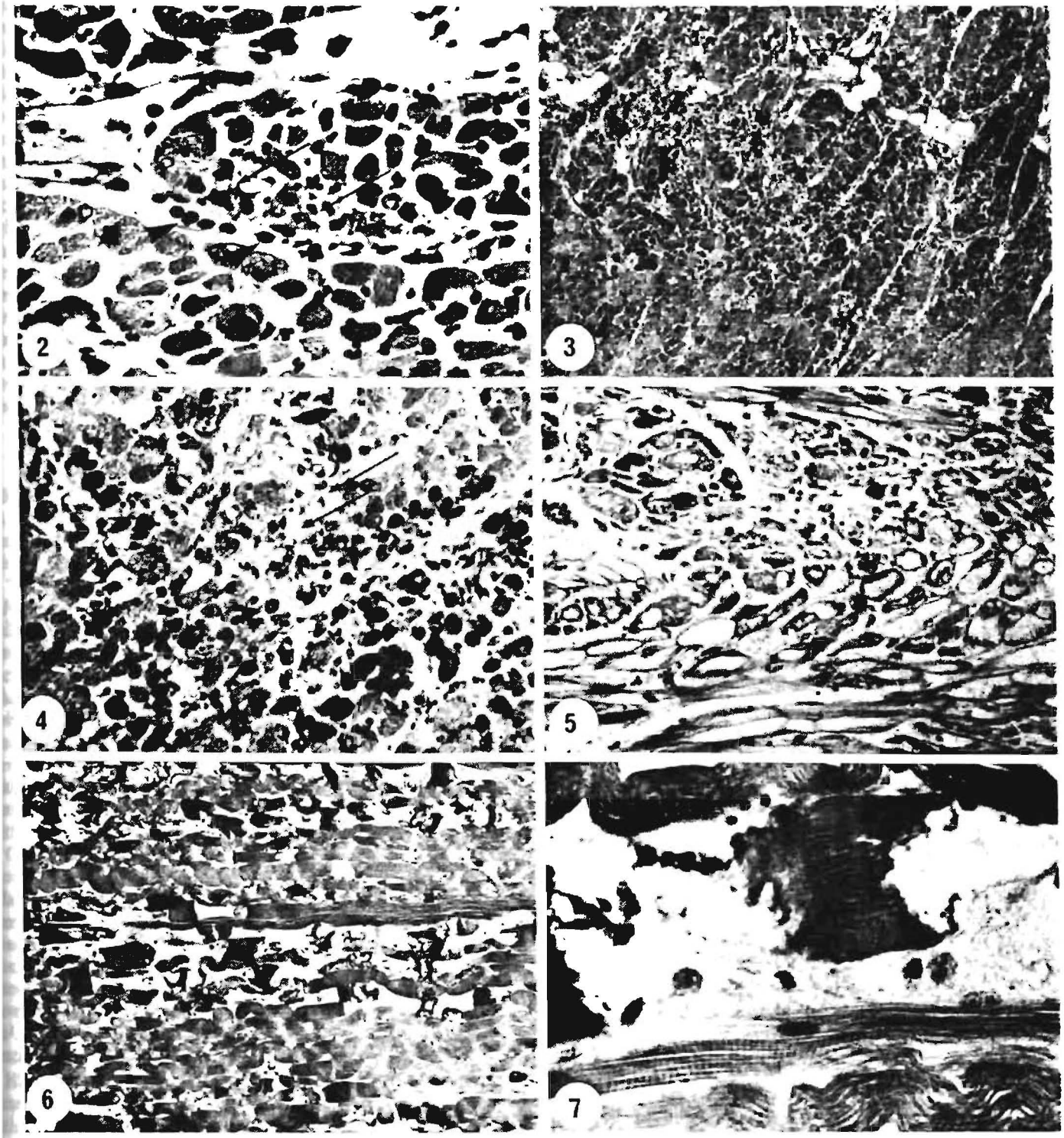

Fig. 2. Small focus of myocardial necrosis (arrows) and associated inflammatory cells (arrowheads). HE X 200

Fig. 3. Extensive myocardial necrosis and mild inflammatory cell infiltrates; necrotic myofibres appear dark. HE X 50

Fig. 4. Area of necrotic myocardial fibres and myofibre loss (arrows). HE X 200

Fig. 5. Vacuolation of myocardial fibres. HE X 200

Fig. 6. Extensive fragmentation of myofibres; semimembranosus muscle. HE X 100

Fig. 7. Sarcoplasmic fragmentation, lysis and mild inflammatory cell infiltrate; semimembranosus muscle. HE X 1 000

quadrigemina in one case and in the internal capsule and cerebral cortex in the other.

Skeletal muscle: In the semimembranosus muscles of the sheep received alive from outbreak 3 acute rhabdomyolysis was extensive. This was characterized by swelling of the myofibres and hyalinized, strongly eosinophilic sarcoplasm which contained pyknotic nuclear remnants. Many of the myofibres were transversely fragmented (Figs. 6 & 7). Segments of sarcoplasm presented a pale, finely granular appearance which suggested lysis (Fig. 7). Mild infiltrates of macrophages and neutrophils were present in the interstitial tissie and occasional neutrophils occupied the sarcoplasm of affected myofibres. Individual myofibres presenting similar changes and attended by a few inflammatory cells were observed scattered in other skeletal muscles from this case and also in skeletal muscles from the live sheep received from outbreak 2 and in fixed muscle received.

DISCUSSION

Historical and clinical features of the outbreaks as well as pathological findings in affected sheep suggest that monensin toxicosis was responsible for the fatal cardiomyopathy observed in these outbreaks.

The level of monensin included in sheep feed in South Africa is recommended not to exceed 22 parts per million (C.G. Cotton, Technical Adviser, Fertilizers, Farm Feeds, Agricultural Remedies and Stock Remedies Act (Act 36 of 1947), personal communication, 1982). This level was exceeded substantially in samples of feed collected from all 3 outbreaks. Deaths were clearly associated chronologically with the introduction of a new batch of feed and ceased shortly after it was withdrawn in 2 of these outbeaks. Rapid death suggestive of acute cardiac failure has also been associated with monensin toxicosis in cattle[2].

Generalized congestion, pulmonary oedema, hydrothorax, hydropericardium, ascites and ventricular dilatation which were observed consistently in these sheep suggested congestive cardiac failure. Similar changes have been reported in cattle with monensin toxicosis[1 2 6]. As in these sheep, congestion of the small intestine and watery, blood-stained intestinal contents have also been described in cattle poisoned by monensin[2]. Catarrhal haemorrhagic duodenitis has been described in lambs given a high dose of monensin[3].

Histopathological features in the heart muscle of these sheep which have also been documented in cattle with monensin toxicosis comprised myocardial necrosis with inflammatory cell infiltrates[6] and cytoplasmic vacuolation of myocardial fibres[1]. In a study of experimental monensin toxicosis in ponies cytoplasmic vacuolation of myocardial fibres was found to be a striking early change, and severe mitochondrial swelling contributed to this vacuolation which was possibly related to the cationic ionophore activity of monensin[9]. Based on these outbreaks, one feature which may assist to differentiate monensin toxicosis from other toxic cardiomyopathies in sheep is the predominantly epicardial distribution of the myocardial lesions.

Skeletal muscle lesions having a microscopical resemblance to those observed in these sheep have been described in experimental monensin toxicosis in chickens[5]. The reason for the selective involvement of the semimembranosus muscles in one sheep is not clear. Red urine in 2 of the cases with skeletal muscle lesions raises the possibility of myoglobinuria associated with muscle necrosis. However, no clinical pathology was undertaken. Myoglobinuria was also suspected in a dog with clinical evidence of myopathy associated with monensin toxicosis[16].

Ataxia, which affected some cases in 2 of these outbreaks has been reported in lambs in experimental monensin toxicosis[3]. We consider that the ataxia and weakness were related to the skeletal muscle lesions which we encountered, rather than to the mild cerebral lesions. However, the possibility of clinical cerebral involvement cannot be excluded. In a series of horses which developed ataxia associated with monensin poisoning no evidence of myopathy could be found, and nervous system involvement was proposed as a possible explanation for the ataxia[8].

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We wish to thank the technical staff of the Pathology Section, Veterinary Research Institute, Onderstepoort, for preparing the histological sections and the staff of the Photography Section for preparing the photographs.

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NOTES ON THE TOXICITY AND CARCINOGENICITY OF SOME SOUTH AFRICAN CYCAD SPECIES WITH SPECIAL REFERENCE TO THAT OF *ENCEPHALARTOS LANATUS**

R.C. TUSTIN**

ABSTRACT:Tustin R.C. **Notes on the toxicity and carcinogenicity of some South African cycad species with special reference to that of *Encephalartos lanatus*. *Journal of the South African Veterinary Association*** (1983) **54** No. 1, 33-42 (En) Department of Pathology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

The South African *Encephalartos* species which are known or have been shown experimentally to be toxic and/or carcinogenic are reviewed briefly. The kernel of *E. lanatus,* the toxic and carcinogenic principle of which is macrozamin, is a potent hepatotoxin and carcinogen when fed to rats. Renal mesenchymal tumours were the most common neoplasm to be induced but hepatocellular carcinoma also developed in one animal. The potential danger to man and animals of ingestion of the seeds of *Encephalartos* species is emphasized.

Key words: *Encephalartos* species, cycads, hepatotoxin, carcinogen, rat renal mesenchymal tumour, hepatocellular carcinoma, macrozamin, methylazoxymethanol.

INTRODUCTION

It has long been known that certain cycad species are toxic for man and animals and that some of them have been proved to be carcinogenic when fed experimentally to animals. Members of this group of plants fall into 3 families: Cycadaceae (containing 1 genus, *Cycas*), Stangeriaceae (1 genus and 1 species, *Stangeria eriopus)* and Zamiaceae (8 genera including *Encephalartos*). In Africa they are represented by 45 or more species of *Encephalartos* and *S. eriopus*. In Southern Africa about 28 *Encephalartos* spp. and *S. eriopus* occur[9].

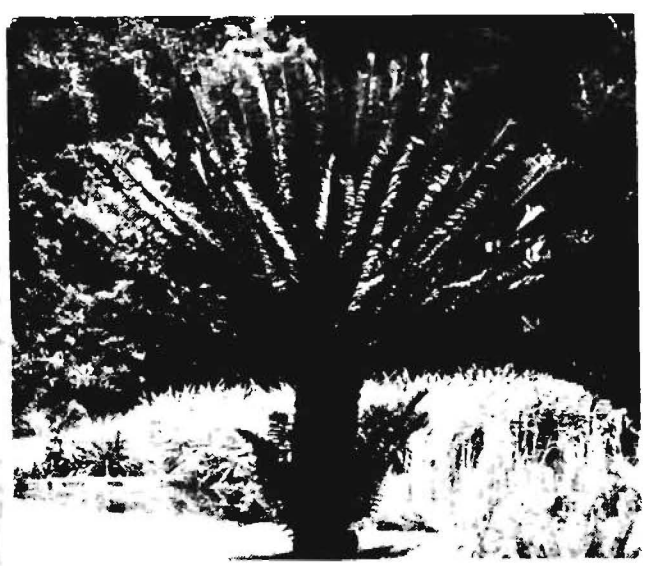

Fig. 1. *E. transvenosus* about 2,5 m in height. National Botanic Gardens, Pretoria.

Despite their toxicity several species have been, and still are, used medicinally and as food for humans in various countries of the world[48]. When used as a regular source of human food the part consumed is first detoxified by a variety of processes, most apparently based on the fact that the toxin is water-soluble.

The first records in South Africa of their preparation and use as human food were given by early travellers (1772-1779) such as Thunberg, Masson, Swellengrebel and Pattersen, who referred to the use by Blacks of the starchy pith from the stems of *Encephalartos* species for making crude bread – hence the popular names of these plants in this country, Hottentot- or Kaffirbread (Kafferbroodboom) or, simply, bread-tree[6]. In the preparation of this bread, the pith is removed from the stem, placed in an animal skin and buried in the ground for up to 6 weeks to ferment. It is then ground, mixed with water to a paste and finally roasted. Detoxification may be aided by fermentation (Nishida, 1936 as cited by Whiting[48]), although it is not definitely known if the pith of the species used does indeed contain a toxic factor. This method of preparation may, however, indicate that it was known to produce a deleterious effect if eaten unfermented. Dyer thinks that all cycad species of the Eastern Cape Province have been used for food on occasion[6].

In times of food shortage and famine in East Africa, starch obtained from the stems and kernels of *Encephalartos hildebrandtii* is used after detoxification for human consumption[25]. Beer is also prepared from the stems of *Encephalartos* species in Central, East and South Africa and in Mozambique[23]. In the Transkei, Rose reports that the bark of *Encephalartos altensteinii* and *S. eriopus* is cut up and boiled, and the liquid drunk as an emetic to cure coughs and to wash away bad dreams[30]. In addition, the bark of *S. eriopus* allegedly cures fever. Women with small babies wear a necklace of *Stangeria* roots and, when a baby is ill, the mother bites off a piece, chews it, and gives it to the child as an emetic. Giddy states that she has actually seen Black children eating the fleshy pulp of the Modjadji palm, *Encephalartos transvenosus* in the Northern Transvaal, while in the Eastern Cape Province the seeds of *E. altensteinii* have been a seasonal addition to the diet of the local tribes for generations[9]. She also mentions that the leaves of all South African *Encephalartos* species are freely eaten by sheep, buck, rock rabbits and baboons, while the fleshy outer covering of the seed, but not the actual kernel, is ingested by baboons, monkeys, rodents, bats and many fruit-eating birds such as the Crowned Hornbill, Trumpeter Hornbill and Brown-necked (Cape) Parrot. Dyer records that louries are said to be particularly fond of them[6].

Poisoning in man by cycads is invariably due either to ignorance or to incomplete detoxification. The manifestations of toxicity range from a mild to a severe (sometimes fatal) gastro-intestinal irritation (nausea,

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vomition, anorexia, diarrhoea), headache, vertigo, muscle paralysis, apathy and rheumatism. Evidence of liver injury can also be expected as this organ is severely affected in toxicity experiments in animals[6 39 40 48]. Mugera & Nderito mention that jaundice develops in children within 2–6 days after ingestion of toxic material[25].

Probably the first account of the toxicity of cycads occurring in the Republic of South Africa was that of Reitz[27] who mentioned that several members of the Boer Commando, including its leader, General J.C. Smuts, ate the seeds of the "Hottentot's bread", *E. altensteinii,* while in the Suurberg of the Eastern Cape Province during the South African War of 1899-1902[7 27]. Serious illness was experienced by many of those concerned and Smuts himself was severely incapacitated for at least 5 days. Dyer thinks that the offending species was *Encephalartos longifolius* rather than *E. altensteinii*[6]. Ingestion of the seeds of *E. longifolius* have been held responsible for the deaths of 2 cattle in the Eastern Cape Province[6].

The outer flesh of the kernels and/or kernels of the South African cycads *Encephalartos cycadifolius,*

Fig. 2. *E. laevifolius* with immature female cones which are approximately 350 mm in length. National Botanic Gardens, Pretoria.

Table 1: SUMMARY OF TOXIC AND CARCINOGENIC EFFECTS OF INCORPORATING VARIOUS CONCENTRATIONS OF DIFFERENT PARTS OF FEMALE CONES OF 4 CYCAD SPECIES INTO THE DIET OF RATS[43]

Cycad species	Effects of feeding various parts of the female cone				
	Mixture of outer flesh of kernel and kernel	Outer flesh of kernel alone	Kernel alone	Mixture of cone axis and scales	Cone axis alone
E. umbeluziensis	Hepatopathy RMT* RAC** CC***	—*	—	Hepatopathy	—
E. villosus	Hepato- & nephropathy HC****	Hepato- & nephropathy RMT HC	Hepato- & nephropathy	—	Nephropathy
E. lebomboensis	—	Hepatopathy RMT	NS**	—	—
E. laevifolius	—	Hepatopathy RMT	Hepato- & nephropathy RMT RAC	—	—

*— = not tested
**NS = no significant lesions at concentrations fed
*RMT = renal mesenchymal tumours developed in one or more of the rats
**RAC = renal adenocarcinoma developed in one rat
***CC = cholangiocarcinoma developed in one rat
****HC = hepatocellular carcinomas developed in one or more rats

Encephalartos eugene-maraisii, Encephalartos kosiensis (now knows as *Encephalartos ferox), Encephalartos lehmanii, E. longifolius* and *Encephalartos villosus* have either been proved to be acutely toxic when administered experimentally to rabbits or have been suspected, or known, to be poisonous when ingested by man[9 40 43]. Tustin has recorded the experimental toxicity and/or carcinogenicity in rats produced by feeding various parts of the female cone of 4 South African cycad species, *Encephalartos umbeluziensis, E. villosus, Encephalartos lebomboensis* and *Encephalartos laevifolius*[43]. The results depended to some extent on the concentration of the material in the diet, the nature of the plant material, the length of time fed and the survival period. A brief summary of the results of these experiments is given in Table 1.

Apart from the neoplasia (*vide infra)*, the most outstanding lesions in rats dying acutely were the hepatocytic necrosis which was chiefly periacinar in distribution and frequently accompanied by architectural collapse of affected liver lobules, nodular hyperplasia of the liver, and hyperplastic and hypertrophic alterations in renal tubular epithelium. These changes have also been described by others concerning other

species of cycads or the active toxic principle[12 13 20 21]. Hepatocytic nodular hyperplasia, mild bile duct hyperplasia and the presence of groups of cystic bile ducts characterized the more chronic hepatic changes. It is interesting to note that nodules of hepatocytic hyperplasia were seen in animals that had received in their rations cone axis and scales of *E. umbeluziensis,* and a mild nephropathy consisting of epithelial hyperplasia and hypertrophy in those receiving the cone axis alone of *E. villosus*[43]. Although only small numbers of rats were used in the experiments because of a limited availability of the plant material, these changes indicate that the toxic principle is probably not just confined to the kernel and its outer flesh.

Toxicity and/or carcinogenicity of several cycad species throughout the world depend largely on the presence of the related azoxyglycosides, macrozamin or cycasin as well as on the presence of certain intestinal bacteria which possess the necessary enzymes to hydrolyse these glycosides, thus releasing the common toxic fraction, methylazoxymethanol[16]. Some animal species or individuals may not harbour such bacteria which might explain apparent differences in susceptibility or resistance to intoxication.

Cooper (1941) was the first to isolate an azoxyglycoside from a cycad[4]. He obtained a crystalline substance which he named macrozamin from seeds of an Australian species, *Macrozamia spiralis*. Macrozamin was toxic to guinea pigs when given by mouth but nontoxic when injected subcutaneously. It was later shown to consist of the aglycone, methylazoxymethanol attached to a carbohydrate component, primeverose, in a betaglucosidic link[22]. It has subsequently been found in several more Australian species of *Cycas, Macrozamia* and *Bowenia*[12 28], in the African species *Encephalartos barteri* (Lythgoe as cited by Riggs[28]), in the East African species *E. hildebrandtii*[5], and in the 2 South African species, *E. transvenosus* and *Encephalartos lanatus*[1].

Cycasin, a glycoside closely related to macrozamin, consists of the same aglycone but combined to the sugar moiety, D-glucose. This has been found in the Japanese cycad, *Cycas revoluta* (Nishida, Kobayshi & Nagahama 1955 as cited by Laqueur & Spatz[21]) and the Guamanian species *Cycas circinalis*[29].

It appears that, in general, the deleterious effects of cycads are related to the age, species, strain and diet of the animals concerned as well as the route of administration and the part of the plant used[3 10 14 31 38 43]. Methylazoxymethanol is an extremely potent toxin (affecting particularly the liver) and carcinogen[20 21 26]. When administered to newborn experimental animals it is also neurotoxic[14 15 19 32], teratogenic[36], mutagenic[33 41] and possesses alkylating and radiomimetic properties[34 42]. Furthermore, the transplacental induction of neoplasms in rats fed on the kernel or husk of the seeds of *C. circinalis* and the presence of the toxin in the milk of rats, cows and sows fed cycasin have been reported[35 37 45 47]. The precise nature of the toxic principle responsible for the cattle disease characterized by degenerative lesions in the spinal cord seems to be still unresolved[8].

Many species of cycads have not yet been scientifically investigated. An opportunity to study the toxicity and carcinogenicity of the kernel of *E. lanatus* in rats arose; this forms the further basis of this report. *E. lanatus* occurs in the catchment area of the Olifants River in the Middelburg, Witbank and Bronkhorstspruit districts of the Transvaal[9].

Fig. 3. A relatively "young" *E. lanatus* growing in the National Botanic Gardens, Pretoria.

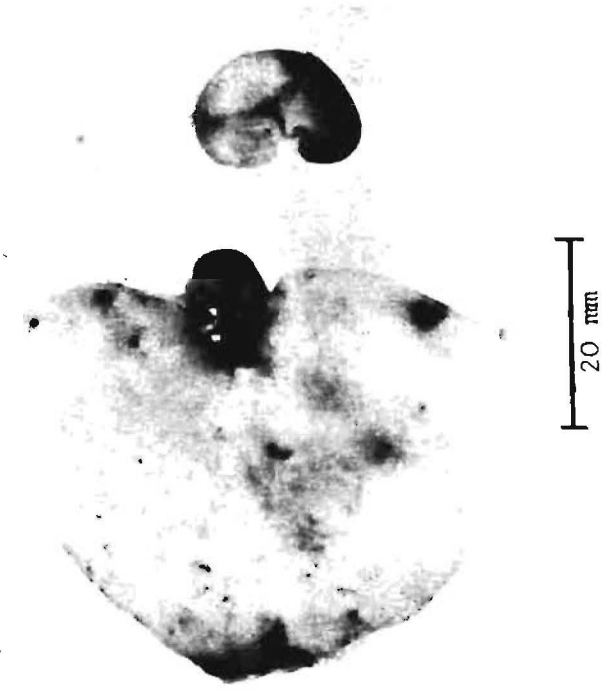


Fig. 4. Bilateral renal mesenchymal tumours in the kidneys of a rat that died 226 days after it had received a ration which contained 10 % of the outer flesh of the kernel of *E. laevifolius* for a period of 40 days[44].

MATERIALS AND METHODS

Forty young female Wistar rats with an initial live mass of between 100 and 150 g were used. These animals originated from the breeding colony of the National Research Institute for Nutritional Diseases, Medical Research Council and throughout their lives they were housed, one or 2 to a cage in air-conditioned rooms maintained at a temperature of 25°C and a minimum relative humidity of 55 % and illuminated with fluorescent lights for 12 hours a day.

(Continued on page 38)

The live mass of each animal was recorded at the commencement of the experiment, thereafter weekly and at death. The rats were divided into 5 groups each of which contained 8 animals and received a different dietary ration.

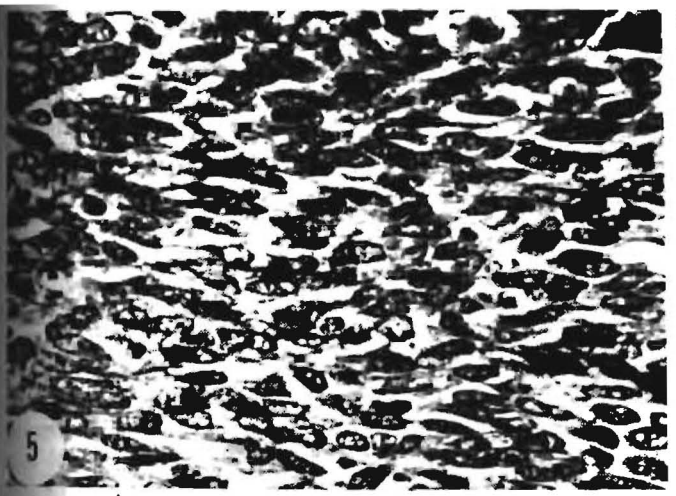
Kernels of the seed of *E. lanatus* were obtained and dried in an oven at 55°C. They were then ground into a powder to facilitate mixing in concentrations of 1 %, 2,5 %, 5 % and 10 % by mass with aliquots of a basic rat ration which comprised a standard commercial rat ration. A control ration consisting of the same basic ration to which 10 % by mass of dextrin was added was also prepared. Each of the 5 experimental rations was fed to a different group of rats at the commencement of the trial, but with the exception of that given to the control group of rats which received the ration containing 10 % dextrin throughout the experimental period, these rations were fed for varying periods of time after which surviving rats were given the standard ration. Both feed and drinking water were available ad libitum to the animals. Details of the groups and the number and mass of the rats as well as experimental rations and the periods for which they were fed are given in Table 2.

After commencement of the experiment all the animals were inspected daily. Those that appeared abnormal were examined clinically and several of the rats in which neoplasms were detected on clinical examination were slaughtered by decapitation. These and all the animals which died during the course of the experiments or were killed by decapitation when it was arbitrarily decided to end the experiment, were necropsied. Specimens from all major organs were then taken and

Table 2: ADMINISTRATION OF *ENCEPHALARTOS LANATUS* KERNEL TO RATS

Group	Ration	Rat		Mass (g)		Duration of feeding Exptl. Ration (Days)	Duration in Expt. (Days)	Manner of Death***	Principal Lesions
		Number	Sex**	Initial	At death				
1	Ration* + 1 % kernel	1	F	137	257	41	546	S	Hepatopathy + kid. mesench. tum.''
		2	F	140	261	41	546	S	Hepatopathy
		3	F	150	252	41	546	S	do
		4	F	146	232	41	546	S	do
		5	F	141	239	41	546	S	do
		6	F	139	269	41	546	S	do
		7	F	132	230	41	546	S	Hepatopathy + kid. mesench. tum.
		8	F	129	246	41	546	S	do
2	Ration* + 2,5 % kernel	9	F	147	293	41	275	S	Severe hepatopathy + kid. mesench. tum.
		10	F	140	180	41	288	D	do
		11	F	142	354	41	501	D	Severe hepatopathy
		12	F	146	120	38	38	D	Severe acute hepatopathy
		13	F	138	250	41	341	D	Severe hepatopathy + kid. mesench. tum.
		14	F	147	254	41	326	D	Hepatocellular Ca.''' + kid. mesench. tum.
		15	F	148	287	41	344	D	Severe hepatopathy + kid. mesench. tum.
		16	F	144	149	41	423	D	Hepatopathy
3	Ration* + 5 % kernel	17	F	135	73	21	21	D	Acute hepatopathy
		18	F	138	88	30	36	D	do
		19	F	136	80	23	23	D	do
		20	F	137	94	22	22	D	do
		21	F	137	86	11	11	D	do
		22	F	138	83	23	23	D	do
		23	F	147	93	22	22	D	do
		24	F	144	70	21	21	D	do
4	Ration* + 10 % kernel	25	F	151	81	21	21	D	Acute hepatopathy
		26	F	141	96	15	15	D	do
		27	F	146	80	20	20	D	do
		28	F	151	83	16	16	D	do
		29	F	146	99	15	15	D	do
		30	F	144	86	19	19	D	do
		31	F	139	75	18	18	D	do
		32	F	141	80	21	21	D	do
5 (Control)	Ration* + 10 % dextrin	33	F	152	239	546	546	S	NS'
		34	F	140	169	546	510	D	Pneumonia
		35	F	135	218	546	546	S	NS
		36	F	139	248	546	546	S	NS
		37	F	149	292	546	546	S	NS
		38	F	136	251	546	546	S	NS
		39	F	159	282	546	546	S	NS
		40	F	160	247	546	546	S	NS

*Ration = Standard commercial rat ration;
**F = Female;
***D = Died of own accord;
S = Slaughtered;
'NS = No significant lesions;
''Kid. mesench. tum. = Renal mesenchymal tumour;
'''Ca. = carcinoma.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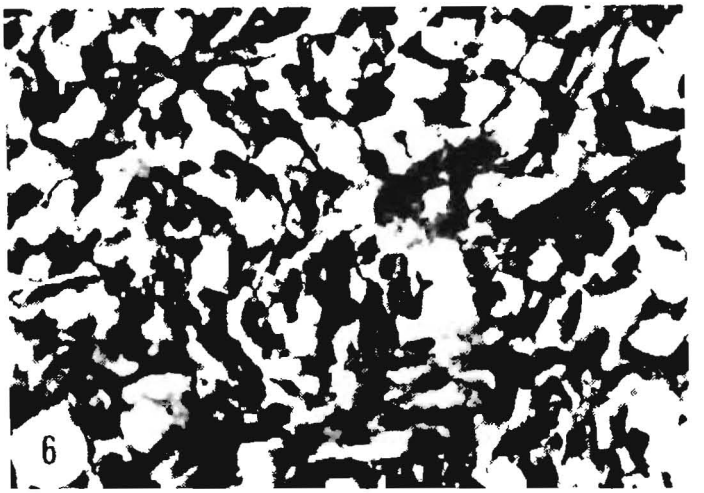
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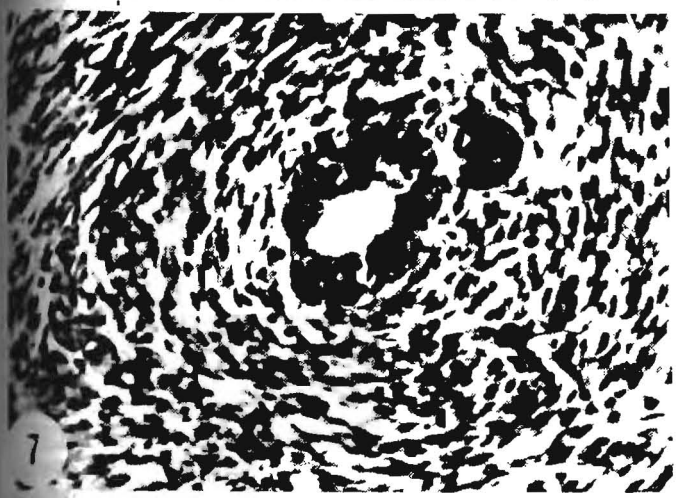
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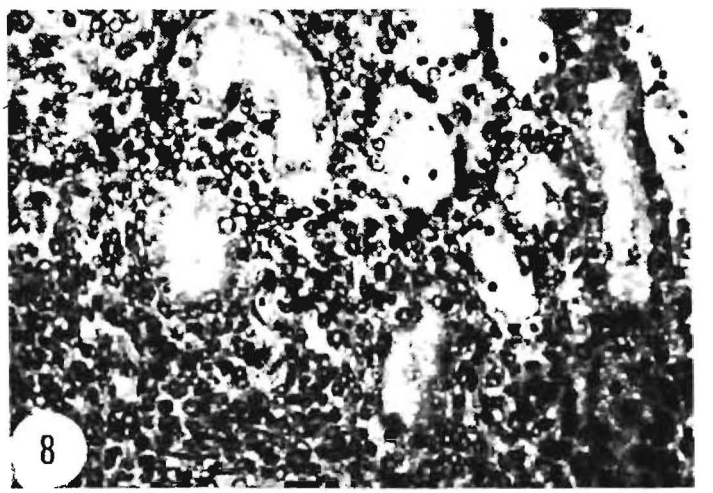
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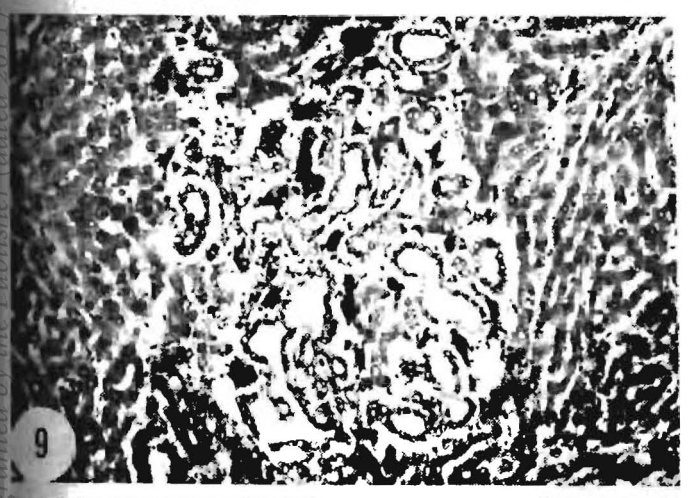
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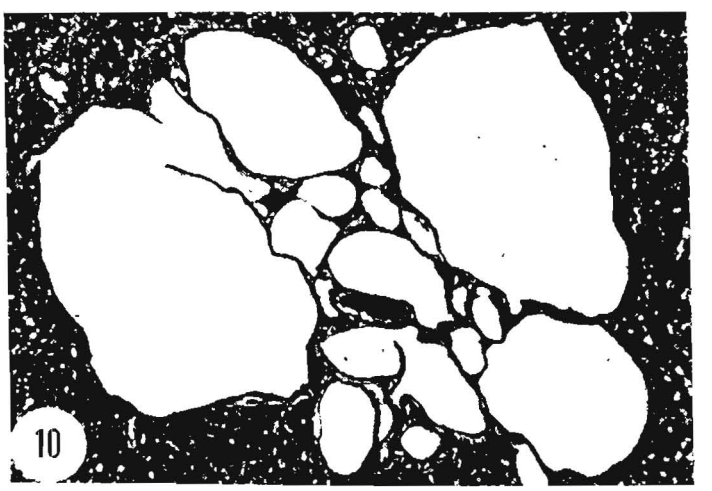
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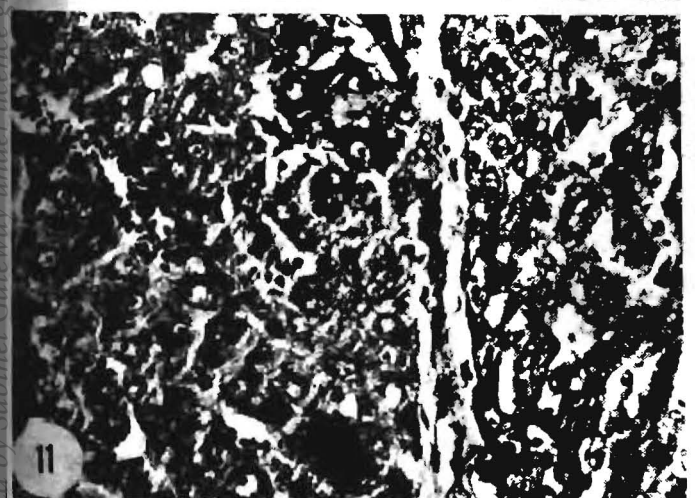
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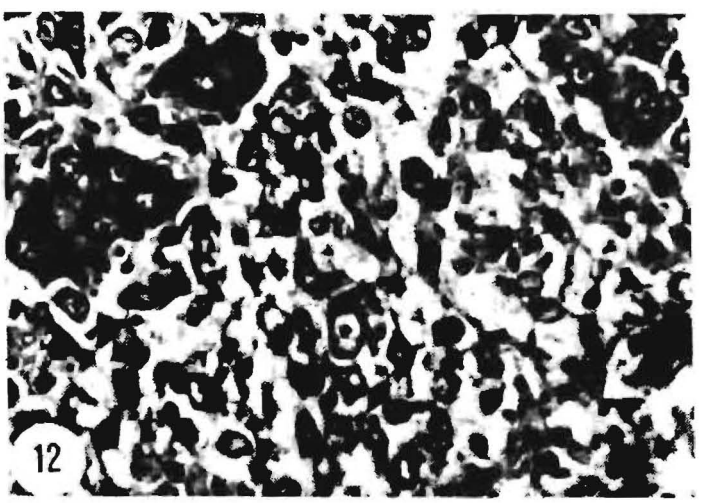
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ed in 10 % buffered formalin solution. Sections for ght microscopy were cut and stained with haematoxin and eosin using routine procedures. Sections from lected organs were stained, in addition, with Masson's chrome stain, by the periodic acid Schiff technique nd by Wilder's method for reticulum.

RESULTS

The survival times, manner of death of the animals, the principal lesions seen and other data are summarized in Table 2.

For the first 41 days of the experiment the rats in *Group 1* were given a diet containing 1 % kernel. All were subsequently slaughtered on Day 546 and on post mortem examination kidney neoplasms were found in 3 animals. In Rat 1 one of the kidneys was completely replaced by white tumour mass approximately 5 times the size of a normal kidney. The other kidney appeared normal. Both the kidneys of Rat 7 were almost completely replaced by large neoplasms about 45 mm in diameter. Bilateral renal tumours were also present in Rat 8 but these were not as large as those in the other 2 rats. The livers of 5 of the 8 rats contained between one and 4 cysts, each being from 1-2 mm in diameter and containing clear colourless fluid. Smaller lighter coloured nodules 1-2 mm in diameter were noticed in the parenchyma. The surface of the livers of several of the animals was slightly irregular.

The renal neoplasms were identified histologically as mesenchymal tumours. On microscopic examination the livers of all the rats revealed mild to moderate amounts of bile duct hyperplasia and varying numbers of nodules of hepatocytic hyperplasia. The cysts noticed macroscopically were severely dilated bile ducts, each lined by a single layer of flattened epithelium.

The animals in *Group 2* had received a diet containing 2,5 % of the kernel. Seven of the 8 rats were able to withstand the acute effects of the toxin for the 41 days that the experimental ration was fed, but one, Rat 12, succumbed on the 38th day. No significant changes were noticed in this animal at necropsy but histologically the liver was severely affected. It showed partial collapse of liver lobules, mild connective tissue increase, single cell necrosis of hepatocytes and severe bile duct hyperplasia and hepatocytic pleomorphism. Of the remaining 7 animals one, Rat 9, was killed on Day 275 and 6 died of their own accord between the 288th and 501st day. On macro- and microscopic examination 2 of these 7 animals (Rats 9 and 15) showed bilateral renal mesenchymal tumours and 3 (Rats 10, 13 and 14) similar unilateral tumours. These neoplasms varied in size from a few millimetres to 50 mm in diameter. Several small metastatic nodules of renal mesenchymal tumour were detected histologically in the lung of Rat 9. Furthermore, the livers of all of these animals were severely affected. Grossly the liver in several rats was swollen and had an irregular surface, numerous small cysts and pale to orange nodules. Microscopically the hepatic architecture was seriously disturbed by the presence of nodules of hepatocytic hyperplasia, bile duct hyperplasia and cystic bile ducts. In Rat 14 a small infiltrating hepatocellular carcinoma was also encountered.

Feeding of the ration containing 5 % kernel to the sole surviving rat in *Group 3* was stopped 30 days after the experiment had begun, the other 7 animals having expired between the 11th and 23rd days. This rat, however, died on the 36th day. In all these animals, apart from a generalized venous congestion and atrophy, the only organ showing prominent changes, in varying degrees of severity, was the liver. These changes comprised the following: a tendency to periacinar necrosis of hepatocytes which, however, was not extensive; necrosis of odd single and groups of hepatocytes, from early to late stages of this process being exhibited by the affected cells, and small areas of congestion sometimes associated with the periacinar necrosis. In some rats there appeared to be attempts at hepatocytic regeneration, with small groups of pleomorphic hepatocytes, which were smaller than normal hepatocytes, occurring within the lobules. Some were even arranged in a tubular pattern. The kidneys of many of the animals manifested cloudy swelling of the convoluted tubules.

All the rats in Group 3 lost body mass, the average mass per animal on Day 1 being 139 g and at death 83 g.

Acute death within 15-21 days characterized the disease syndrome of the animals in *Group 4* that had received 10 % *E. lanatus* kernel in their diet throughout the experimental period. In general, the pathological changes resembled those in the animals of Group 3 but were slightly more severe and acute. These animals also lost considerable body mass; the average mass per animal when feeding the experimental ration commenced was 127 g and at death 85 g.

The 8 animals in *Group 5* constituted the control group. One of them, Rat 34, died from pneumonia on the 510th day. The others were slaughtered on Day 546. No noteworthy lesions were detected on post mortem examination.

Fig. 5-12. Microphotographs of organs of rats that had received the kernel of *E. lanatus* in their diet.

Fig. 5. Renal mesenchymal tumour: an area containing dense spindle-shaped cells. Kidney of Rat 1. HE X480

Fig. 6. Renal mesenchymal tumour: an area where the cells are less dense than in Fig. 5. Kidney of Rat 15. HE X480

Fig. 7. Renal mesenchymal tumour: remnants of a kidney tubule deep in the neoplasm which is surrounded by tumour cells in a whorled pattern. Rat 1. HE X300

Fig. 8. Renal mesenchymal tumour: infiltration of the neoplasm into adjacent kidney tissue. Rat 14. HE X300

Fig. 9. Bile duct hyperplasia in liver of Rat 14. HE X120

Fig. 10. Cystic bile duct hyperplasia in liver of Rat 10. HE X75

Fig. 11. One nodule of hepatocellular hyperplasia abutting on another. Rat 10. HE X300

Fig. 12. Pleomorphic neoplastic cells in the hepatocellular carcinoma of Rat 14. HE X480

DISCUSSION

The results obtained in this experiment demonstrate the toxic and carcinogenic nature of the kernel of *E. lanatus* to rats. This is perhaps not unexpected as macrozamin had been determined in this cycad species[1]. Although only a small number of animals was used per group and one cannot therefore make unequivocal deductions, it does appear that in acute intoxications, as manifested by the animals in Groups 3 and 4, the liver is the principal target organ. It showed, basically, periacinar necrosis, single cell necrosis and early attempts at regeneration. The latter change, as well as the severe body mass loss of the animals concerned, is an indication that they had ceased to ingest the experimental rations some days before death once they had realised its toxic nature, and the lesions are not as severe as would have been the case had the plant material been dosed by means of a stomach tube. Fifteen of the 16 rats in Groups 1 and 2 which had received 1 % and 2,5 % of the plant material respectively in their rations for 41 days and had survived for relatively long periods thereafter, revealed severe kidney and/or liver lesions. These included kidney mesenchymal tumours in 8 animals, hepatocellular carcinoma in one, and hepatopathy manifested chiefly as nodular hepatocellular hyperplasia, bile duct hyperplasia and cystic bile ducts in varying degrees, in most of them.

It is well known that the kidney (adenoma, carcinoma, undifferentiated tumours, nephroblastoma, sarcoma, haemangioma and papilloma), liver (hepatocellular carcinoma, adenoma, cholangioma, cystadenoma, reticuloendothelial neoplasms, haemangioma and fibrosarcoma) and intestinal tract (adenoma, carcinoma and sarcoma) are the most sensitive to the carcinogenic activity of macrozamin. However, other tumours also associated with its administration in rats include glioma, meningioma, tumours of the ear canal, preputial gland adenoma, lung adenoma and schwannoma[16 18 46].

Table 3: NUMBER AND TYPE OF NEOPLASMS INDUCED IN RATS BY FIVE SOUTH AFRICAN *ENCEPHALARTOS* SPECIES[43 44]

Tumour	Plant	Part of Plant Fed		
		Outer Flesh & Kernel	Kernel	Outer Flesh
Renal mesenchymal tumour	*E. umbeluziensis*	5/6		
	E. villosus			1/2
	E. lebomboensis			1/4
	E. laevifolius		2/6	1/6
	E. lanatus		8/16	
Renal adenocarcinoma	*E. umbeluziensis*	1/6		
	E. laevifolius		1/6	
Cholangiocarcinoma	*E. umbeluziensis*	1/6		
Hepatocellular carcinoma	*E. villosus*	2/6		1/2
	E. lanatus		1/16	

In Table 3 a resumé of the neoplasms induced in rats following the ingestion of material from 5 different South African species of *Encephalartos* is given [43 44]. The predilection site for tumour development is dependent to some extent on the duration of feeding, hepatocellular carcinomas generally requiring prolonged administration of the carcinogen, cycasin whereas renal tumours develop after shorter periods[16]. Furthermore, the age of the animal when exposure occurs influences the relative frequency of various types of kidney tumours. Nephroblastomas, renal sarcomas and interstitial tumours are more common when immature rats are used, while renal adenomas appear to develop with about equal frequency in immature and mature animals. Neoplasms rarely develop before the age of 6 months irrespective of the age at first exposure. According to Laqueur & Spatz, the sex of the experimental animal used does not appear to influence its susceptibility to the toxin[21] but in the experiment described here only female rats were used.

A difference of opinion exists as to the precise classification and nomenclature of the neoplasm referred to above as the rat renal mesenchymal tumour. Hottendorf & Ingram consider that in rats both the neoplasm that has been named nephroblastoma and the renal mesenchymal tumour constitute the rat counterpart of the human nephroblastoma or Wilms' tumour[17]. This is in general agreement with the definition of human nephroblastoma proposed by Bennington & Beckwith who consider that "the traditional definition of nephroblastoma as a mixed renal tumour composed of metanephric blastema and its recognized stromal and epithelial derivatives at variable stages of differentiation, should be expanded to include as variants those monotypic neoplasms composed predominantly or exclusively of one of the histologic patterns common to typical mixed nephroblastomas of the kidney"[2]. Hard & Grosso, however, think that the rat nephroblastoma only corresponds to the epithelial component of Wilms' tumour in humans[11]. Rat mesenchymal tumour seems to correspond to the mesenchymal component of the same human tumour and, indeed, closely resembles the neoplasm classified as the congenital mesoblastic tumour. They stress that in their opinion these 2 tumours are separately occurring entities. In the classification of this type of renal neoplasm induced in the experiment described here and by Tustin[44] the opinion of Hard & Grosso was followed. The rat renal mesenchymal tumour is comprised of spindle cells organized into fibrosarcoma-like tissue, aggregations of stellate cells resembling primitive mesenchyme and well-developed reticulin network[11]. Spontaneously occurring neoplasms in the kidneys of rats are rare[11].

Nodules of hepatocytic hyperplasia have also been seen in rats that have received in their rations a mixture of cone axis and scales or the whole seed of *E. umbeluziensis*; the whole seed outer flesh of the seed, and kernel of *E. villosus*; outer flesh of *E. lebomboensis*; and kernel and outer flesh of *E. laevifolius*. It is well known that hepatocellular carcinomas may develop in some animals with hyperplastic liver nodules, which apparently was the case in Rat 14, Group 2. The development of hepatocellular carcinomas as well as other liver tumours has also been described in rats fed *E. umbeluziensis* and *E. villosus*[43] (Table 3) and the East African species, *E. hildebrantii*[24 26].

Although to date only 12 of the 28 species of *Encephalartos* occurring in South Africa have been proved to be toxic and/or carcinogenic when experimentally administered to animals or have been suspected, or known, to be poisonous when ingested by man, it should be assumed, until proved otherwise, that the remaining 16 species should be considered as potentially poisonous. Dyer has warned that it would be most inadvisable for humans to sample them[6].

ACKNOWLEDGEMENTS

This work was done under the auspices of the National Research Institute for Nutritional Diseases of the South African Medical Research Council. I wish to thank the Director of this Institute for generously providing the facilities for this work and the following for their assistance and interest: Drs S.J. van Rensburg, J.J. van der Watt, I.F.H. Purchase and R.A. Dyer; Messrs M. van Wyk, P. Lange, M. Hardy and R. Watermeyer; Mesdames J.F. Ueckermann and V. Käber; and the Department of Nature Conservation of the Transvaal Provincial Administration. The permission of the Director, Botanical Research Institute to reproduce the photographs of the cycads in the garden of Institute is gratefully acknowledged.

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COPPER DEFICIENCY IN PIGLETS CHARACTERIZED BY SPONGY MYELOPATHY AND DEGENERATIVE LESIONS IN THE GREAT BLOOD VESSELS

J.M. PLETCHER* and L.F. BANTING**

ABSTRACT: Pletcher J.M.; Banting L.F. **Copper deficiency in piglets characterized by spongy myelopathy and degenerative lesions in the great blood vessels.** *Journal of the South African Veterinary Association* (1983) **54** No. 1, 43-46 (En) Veterinary Research Institute, Section of Pathology, P.O. Box 12502, 0110 Onderstepoort, Republic of South Africa.

Copper deficiency was diagnosed in piglets from a farm in the Transvaal. The mortality rate among 5 to 8-week-old piglets had been sporadic but considerably high over a 12 month period. Affected animals showed signs of ataxia, posterior paresis, horizontal nystagmus, inability to stand and front and hind limb paddling movements. Death ensued from 3 – 5 days after the onset of clinical signs. A paucity of myelin in the spinal cord and degenerative lesions involving elastic fibres of the aorta and pulmonary artery were detected. Tissue copper levels were low in both clinically affected and unaffected piglets on the farm, and the copper content of the diet fed to sows and growing piglets was considered inadequate.

The lesions related to copper deficiency are described with emphasis on those in the spinal cord and great blood vessels, and the pathologic processes resulting in hypocupraemic lesions are considered.

Key words: Copper deficiency, pigs, nervous system, blood vessels, myelopathy, elastic fibre degeneration.

INTRODUCTION

Copper deficiency in lambs and kids is a well-recognized entity characterized clinically by ataxia, incoordination, stilted gait, inability to suckle and eventual death due to starvation.[3 9 15]. A similar syndrome of bovine neonatal ataxia associated with hypocupraemia in pregnant cows has recently been described[13]. Reports of naturally occurring and experimentally induced copper deficiency in pigs have, for the most part, centered on changes in the elastic component of the cardiovascular system, which often results in sudden death due to vascular rupture and haemorrhage[1 4 14].

This report deals with copper deficiency in piglets that died with prominent central nervous system (CNS) signs and spinal cord lesions similar to those reported in lambs suffering from copper deficiency. In addition, these piglets had gross and microscopical vascular lesions. Such findings suggest that copper deficiency, either simple or complicated by such nutritional factors as molybdenum, lead and/or inorganic sulfate, should be included in the differential diagnosis in cases involving piglets with central nervous signs.

HISTORY AND CLINICAL SIGNS

For over a year deaths occurred sporadically among 5 to 8-week-old Landrace piglets on a farm near Klerksdorp in the Transvaal. Although several months would pass without a loss, the problem invariably recurred. The farmer estimated that he had lost over 100 piglets. Some died without recognizable clinical signs, but most showed a variety of CNS symptoms including ataxia, posterior paresis, horizontal nystagmus, inability to stand and paddling leg movements. Affected piglets were afebrile and had tachycardia (230 beats per minute). Those that were able, ate and drank normally. Clinical signs gradually worsened, culminating in death after 3 – 5 days.

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Management on the farm was typical of many small pig-raising operations in South Africa. All pigs were fed skimmed milk and maize soaked in water. Fresh well water was provided and, occasionally, a small amount of lucerne was added to the diet. Sows and piglets were kept in concrete pens. At the request of the farmer, the state veterinarian (L. F. B.) visited the premises, recorded the clinical features of the disease, and conducted necropsies. Several weeks later, he visited again. At that time there were no clinically apparent cases. Samples of feed and water were obtained, and a clinically normal 5-week-old piglet was killed and necropsied. A post mortem examination was also conducted on a piglet of about the same age which had died suddenly the previous night. In addition, blood was collected from the clinically normal piglet and from a sow.

PATHOLOGICAL FINDINGS

Macroscopical Findings

The most consistent macroscopical lesions seen in both clinically affected and, to a lesser extent, unaffected piglets were multifocal areas of haemorrhage measuring 0.5 to 2.0 mm that were seen from the intimal surface of

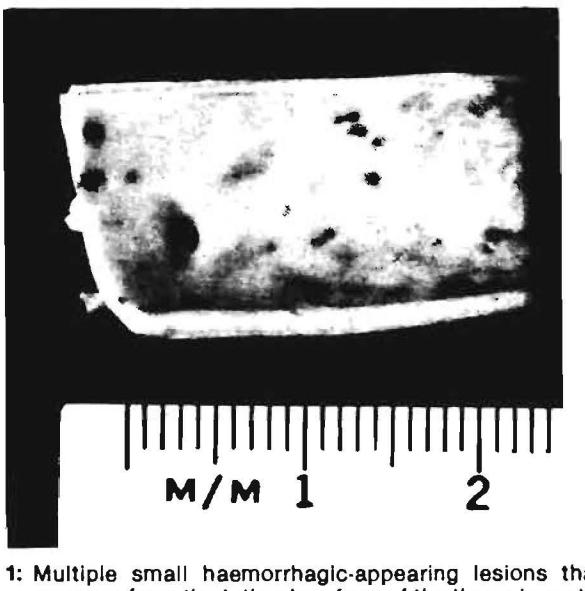

Fig. 1: Multiple small haemorrhagic-appearing lesions that are seen from the intimal surface of the thoracic aorta.

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the aorta and pulmonary artery (Fig. 1). These lesions were often slightly sunken, disturbing the usually smooth contour of the vessel wall. Lymph nodes adjacent to the thoracic and abdominal aorta were enlarged and reddish-brown in colour. All piglets that were necropsied had prominent costochondral junctions which were markedly pale and opaque, and several calluses denoting previously fractured ribs were observed. Other tissues and organs were macroscopically unremarkable with the exception of the peritoneal and pericardial serous membranes in the piglet which had died without clinical signs. A serofibrinous peritonitis and pericarditis was observed in this animal; these lesions were compatible with *Mycoplasma* or *Haemophilus* infection and were considered of primary importance in the death of this piglet.

Microscopical Findings

Selected tissues were preserved in 10% neutral buffered formalin, and representative samples were embedded in paraffin. Six-micron sections were cut and stained with haematoxylin and eosin (HE) and luxal fast blue (LFB) or a staining technique specific for elastic fibres. Tissue preparation and staining were carried out in accordance with commonly accepted methods.

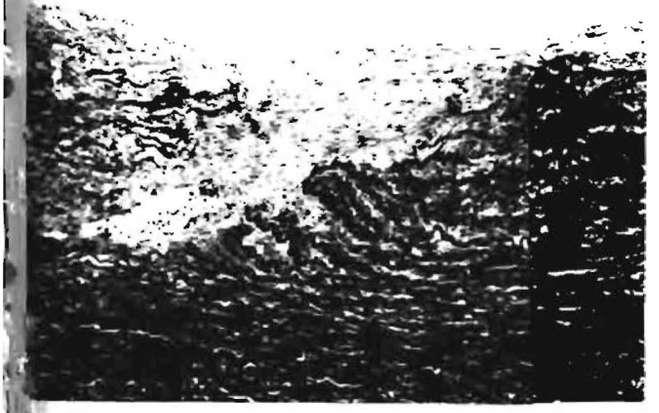

Fig. 2: Microscopic section through one of the macroscopic aortic lesions. Elastic laminae in the subintimal media are ruptured, and a cleft-like space has formed due to the separation of the weakened tissues. Note the proliferation of aortic smooth muscle cells. HE X 75

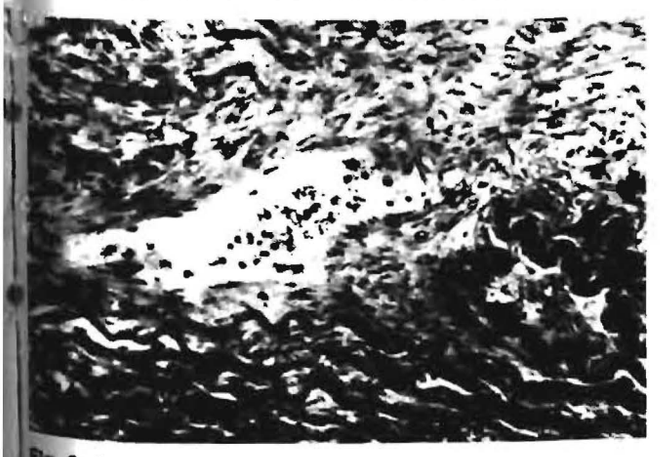

Fig. 3: Many cleft-like spaces such as this one containing erythrocytes, macrophages and homogenous eosinophilic material, were seen where elastic laminae were ruptured and separated. The proliferation of smooth muscle cells appears to be an attempt at repair. HE X 190

Microscopical lesions attributable to copper deficiency were seen in the spinal cords and in the walls of the aorta and pulmonary arteries. The haemorrhagic foci that were observed in the aortas provided excellent histologic evidence of the disease process in the great blood vessels. The gross vascular lesions corresponded to areas where elastic laminae were ruptured and separated. The separated ends of elastic fibres were often seen to be pale and frayed. Cleft-like pools of serum containing erythrocytes and macrophages were often seen where elastic laminae were ruptured. Increased numbers of smooth muscle cells formed sheets bridging the broken and separated ends of elastic fibres and surrounding the cleft-like spaces where aortic tissues had separated (Fig. 2 & 3). Often the axes of the smooth muscle cells were aligned at oblique angles to the concentric lamina of the vessel wall. These lesions disrupted the normal laminar anatomy of the aortic wall and often caused a shallow depression of the intimal surface of the vessel. Intramural haemorrhages were also seen. Lesions were most numerous and severe in the subintimal portion of the wall and became progressively less notable through the media to the adventitia. Erythrophagocytosis was evident among the fixed macrophages of the lymph nodes adjacent to the aorta.

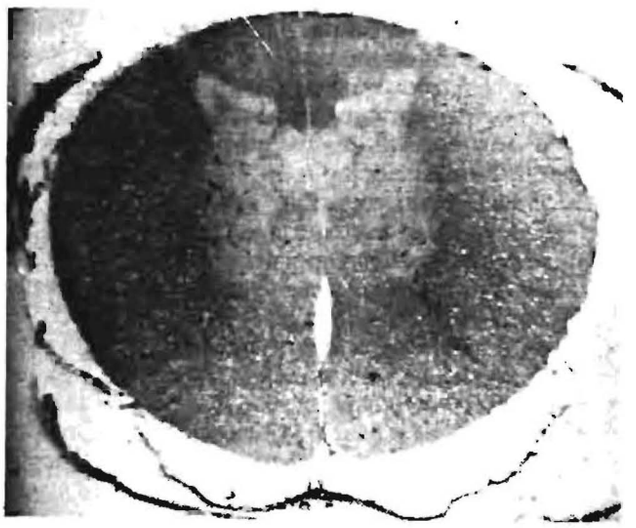

Fig. 4: This cross section of thoracic spinal cord reveals a moderate spongy myelopathy of the ventral and lateral tracts. The dorsal tracts are not affected. HE X 12

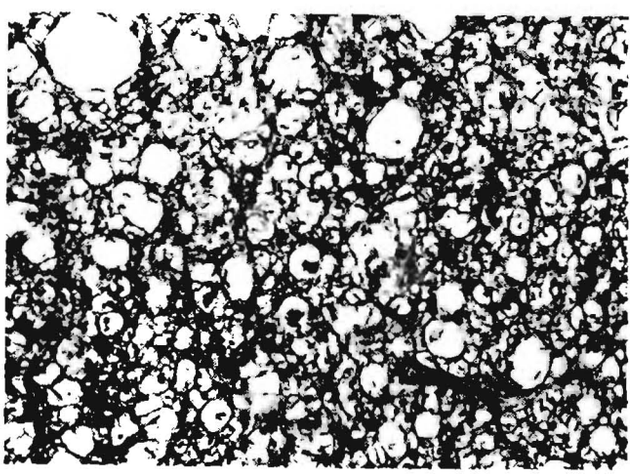

Fig. 5: Although myelin deficiency was often substantial in the ventral and lateral tracts, denuded axons appear normal, and there are no indications of myelin destruction or removal. LFB X 190

A spongy myelopathy was observed in the spinal cords of clinically affected piglets. A spongy appearance of the white matter was apparent throughout the length of the cord and was generally limited to the ventral and lateral tracts, with relative sparing of the dorsal funiculi (Fig. 4). Although the lack of myelin was marked, there was no evidence of inflammation or myelin destruction (Fig. 5). Neurons of both dorsal and ventral horns were not affected, and the axons appeared otherwise normal. Microscopical examination of the cerebrum, cerebellum, midbrain and medulla oblongata revealed no lesions. Central nervous system tissues of clinically unaffected piglets from the farm were unremarkable.

Clinical Pathological Findings

Chemical analyses were conducted on the livers of 2 clinically affected piglets (1 and 2), 2 clinically normal piglets from the farm which had elastic vessel lesions (3 and 4), and one control piglet (5). The results appear in Table 1 as parts per million (ppm) of respective trace elements on a wet basis.

Kidney copper levels in Piglets 3 and 4 were 2,6 and 2,5 ppm respectively, while kidney from the control piglet contained 5 ppm copper. Serum from Piglet 4 contained 0,5 ppm copper, and serum from a pregnant sow on the farm had 0,8 ppm copper. The copper content of feed and water from the farm was less than 1 ppm.

Haematologic findings in the sow and one piglet were unremarkable except for a mild microcytic, hypochromic anaemia ($5{,}0 \times 10^6$ mm^3) seen in the piglet.

DISCUSSION

Considering the results of gross and microscopical examinations as well as the chemical analyses, there is little doubt that the piglets described herein were experiencing a simple copper deficiency. Simple copper deficiency is relatively uncommon among domestic animals. Most copper deficient states are the result of nutritional factors which interfere with copper utilization. An excess of molybdenum, lead or inorganic sulfates in the diet of ruminants is known to produce clinical signs and lesions of copper deficiency[13 15]. Although not assayed in the feed and water of the piglets, the very low copper content in their diet renders these nutritional factors irrelevant.

The role of copper in mammalian physiology remains somewhat recondite; however, it is known that copper is an integral part of several enzymes or enzyme-like proteins. In copper deficient states, these enzymes decrease in activity, and should this biochemical lesion be sufficiently severe and prolonged, morphological lesions can result. The location and nature of such lesions are apparently dependent on the age and species of the deficient animal and the degree of copper diciency[15]. For example, a specific anaemia is often associated with copper deficiency in a variety of animals including piglets. This anaemic state is at least partly due to the reduced activity of the copper-containing globulin ceruloplasmin (ferroxidase), which is necessary for iron utilization in erythropoiesis[6 9 15]. Achromotrichia and physical changes in the hair and wool are likewise examples of lesions resulting from the reduced activities of copper dependent enzymes. These indications of copper deficiency are common among older cows and sheep grazing in pastures deficient in copper[9 15] but have not been described in pigs. No changes in hair keratinization or pigmentation could be discerned in the sows or piglets from the farm in this study, but their mild microcytic hypochromic anaemia seen in the only piglet sampled may reflect copper deficiency, since all piglets received iron injections at 2 weeks of age.

The association of copper deficiency and the rupture of major blood vessels in pigs was made nearly 20 years ago.[1 14]. A similar association was seen in domestic fowl[7 11]. Subsequent studies have documented a defect in blood vessel elastogenesis due to a decrease in intramolecular cross-links of the elastin polymer[16]. Appropriate molecular cross-linking only occurs if the copper-dependent enzyme lysyl oxidase is optimally active[8 12 15 16]. Major vessels and other arteries with defective elastic laminae are susceptible to dissecting mural haemorrhages, occlusion and/or rupture resulting in fatal haemorrhage or haemopericardium[4]. The rupture of elastic fibres, separation of tissues, mural haemorrhages and proliferation of smooth muscle cells observed in the elastic vessels of the piglets are virtually identical to the vascular lesions described in pigs with experimentally induced copper deficiency[4 14] with the exception that fatal rupture did not occur. Had the piglets not died at an earlier stage of CNS disease, advanced vascular lesions would probably have developed that would have resulted in rupture.

Spinal demyelination and/or hypomyelination resulting from copper deficiency is a well-recognized and consistent part of the syndrome in lambs and kids[3 9 15]. It has been reported in 4 to 6-month-old pigs as well, although it is not a common occurence[10 15]. Clinically apparent myelination defects in pigs, particularly young piglets, resulting from copper deficiency is an unusual finding and may reflect the severity of the deficiency on the farm. Cases of enzootic neonatal ataxia (swayback or enzootic ataxia) in lambs and kids often show, in addition to the spongy myelopathy, various CNS lesions including degenerative neuronal change in both the brain and spinal cord[3 9 15]. Although some of the clinical signs such as nystagmus and limb paddling suggest otherwise, myelin deficiency in the spinal cord was the only change observed in the CNS of the affected piglets.

The pathogenesis of the various CNS lesions associated with copper deficiency is still obscure; however, evidence is accumulating that suggests these lesions may also be the result of the reduced activity of copper-containing enzymes. Diminished cytochrome oxidase activity in neurons may explain the chromatolytic changes often seen in lambs and kids with the neonatal ataxia syndrome[2 3]. It is suggested that the lack of myelin observed in these cases may be the result of secondary demyelination[3]. Neither neuronal degeneration nor evidence of active myelin destruction were seen in the spinal cords of the piglets. Myelin deficiency in this instance may be due to myelin hypoplasia rather than secondary demyelination. Some experimental work suggests that lowered cytochrome oxidase activity in the livers of perinatal animals deficient in copper leads to depressed phospholipid synthesis, which in turn leads to the inhibition of myelin synthesis since phospholipids are the major component of myelin[15]. It is an attractive theory, but one is left with the difficulty of explaining how a failure to produce sufficient myelin can result in the spongy appearance of the spinal white

matter. Whatever the mechanism, it is certain that copper is essential to the formation and/or maintenance of myelin, and its effects are probably mediated through the activity of copper-containing enzymes.

Skeletal changes resulting from copper deficiency in dogs, rabbits, chicks, foals and pigs have been studied. The lesions observed are not unlike those of classic rickets, and bones are predisposed to fracture. The role of copper in osteogenesis appears to be related to the amine oxidase enzymes, which are necessary for the strength and stability of bone collagen and well as for proper elastic fibre formation[15]. The enlargement of costochondral junctions as well as evidence of previous rib fractures in the piglets that were necropsied could be further evidence of copper deficiency; however, considering the general inadequancy of the diet, it would be unwise to attribute such skeletal abnormalities to a deficiency of copper alone.

This occurrence of copper deficiency in piglets is worthy of notice for several reasons. First, it reveals that very young piglets suffering from copper deficiency can show CNS symptoms similar to those described in the neonatal ataxia syndromes of other domestic species. Secondly, it shows that both deficient myelination of the cord and degenerative vascular lesions can occur together in piglets, if the deficiency is severe enough and occurs at the appropriate time in development. Interestingly, no vascular lesions were found in a study of lambs with swayback[5]. Third, it provides documentation that, although myelin deficiency was marked, no lesions involving neurons were seen in the cord of the brain. This is in contrast to the neonatal ataxic syndromes of lambs and kids, where demyelination seems to occur subsequent to neuronal degeneration. Fourth, it alerts one to the fact that diets for pigs that are formulated without adequate consideration can easily be deficient in copper. And last, it emphasizes the significance of copper in a variety of seemingly unrelated but important physiological processes.

ACKNOWLEDGEMENT

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Table 1: CHEMICAL ANALYSES OF LIVERS OF AFFECTED PIGLETS (1 AND 2), CLINICALLY NORMAL PIGLETS (3 AND 4), AND A CONTROL PIGLET (5)*

Piglets	CA	Mg	Cu	Mu	Zn
1	42	175	5,8	4,7	60
2	40	175	6,2	4,9	65
3	29	160	2,2	4,3	64
4	51	125	1,8	4,3	62
5	34	192	39.6	4,6	75

* Results given in parts per million (ppm) on a wet basis.

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A COMPARISON OF THE EFFICACY OF ISOMETAMIDIUM, AMICARBALIDE AND DIMINAZENE AGAINST *BABESIA CANIS* IN DOGS AND THE EFFECT ON SUBSEQUENT IMMUNITY

C.G. STEWART*

ABSTRACT: Stewart C.G.; **A comparison of the efficacy of isometamidium, amicarbalide and diminazene against *Babesia canis* in dogs and the effect on subsequent immunity.** *Journal of the South African Veterinary Association* (1983) 54 No. 1, 47-51 (En.) Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

Isometamidium, amicarbalide and diminazene were used to treat experimentally induced canine babesiosis. Relapse parasitaemias developed after treatment in all groups of animals. The relapse interval, however, was shorter and more relapses occurred after treatment with amicarbalide than either of the other 2 drugs. Only half of the dogs treated with either isometamidium or diminazene relapsed to infection. Challenge with homologous parasites 62 days after initial infection resulted in severe babesiosis in all 3 animals which had not developed relapse infections. Of the 9 animals which had relapses after treatment only 1 developed severe babesiosis following homologous challenge.

Key words: *Babesia canis,* treatment, isometamidium, amicarbalide, diminazene, immunity.

INTRODUCTION

Dogs, which survive an initial acute infection with *Babesia canis*, either due to natural recovery or treatment, frequently develop patent parasitaemias at varying intervals following infection[1 2 3 7 9]. In many cases the dog may return to normal health although a mild parasitaemia may persist for as long as 2 years[7]. In other cases relapses may be associated with the development of clinical signs of babesiosis.

Acquired immunity to babesiosis has traditionally been regarded as co-incident with the presence of infection[4]. These parasites may or may not be seen in blood smears but they can be detected by inoculation of whole blood into susceptible animals[6]. Although Sergent[6] considered such "premunity" to be a prerequisite for good immunity, sterile immunity has been shown to be important in *B. canis* infections[5] and other *Babesia* spp[4]. Antibodies to a specific serum antigen occurring in the acute stage of canine babesiosis have been shown to be associated with immunity with a decrease of antibody titre being accompanied by fever, transient parasitaemia and anaemia[8]. Variable results, however, have been reported of the protective effect of *Babesia* antibodies against *Babesia* infection[10].

MATERIALS AND METHODS

Two litters of Beagles were obtained from the Beagle colony, Faculty of Veterinary Science, University of Pretoria. At 12 weeks of age they were vaccinated against parvovirus (Parvoid, Salisbury) and at 14 weeks of age against distemper, hepatitis and parainfluenza (Fromm, Salisbury). All dogs were sprayed weekly throughout the trial with oxino-thiophos (Bacdip, Bayer) to maintain them in a tick-free condition.

B. canis was obtained from Dr Potgieter, Veterinary Research Institute, Onderstepoort. This strain was obtained from a clinical case of babesiosis in 1975 and stored in liquid nitrogen. At 21 weeks of age Dog 8 was inoculated intravenously (iv) with 0,5 ml of this frozen material (Table 1). When parasites were fairly numerous in the blood smear, 20 ml of blood were collected in heparin and 0,5 ml of infected blood was then inoculated iv into the experimental animals. The remainder of the blood was frozen and stored in 1 ml ampoules in liquid nitrogen using dimethyl sulphoxide as a cryoprotectant. This material was used to challenge all dogs 2 months after the initial infection as well as an uninfected 8 week old Beagle which was used as a control (Dog 18).

Blood smears, haematocrit (Ht) and temperatures were recorded daily after infection in those animals which showed infection or a low Ht. When the parasitaemia in blood smears had converted to negative for *B. canis* and the Ht had returned to normal, blood smears were made from the animals 3 times a week for a few weeks and thereafter once a week. The ears were rubbed with zylene, pricked with a sharp instrument and the first drop of blood used to make a blood slide. The free-flowing blood was used to fill a micro-haematocrit tube which was then centrifuged in a Damon IEC MB centrifuge for 4 minutes and the Ht measured. All blood slides were stained with Giemsa.

The dogs were divided into 4 groups as follows:

Group I (4 animals): Treated with amicarbalide (Diampron, May & Baker) at 20 mg/kg intramuscualrly (im).

Group II (4 animals): Treated with a 1 % solution of isometamidium (Samorin, May & Baker) at 2 mg/kg im.

Group III (2 animals): Treated with diminazene diaceturate (Berenil, Hoechst) 4,2 mg/kg im.

Group IV (2 animals): A control group which was not treated until severe infection had developed.

The data are summarized in Fig. 1-4.

Animals in Groups I, II and III were treated when parasites were first seen in the blood smears and then at various stages during the relapse parasitaemia as shown in Fig. 1-4. The same dose of each drug was used to treat relapse infections as was used for primary infections.

The parasitaemia was estimated by counting the number of red blood cells in an average microscopic field and then counting the number of infected red blood cells seen in 10 fields. The percentage parasitaemia was then calculated. At least 200 microscopic fields were examined before a smear was classed as negative.

RESULTS

The results of treatment and homologous challenge are shown in Fig. 1-4.

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Group I (4 dogs treated with amicarbalide)

Treatment with amicarbalide was effective in preventing the development of a high parasitaemia with all 4 animals converting to negative parasitaemia within 48 hours. All animals relapsed 5-6 days after treatment with signs of listlessness. Low Ht combined with a high parasitaemia had developed. Two of these animals were then again treated with amicarbalide and the other 2 with isometamidium. Blood smears in all dogs were negative within 48-72 h after treatment. The 2 amicarbalide treated animals again relapsed 7 days later, whereas in the 2 dogs treated with isometamidium relapses were delayed 16 and 26 days after treatment respectively. A fluctuating parasitaemia developed in these dogs with improvement being followed by further relapses. Clinical signs were mild in Dogs 4, 13 and 15 and they recovered from these relapses without treatment. The relapse infection in Dog 3 developed into severe babesiosis with a Ht of 0,15 and the dog was treated on Day 35 with isometamidium. On this occasion a 0,5% solution was used. The drug did not cause pain on injection and no lump developed after injection. This dog then made a rapid recovery with its Ht returning to normal within 10 days.

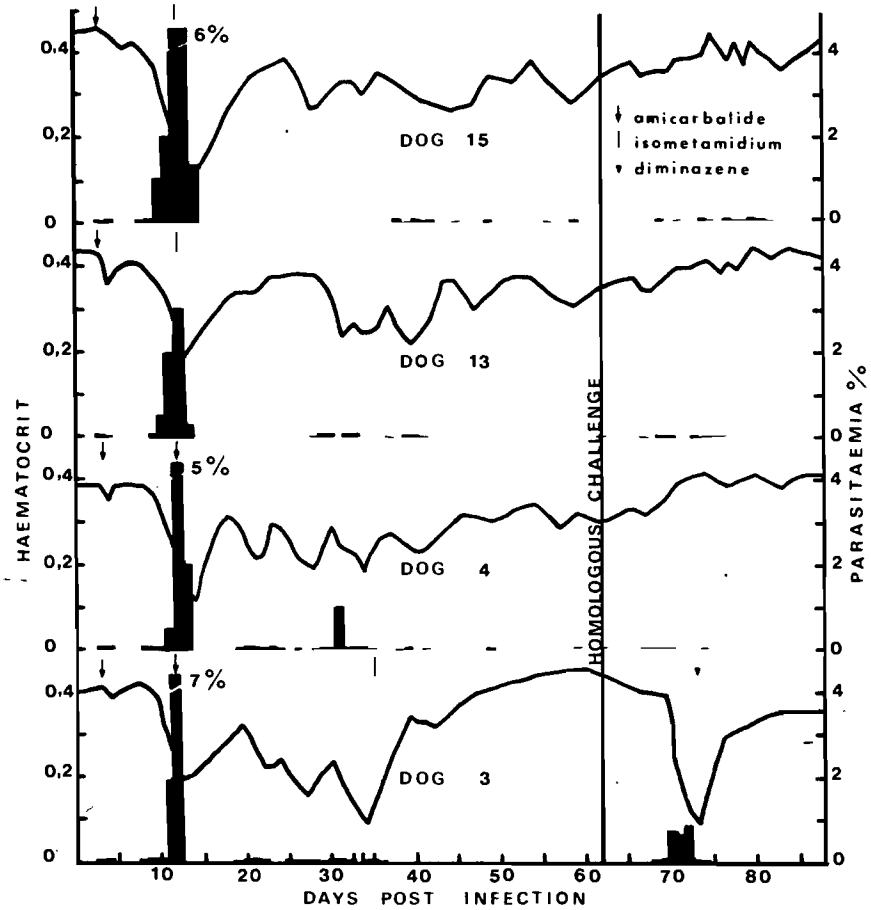

Fig. 1: The effect of treatment of primary infection of *B. canis* with amicarbalide and relapse infections with either isometamidium or amicarbalide and the effect on subsequent immunity. ■ Parasitaemia.– Ht

Group II (4 dogs treated with isometamidium)

Treatment with isometamidium was effective in preventing the development of a high parasitaemia. All smears were negative 48 hours after treatment. Considerable pain occurred on injection of the drug with all dogs yelping for about ½ minute after injection. A hard lump could be palpated in the leg muscle at the injection site for about 1 week after treatment. The blood slides from Dogs 12 and 14 remained free of parasites for 62 days when all dogs were challenged with homologous parasites. Dog 7 and 10 relapsed to infection 16 and 18 days after treatment respectively.

The relapse infection in Dog 7 was used to test the activity of a 0,5 % solution of isometamidium against chronic infection. The dog was showing clinical signs of babesiosis and had a Ht of 0,16 at this stage. It did not object to the injection however the following day is was lame and a hard lump could be palpated. The dog remained lame for the remainder of the trial. This may have been due to the fact that both treatments were injected into the same leg.

Group III (2 dogs treated with diminazene)

Treatment with diminazene was effective in preventing the development of a high parasitaemia with parasites being eliminated from blood smears within 48 hours. Dog 17 remained negative for the remainder of the trial whereas Dog 9 relapsed to infection 16 days after treatment. No further treatment was given to this dog and the first relapse was followed by a second relapse when the animal showed listlessness, bile stained faeces and a low Ht of 0,14. The animal subsequently improved and eventually recovered spontaneously. It lost a considerable amount of mass during this period.

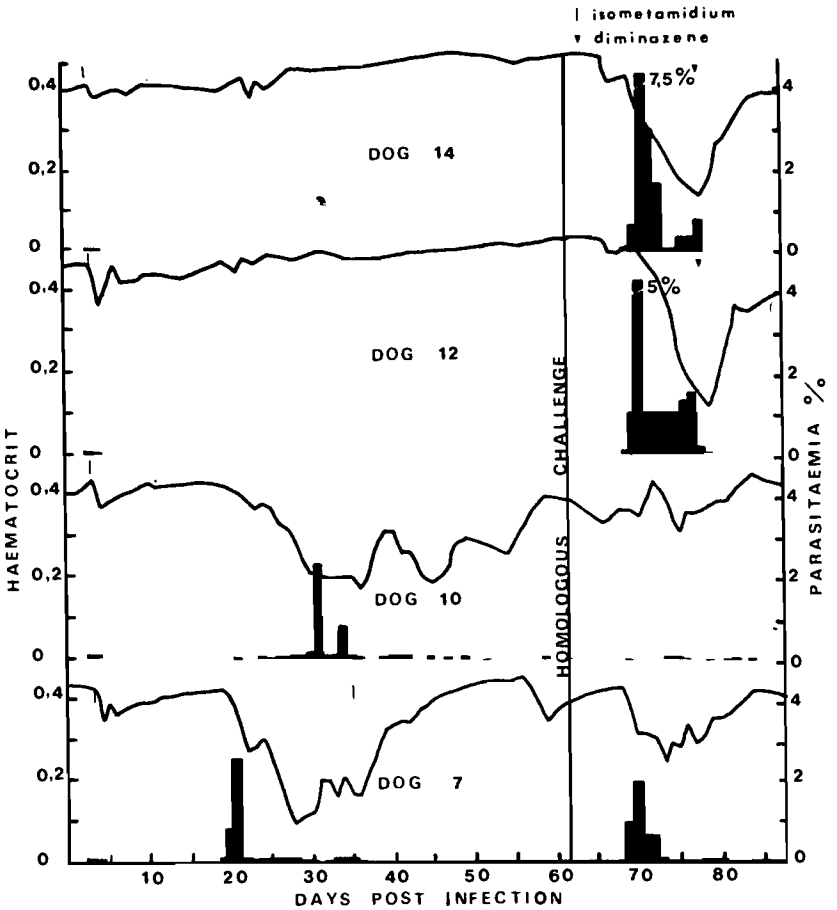

Fig. 2: The effect of treatment of primary and relapse infections of *B. canis* with isometamidium and the effect on subsequent immunity. ■ Parasitaemia – Ht

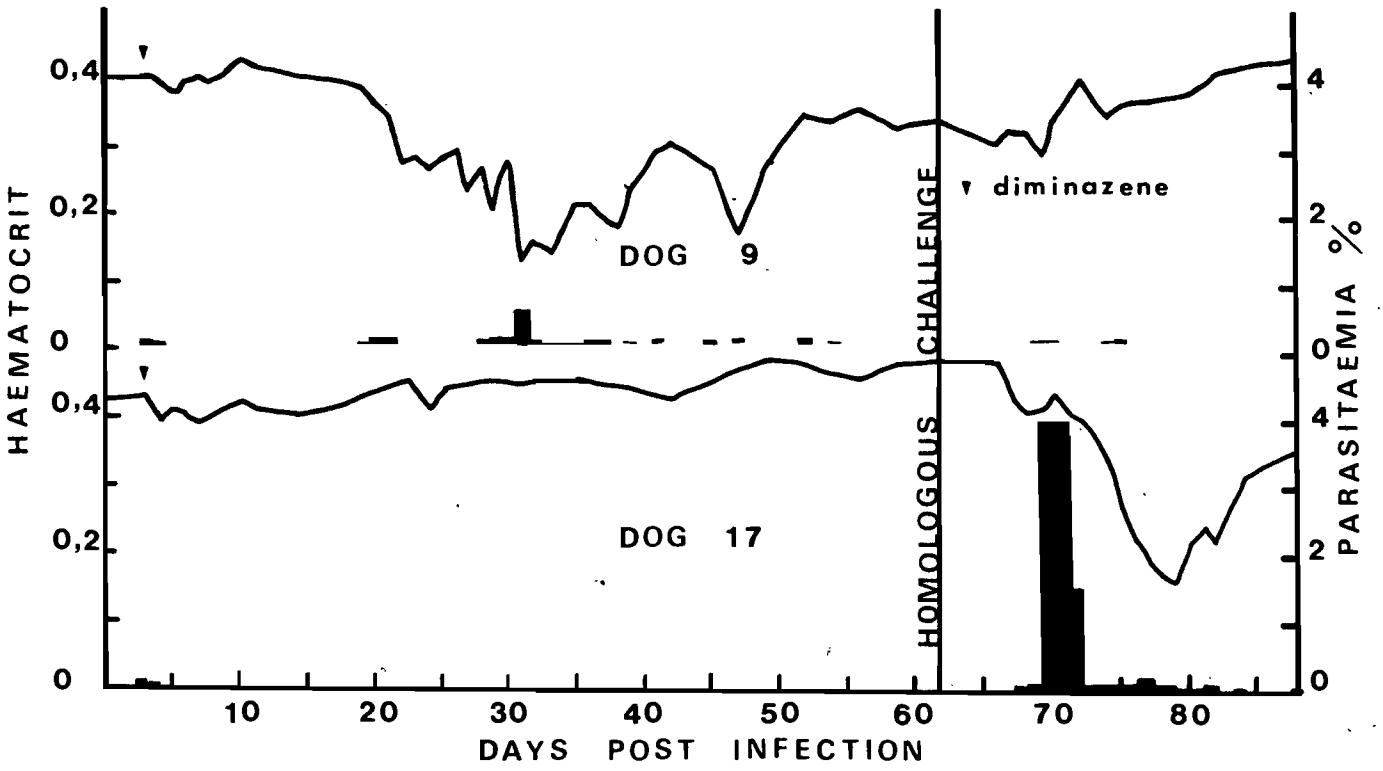

Fig. 3: The effect of treatment of primary infection of *B. canis* with diminazene and the effect on subsequent immunity. ■ Parasitaemia – Ht

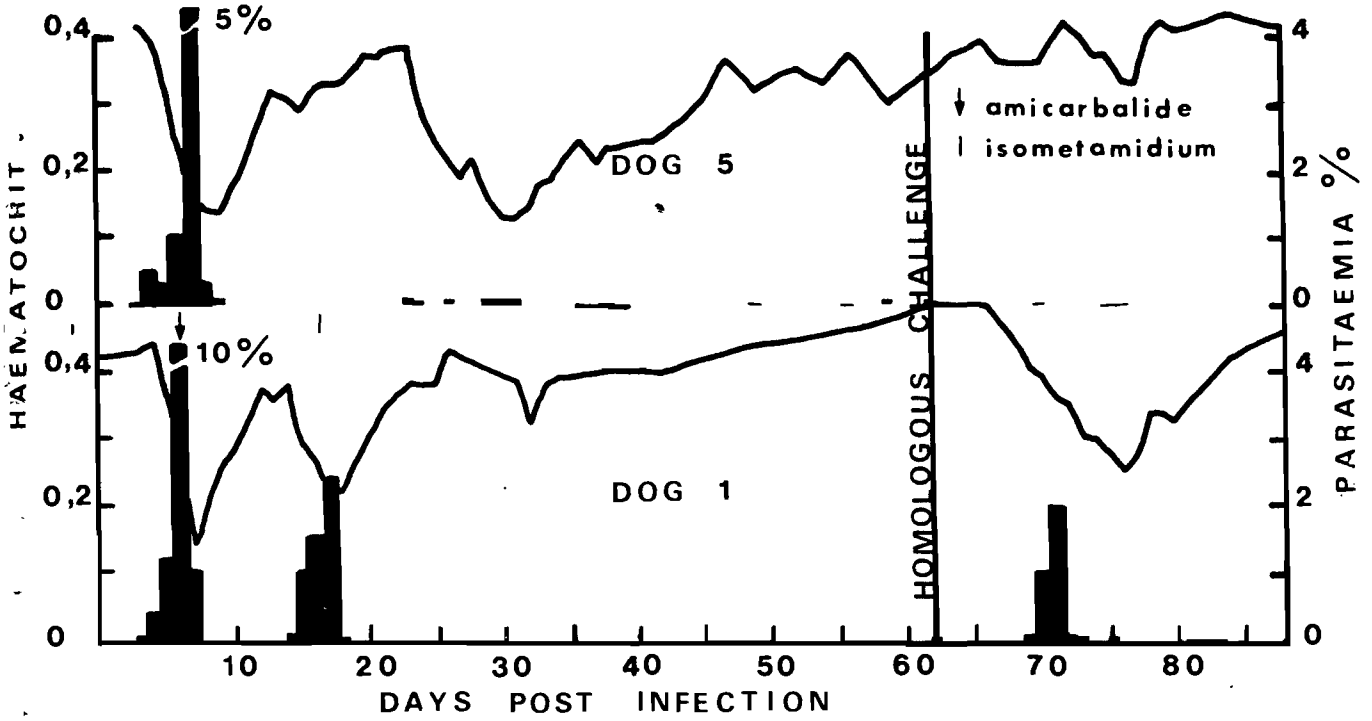

Fig. 4: Control group. Primary infection of *B. canis* treated to prevent mortality and the effect on subsequent immunity. ■ Parasitaemia – Ht

Group IV (2 dogs that acted as controls)

The control group developed a high parasitaemia of *B. canis* with a low Ht and clinical signs of babesiosis within 6 days of infection and both dogs had to be treated to prevent mortality. Dog 1 was treated with amicarbalide which eliminated the parasites from the blood slide within 48 hours. This dog relapsed to infection 8 days later and was treated with isometamidium, at 2 mg/kg by slow intravenous injection to prevent the pain associated with intramuscular injection. The dog collapsed within 1 minute after treatment, the pulse increased to 145/min and a mild watery diarrhoea and increased salivation was observed. Thirty minutes later the dog was able to stand and passed a large amount of foul smelling faeces. A brief relapse parasitaemia with a lowering of Ht occurred 14 days after the last treatment. Dog 5 was treated with isometamidium. Parasites were eliminated within 72 hours from the blood smear and a relapse infection occurred 16 days later which was not treated. The dog made a partial recovery and then again relapsed 6 days later. The dog was not treated and the Ht dropped to 0,13 before the animal again recovered spontaneously. At this stage it showed signs of listlessness, loss of mass with a typical water hammer pulse and bile stained faeces.

Control Dog 8

This animal was infected with *B. canis* which had been stored in liquid nitrogen for approximately 6 years. It developed peracute babesiosis and was found dead 9 days after infection. The day before it died, the dog was listless with injected mucous membranes but the Ht was still normal. This shows the virulent nature of the strain of *B. canis* used in this trial.

Control Dog 18

This dog showed parasites in its blood 6 days after infection. On the following day the parasitaemia was 2 % and the dog was showing clinical signs of babesiosis and a temperature of 39 °C. These signs were not severe and consisted mainly of listlessness with a typical water hammer pulse. The next day the animal was found dead. This confirms the peracute nature of this strain of *B. canis*.

Table 1: *BABESIA CANIS* INFECTION IN UNTREATED DOGS

Day Post Infection	Dog 8 Parasitaemia	Dog 8 Haematocrit	Dog 18 Parasitaemia	Dog 18 Haematocrit
3	-	0,48	-	0,40
4	-	0,48	-	0,41
5	-	0,48	-	0,42
6	-	0,48	+ + +	0,41
7	+ + +	0,40*	2 %	0,37
8	1 %	0,43	8 %	Dead
9	2 %	Dead		

* blood collected for challenge and storage in liquid nitrogen for homologous challenge.

Homologous challenge

All dogs developed a parasitaemia following challenge with homologous parasites 62 days after the initial infection with *B. canis*. Dogs 4, 5, 9, 10, 13 and 15 which had all had previous relapses developed low parasitaemias which were not accompanied by clinical signs or any significant decrease in Ht. Dogs 1 and 7 which had also had relapses showed a drop in Ht, but they did not develop obvious clinical signs and were able to recover without treatment. Dogs 12, 14 and 17 which had not developed relapses all developed severe signs of babesiosis after the challenge with listlessness, loss of mass, weakness, salivation, bile stained faeces and a

strong pulse. Dogs 12 and 14 were treated when their Ht were low and they were in obvious distress. Dog 17 in spite of the severe clinical signs and Ht of 0,16 was able to overcome the homologous challenge without treatment. Dog 3 represents an intermediate stage. It developed a relapse infection following the initial treatment however, in spite of this it also developed severe babesiosis with a Ht of 0,09 which had to be treated following the homologous challenge.

DISCUSSION

All the drugs used in this trial were able to prevent the development of severe babesiosis when given in the early stages of infection with the parasitaemia converting to negative within 48-72 hours. Relapses to infections were observed in all groups irrespective of the compound used. All dogs treated with amicarbalide relapsed to infection whereas in the case of isometamidium and diminazene only half the dogs treated developed relapse parasitaemias. The mean period between initial treatment and relapse infection was 5,3 days with amicarbalide, 17 days with isometamidium and 16 days for diminazene. The 3 dogs which did not relapse to infection failed to develop sufficient immunity to withstand challenge with homologous parasites 62 days after infection.

In most cases where relapse infections occurred the dog was able to reduce the parasitaemia only to relapse at a later date and then again recover. During these relapse infections dogs were not obviously sick and it was only when the Ht dropped below 0,20-0,15 that the dog would show obvious signs of babesiosis. It is probable that during relapse infections an owner would only notice a sick dog in the later stages of the disease and if left for any length of time could develop severe liver and kidney damage due to tissue anoxia making subsequent treatment difficult. Some dogs however, were able to overcome relapse infections without treatment. Dogs 4, 9, 10 and 13 developed high relapse parasitaemias (above 0,1 %) which they were able to overcome without treatment. These dogs had signs of babesiosis with parasitaemias ranging fro 0,5 to 4% and Ht ranging from 0,13-0,24, however, they all recovered without treatment.

Relapse infections in canine babesiosis appear to be important in the development of immunity. Dogs 4, 5, 9, 10, 13 and 15 which had all relapsed following initial treatment were able to withstand the challenge with homologous parasites 62 days after the initial infection. Parasites were seen in blood smears in all these dogs following challenge but this only lasted for a short time and was not accompanied by a marked drop in Ht.

In contrast to this Dogs 3, 12, 14 and 17 all developed severe babesiosis following challenge. In Dogs 12, 14 and 17 relapse infections had not occurred following the initial treatment. It is not known if in these 3 cases treatment resulted in sterilization of infection. If this did occur it may have limited the ability of these animals to develop a proper immunity. Relapse infections are however, not the only factor involved in development of immunity as shown by Dog 3. Although it had 2 relapses, both of which were treated, it still developed severe babesiosis after challenge.

Dog 1 and 7 represent an intermediate stage in that they both developed high parasitaemias following challenge and a significant drop in haematocrit values. These dogs were able to recover without treatment and the clinical signs shown were mild and could go unobserved by the average dog owner.

The effectivity of isometamidium and diminazene were very similar in this trial. Isometamidium, however, cannot be recommended for general use in dogs due to the severe pain caused by injection.

These results show that the immunity developed by dogs against babesiosis is poor and requires continuous stimulation of the immune system to maintain itself and even when this occurs dogs may still be susceptible to challenge. In these circumstances there may be some advantage in using a drug such as amicarbalide to allow the development of relapses so as to encourage stimulation of the immune system. This is associated with a certain amount of risk as owners may not observe a relapse infection until severe liver and kidney damage has occurred making subsequent treatment difficult.

ACKNOWLEDGEMENTS

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CASE REPORT **GEVALVERSLAG**

SUSPECTED VITAMIN E – SELENIUM DEFICIENCY IN TWO OSTRICHES

J. VAN HEERDEN*, S.C. HAYES** and M.C. WILLIAMS***

ABSTRACT: Van Heerden J.; Hayes S C; Williams M.C. **Suspected vitamin E-selenium deficiency in two ostriches.** *Journal of the South African Veterinary Association* (1983) **54** No. 1, 53-54 (En) Department of Medicine, Faculty of Veterinary Science, Medical University of South Africa, 0204 Medunsa.

Paresis of the limbs of two 4-month-old ostriches fed a diet predominantly of crushed maize was investigated. Raised levels of serum aspartate transaminase and creatine kinase were demonstrated in both birds. The less severely affected ostrich recovered after a single intramuscular injection of a vitamin E-selenium preparation but the other died despite therapy. An autopsy of the latter revealed focal pale areas in the thigh muscle. Microscopically affected muscle fibres showed degeneration, necrosis and regenerative changes. Fibrinoid degeneration and necrosis of some arterioles was observed as well as varying degrees of interstitial fibrosis. The above findings suggest a diagnosis of vitamin E-selenium deficiency.

Key words: Vitamin E-selenium, muscle, ostrich.

Two ostriches *Struthio camelus* (A & B) approximately 4 months of age which were kept in a small enclosure, developed clinical signs of paresis of their legs. Bird B developed extremely severe paresis of its legs and was unable to stand. Both birds were in very poor physical condition and were heavily infested with lice *Struthiolipeurus struthionis*. As far as could be established, the ostriches were fed on a diet consisting predominantly crushed maize.

Bird A was presented with moderate paresis of the leg muscles but could still support itself and walked short distances if it was raised from the sitting position. Whilst showing clinical signs, the bird had serum levels of aspartate transaminase (AST) and creatine kinase (CK) of 300U/*l* at 25 °C (German Society of Clinical Chemists) and 18,200 U/*l* at 25 °C (CK "NAC" activated (Boehringer)) respectively. The bird was treated with a single intramuscular injection of a vitamin E-selenium mixture (Bo-Se, Cyanamid). The ostrich made an uneventful recovery, regaining full use of its legs within 26 h after treatment.

Bird B developed severe paresis of its legs and was unable to support itself. Radiographic examination of the bones of the legs failed to detect any skeletal abnormality. Serum levels of AST and CK were 1600 U/*l* and 69600 U/*l* respectively. Treatment was commenced with the injection of 2 ml vitamin E-selenium mixture (Bo-Se, Cyanamid). The bird showed a marked improvement in muscular tone, but was still unable to stand. Treatment with vitamin E-selenium was repeated after 48 h and, in addition, a vitamin mixture containing thiamine, riboflavine, pyridoxine, nicotinamide and ascorbic acid (Parentrovite, Beecham) as well as good food and nursing were given. Despite these measures the bird died 5 d later.

An autopsy was performed on the dead ostrich. The only significant abnormality observed macroscopically was degeneration of the thigh muscles. This degeneration was seen as multiple pale streaky areas approximately 2 × 1 mm in size.

Six sections of formalin-fixed and haematoxylin and eosin – stained skeletal muscle were examined microscopically. Some sections showed virtually no lesions while in others changes varied from severe acute degeneration and necrosis to early regeneration. Many fibres were slightly to markedly swollen and showed a homogenous bright eosinophilic cytoplasm in which cross striations were retained. Scattered erythrocytes and a number of heterophils were present in the interstitium. Some of the arterioles were necrotic, a few showed fibrinoid degeneration, while the largest arterioles appeared normal. In some foci there was lysis of the muscle fibres, ingrowth of capillaries and a few fibroblasts accompanied by a small amount of immature collagen. In other areas quite marked fibrosis was evident (collagen bands 4 – 5 muscle fibres wide). In some sites muscle fibres were undergoing repair (proliferation of rows of sarcolemmal nuclei). There were a few very small areas of dystrophic calcification.

Histopathological examination of brain tissue failed to reveal any pathological changes.

Ideally the diagnosis of a vitamin E-selenium deficiency is confirmed by the measurement of glutathione-peroxidase levels in the serum[1].

In chickens, vitamin E deficiency is associated with encephalomalacia, exudative diathesis and myopathy[2]. It is not known whether the same response can be expected in ostriches. Vitamin E is very unstable in feeds and oxidative destruction is usually enchanced by minerals and unsaturated fatty acids in the diet. Unmilled cereals, vegetable oils and eggs are rich in vitamin E. The concentration of vitamin E in cereals may vary tremendously from one area to another[2].

It is postulated that these ostriches were suffering from vitamin E-Selenium deficiency. This diagnosis is supported by the clinical findings, the therapeutic response to a vitamin E-Selenium preparation, and to a lesser extent by the histopathological findings.

Capture myopathy has been suspected as a cause of mortality amongst ostriches[3]. However, there was no evidence of any stressful event being imposed upon the ostriches.

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CASE REPORT GEVALVERSLAG

LYMPHOSARCOMA IN A CAT

F. DE ST.J. VAN DER RIET*. R.M. McCULLY**, G.A. KEEN* and A.A. FORDER*

ABSTRACT: Van der Riet F. de. St.J.; McCully R.M.; Keen G.A.; Forder A.A. **Lymphosarcoma in a cat.** *Journal of the South African Veterinary Association* (1983) **54** No. 1, 57-59 (En) Department of Medical Microbiology, University of Cape Town. 7925 Observatory, Republic of South Africa.

Feline leukaemia virus (FeLV) antigen was detected in a cat from Cape Town which was subjected to euthanasia after a long history of upper respiratory infection and progressive emaciation. Based on the necropsy findings and subsequent histopathological examinations, a diagnosis of lymphosarcoma was made. It is regarded as likely that this tumour was caused by infection with FeLV.

Key words: Feline lymphosarcoma, cat, leukaemia, feline leukaemia virus.

INTRODUCTION

Feline leukaemia virus (FeLV) is transmitted horizontally between cats and often, if not always, participates in the aetiology of various forms of lymphosarcoma and leukaemia in this species[2 3 5 6]. In addition the virus is involved in the causation of several non-neoplastic diseases in cats. It may also have an immunosuppressive effect and in these cats secondary infection is common[6].

In recent years a serological test, the Leukassay-F test (Pittman-Moore (Inc.) for detecting FeLV infections in cats has become commercially available in South Africa[1] where it is being increasingly employed by veterinarians. We thought it would be of interest to record our findings from a case that occurred in Cape Town (a major cat breeding area in South Africa) and in which antigenic evidence of infections with FeLV was found using this test.

CASE HISTORY

The 10-year-old, spayed female Siamese cat belonged to one of us (GAK), who resides in Rondebosch, Cape Town. Prior to 1981 she had suffered episodes of respiratory infection but always recovered following antibiotic therapy. In May 1981 she developed severe pneumonia and purulent nasal discharge. She recovered from the pneumonia after treatment with antibiotics but the nasal discharge continued in spite of further treatment with penicillin, streptomycin, metronidazole and decongestants. *Bacteriodes fragilis* was recovered from the discharge. The cat became progressively thinner and as a malignancy was suspected it was subjected to euthanasia and presented for necropsy.

PATHOLOGY

Macroscopic findings

The cat was emaciated and there was a bilateral nasal discharge. Two lymph nodes at the angle of each mandible were bilaterally enlarged and each of the larger ones measures $25 \times 18 \times 12$ mm. Regional lymph nodes throughout the body were carefully examined but all others were found to be normal in size, appearance and consistency. There was purulent sinusitis of both frontal sinuses and some portions of the nasal turbinates appeared thickened and coated with a purulent exudate. Turbinates were collected along with other tissued in neutral buffered formalin for sectioning and the head was placed in a larger volume of formalin. After it was discovered on microscopic examination that the turbinates contained tumour cells, the nasal passage of the fixed specimen was carefully examined. Additional tumour tissue was recognized in the floor of the nasal passage and involved the lining of the posterior portion of the nasal cavity. It was poorly delineated so its exact extent was not determined. The lungs appeared normal. Except for the spleen, which contained several small white nodules 2-3 mm in diameter just beneath the capsule and deeper in the pulp, other organs and tissues, including bone marrow showed no gross pathological changes. There was a rather heavy tapeworm infestation with some catarrhal exudate in the small intestine. Body fluids (serum and urine) and fresh tissues (lymph nodes, brain, lung, nasal turbinates, liver, kidney, spleen) for serology and for freezing were taken.

Microscopic findings

Lymph nodes

There was complete effacement of the normal morphology of the enlarged lymph nodes from the angle of the mandibles. Diffuse, neoplastic growths of large histiocytic cells with only a few remaining small lymphocytes occurred throughout the parenchyma. The tumour cells had large round to oval nuclei with one, sometimes two, prominent nucleoli and confluent cytoplasm. There were numerous round spaces as though cells had dropped out, giving a starry sky effect. Other lymph nodes were unaffected.

Spleen

There were several small foci of tumour in the splenic pulp. The white pulp was generally atrophic. The tumour cells looked very similar to the histiocytes of the sheathed arteries due to their reticulum cell nature.

Nasal cavity

Between the epithelial covering and the underlying osseous fragments of the scrolls of the turbinates there were neoplastic cells identical to those described above. The tumour varied in size from only a few cell layers

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thick to massive infiltration which elevated the overlying epithelium. The neoplastic cells were insinuated between the various glands of the turbinates.

The mass of tumour tissue on the floor of the nasal passage was overlaid by low cuboidal cells, some of which were more flattened by pressure than others. The tumour cells which were similar to those described in the affected lymph nodes were growing in confluent masses or sheets but with no specific pattern.

Intestine

There was heavy tapeworm infestation, and this had caused a slight increase in mucus production.

Other tissues

Except for a small acinar cell adenoma in the pancreas, no neoplasia was found in the bone marrow or any of the other tissues including the brain, meninges, kidney and adrenal.

SEROLOGY

The results obtained in Leukassay-F tests of body fluids and crude tissue extracts from the cat for FeLV are presented in Table 1.

Table 1: EXAMINATION OF BODY FLUIDS AND CRUDE TISSUE EXTRACTS FOR FeLV ANTIGEN (LEUKASSAY-F TEST)

Body fluid or tissue extract	Result
Serum	+
Urine	±
Extracts (± 50 %) of	
Enlarged lymph nodes	+
Brain	±
Lung	+
Nasal turbinates	+
Liver	+
Kidney	+
Spleen (including a tumour focus)	+
Controls	
Positive reference	+
Negative reference	−
Extract medium	−

The extract medium was HANKS medium supplemented with 0,5 % lactalbumin hydrolysate and 0,35 % sodium bicarbonate.
+ = positive
− = negative
± = equivocal

BACTERIOLOGY

A moderate growth of *Pseudomonas aeruginosa* was cultured from the lung material and a mixed growth of commensals; including *Haemophilus influenzae* and *Staphylococcus epidermidis* from nasal turbinate material. Culture of pus from the frontal sinuses yielded a scanty growth of α-haemolytic streptococci. No bacteria were cultured from mandibular lymph node material or spleen.

ELECTRON MICROSCOPY

A few C-type virus-like particles were observed in ultra-thin sections of the nasal tumour (Fig. 1) but none were found in sections of the affected lymph nodes.

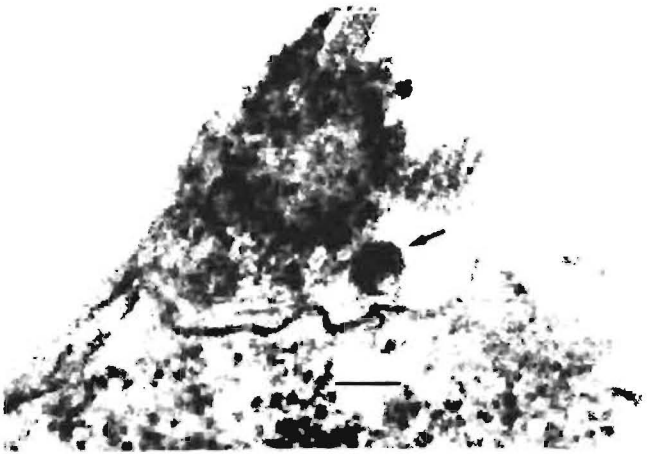

FIG. 1: C-type virus-like particle (arrow) in ultra-thin section of the nasal tumour. The scale bar represents 100 nm.

DISCUSSION

Based on both gross and microscopic pathology, a diagnosis of a histiocytic lymphosarcoma was made. It is presumed that the tumour growing into the nasal passage represents the primary and that the neoplasia in the regional lymph nodes and spleen was secondary as there was no generalized lymphoid involvement. In addition, the cat was severely emaciated with muscle wasting and showed evidence of a purulent frontal sinusitis and rhinitis.

As no blood samples had been taken during life it is uncertain whether the cat also suffered from a blood disorder. As lymphoid leukaemias or subleukaemias do not originate in the bone marrow but either arise from lymphosarcoma or begin as haematological dyscrasias with secondary involvement of solid tissue[4], the absence of malignant cells in the bone marrow does not exclude these conditions.

Evidence of FeLV in cats with lymphosarcoma is not always easily detectable[2,3]. In the present instance the FeLV antigen was found in the serum by the Leukassay-F test. Several organs were also positive but this may represent antigen present in blood or in lymphoid tissue in these organs. However, cells other than blood or lymphoid cells in the organs could also have been infected as FeLV replicates in epithelial cells in vivo[4]. Additional evidence for infection of the cat with FeLV was the observation of particles resembling C-type viruses in thin sections of the tumour.

The purulent sinusitis and rhinitis in the cat were most likely due to the irritation caused by the combination of the mechanical pressure by the tumour and by the accumulation of sinus and nasal secretions behind the partial obstruction of the nasal passage by the tumour.

The results of the bacteriological examination of the frontal sinus pus and nasal turbinates is of doubtful significance as the organisms cultured are frequently present in the nasal passage of healthy cats. As FeLV is immunosuppressive, it seems likely that immunosuppression could have played an additional role in the development of the pneumonia from which the cat recovered after treatment and in the development and persistence of the sinusitis and rhinitis. The progressive emaciation of the cat was probably due to a combination of malignancy, tapeworm infestation and chronic respiratory infections.

It is hoped that the reporting of this case of feline lymphosarcoma will increase awareness of South African veterinarians to FeLV virus infection and to the diseases caused by or associated with such infections. In particular this case emphasizes the advisability of conducting haematological examinations and tests for FeLV in cats with a long history of respiratory disease that may be secondary to lymphosarcoma and/or leukaemia.

ACKNOWLEDGEMENTS

We wish to thank Mr M. Emms for electronmicroscopy, Dr M. Hayes for his observations regarding current terminology concerning the pathological findings and Prof. J.W. Moodie for assistance in preparing the manuscript. We also thank Ethnor Laboratories, South Africa for supplying the Leukassay-F test kit.

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ABSTRACT: Potgieter, F.T. & Van Rensburg, L., 1982. **The effect of incubation and prefeeding on infected *Rhipicephalus simus* nymphae and adults on the transmission of *Anaplasma marginale*.** *Onderstepoort Journal of Veterinary Research*, 49, 99-101 (1982).

Two batches of unfed *Rhipicephalus simus* nymphae carrying *Anaplasma marginale* were incubated for 72 h and 96 h respectively at 37°C. Fifty ticks were triturated at a time and the homogenates were used to infect susceptible cattle. Others were prefed on a bovine host for 72 h before tick suspensions were prepared. The same procedure was followed, using a single batch of infected adult ticks.

Incubation had no effect on the infectivity of the nymphal homogenates, but prefeeding of nymphae reduced the prepatent period by 8 days in 1 of the 2 attempts. In the case of the adult ticks both incubation and prefeeding reduced prepatent periods by 5 and 8 days respectively.

ABSTRACT: Coetzer, J.A.W. & Ishak, K.G., 1982 **Sequential developments of the liver lesions in new-born lambs infected with Rift Valley fever virus. I. Macroscopic and microscopic pathology.** *Onderstepoort Journal of Veterinary Research*, 49, 103-108 (1982).

Ten new-born lambs were necropsied at various intervals after artificial infection with Rift Valley fever virus for the study of the sequential development of the hepatic lesions.

During the late stage of the disease, the livers were slightly to moderately swollen and mottled yellow, orange-brown and red. Greyish-white foci, approximately 0,25-0,5 mm in diameter, were also scattered throughout the parenchyma.

Microscopically, the liver lesions progressed from sparsely distributed acidophilic bodies and hepatocytes, revealing acidophilic degeneration and necrosis 6-12 h post-inoculation, to small randomly scattered primary foci of necrosis 12-24 h after infection. At 30-36 h. these primary foci were larger and more circumscribed. In addition, numerous acidophilic bodies and necrotic hepatocytes were dispersed throughout the markedly degenerated parenchyma. The terminal stage of the disease (48-53 h after inoculation), was characterized by massive hepatic necrosis in which primary foci of necrosis could still be recognized as dense aggregates of cytoplasmic and nuclear debris.

ABSTRACT: Coetzer, J.A.W., Ishak, K.G. & Calver, R.C., 1982. **Sequential development of the liver lesions in new-born lambs infected with Rift Valley fever virus. II. Ultrastructural findings.** *Onderstepoort Journal of Veterinary Research*, 49, 109-122 (1982).

The macroscope and microscopic lesions in livers of new-born lambs experimentally infected with Rift Valley fever virus and killed at various intervals between 6-53 h after inoculation, were described in a previous paper. This communication gives an overview of the ultrastructural changes affecting hepatocytes, sinusoids and spaces of Disse, biliary tree and portal triads as well as observations on the morphology and morphogenesis of the virus.

Hepatocytes were those primarily affected, while inflammatory and architectural changes were secondary. The changes included prominent nuclear alterations, fragmentation or disintegration of necrotic hepatocytes, focal cytoplasmic degradation and sequestration, and the presence of acidophilic bodies. The ultrastructure and origin of the intranuclear inclusions are discussed.

ABSTRACT: Pollard, B., 1982 **Immune response to the simultaneous vaccination of day-old chickens with live and inactivated oil-based Newcastle disease vaccines.** *Onderstepoort Journal of Veterinary Research*, 49, 123-125 (1982).

The immune response, as measured by the haemagglutination-inhibition test, to the simultaneous administration of live Hitchner B1 and 2 commercially available, inactivated, oil-based, emulsified, Newcastle disease vaccines at day-old is described. The response was monitored from day-old to 18 weeks, when the birds were challenged with a standardized virulent virus. It was found that the haemagglutination-inhibition titre fell below $\log_2 5$ when the chicks were 10 weeks of age. Challenge at 18 weeks yielded a mortality rate of 25% in the groups receiving both live Hitchner B1 and an oil-based vaccine in comparison with 94% in the group receiving Hitchner B1 alone.

Simultaneous application of live and oil-based vaccines at day-old is conclusively insufficient to maintain adequate protection until 18 weeks and it is recommended that a booster vaccine be administered at 10 weeks.

ABSTRACT: Myer, M.S., 1981. **The presence of Paneth cells confirmed in the pig.** *Onderstepoort Journal of Veterinary Research*, 49, 131-132 (1982).

The presence of Paneth cells, traditionally believed to be absent in the crypts of the domestic pig, *Sus scrofa* (Linnaeus, 1758), has been confirmed in this report. The cells were found approximately 104 cm from the pyloric valve and occurred mainly along the sides and in the lower half of the crypts of Lieberkühn.

CASE REPORT **GEVALVERSLAG**

LIMFOSARKOOM AS 'N RAAR OORSAAK VAN REKTALE PROLAPS IN DIE HOND

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

ABSTRACT:Odendaal J.S.J.; Cronje J.D.E. **Lymphosarcoma as a rare cause of rectal prolapse in the dog.** *Journal of the South African Veterinary Association* (1983) **54** No. 1, 61-62 (Afrik.) 152 Benade Drive, Fichardt Park, 9322 Bloemfontein, Republic of South Africa.

A case is presented of a dog with a lymphocytic lymhposarcoma of the rectum without systemic involvement. The dog showed bloody stools and rectal prolapse. The diagnosis and treatment is discussed.

Key words: Lymphosarcoma, lymphoma, dog rectal prolapse.

INLEIDING

Limfosarkoom (kwaadaardige limfoom) is 'n bekende gewas in honde. Die siekte kom wêreldwyd voor en is die eerste keer in Duitsland in 1872 gediagnoseer.

Volgens Thielen & Madewell[1] het limfosarkoom in 0,36 % van 2 763 honde voorgekom in 'n opname wat in 'n Weense veterinêre kliniek gemaak is. Met 'n opname wat in New York opgestel is, is die gewas in 0,2 % uit 10 000 honde aangeteken terwyl in Alford, Frankryk het 1,4 % van die nadoodse ondersoeke en 0,13 % van die kliniese diagnoses, limfosarkoom getoon. Dit wil voorkom of die insidens van limfosarkoom hoër is in manlike diere en in honde tussen 7 en 10 jaar ouderdom.[1]. Tydens 'n studie in 146 honde met limfosarkoom, is die gewas in Labrador Retrievers, Duitse Herdershonde, Poodles, Boxers en Bassets in die volgorde van voorkoms aangeteken. Alle honderasse is egter vatbaar vir limfosarkoom. Volgens Theilen & Madewell[1] het die dermkanaalvorm van limfosarkoom in honde, slegs 6,9 % uit 'n totaal van 144 gevalle, uitgemaak.

Die oorsaak van limfosarkoom in die hond is nog steeds onseker.

GESKIEDENIS

'n Vyfjarige Basset reunhond is verwys met 'n geskiedenis dat die ontlasting vir 'n geruime tyd af en toe bloederig vertoon het. Later het die hond herhaaldelik rektale prolaps ontwikkel as gevolg van

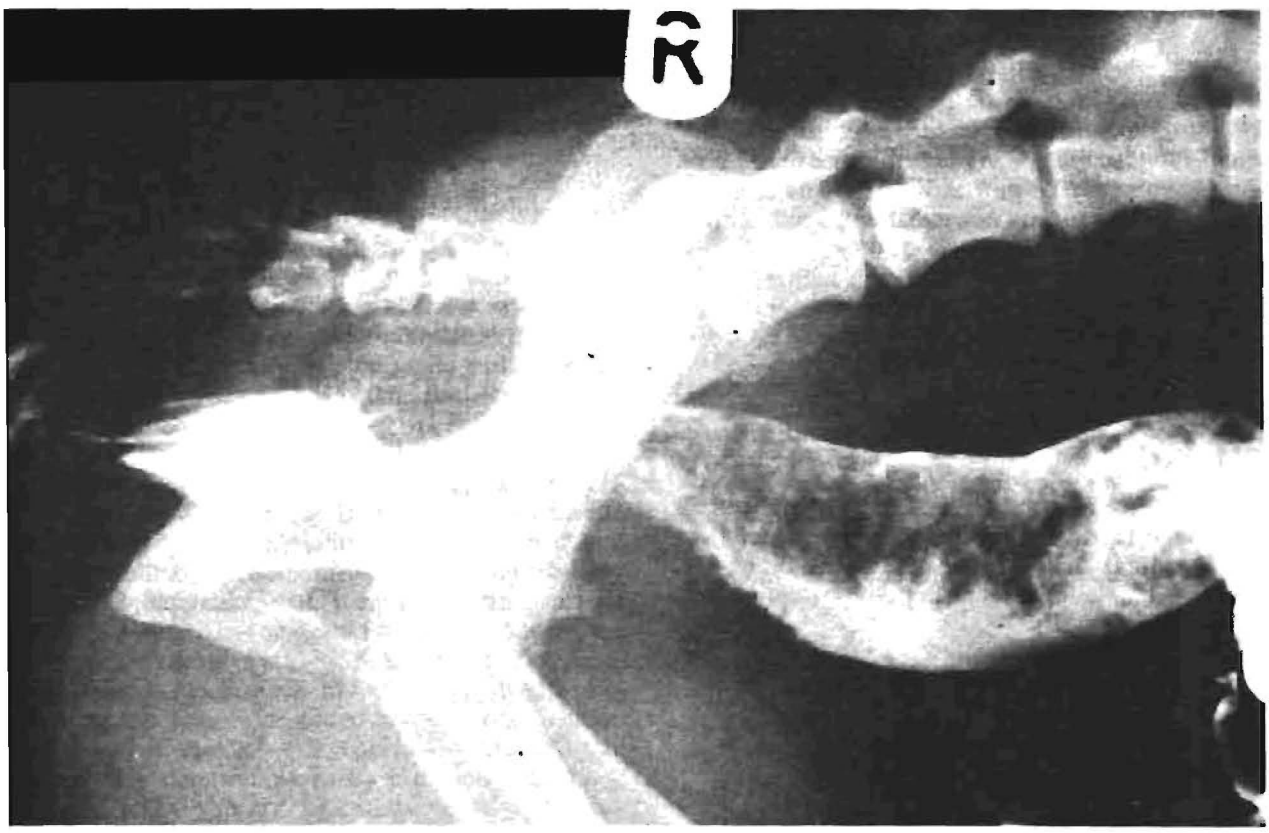

Fig. 1. Röntgenfoto wat die vernouing van die rectum met gepaardgaande proksimale verwyding van die col-on aandui

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parsing. Volgens 'n veearts is die prolapse 4 keer chirurgies gereduseer. Behalwe vir bogenoemde afwyking was die aptyt van die hond normaal, sy kondisie goed en het lewendig en belangstellend voorgekom.

Om die parsing te verminder is die hond op 'n meer vloeibare dieët geplaas wat tot 'n mate die rektale prolapse verminder het. Daar was geen geskiedenis van koorsaanvalle of gewigsverlies nie.

KLINIESE ONDERSOEK

Met rektale ondersoek kon 'n verdikking net-net gevoel word weens die anterior lokalisering van die gewas. Geen prolaps was op hierdie stadium teenwoordig nie. Met abdominale palpasie kon 'n massa van ongeveer 20 mm by 20 mm gevoel word. Daar is toe besluit om barium röntgenfoto's van die dermkanaal te neem. 'n Vernouing in die rektum met gepaardgaande proksimale verwyding van die kolon kon waargeneem word (Fig. 1).

Die foto is opgevolg met proktoskopie wat op 100 mm 'n gladde verdikking in die rektale mukosa getoon het. Tydens die proktoskopie is 'n biopsie van die verdikte rektale slymvlies geneem vir histopatologiese ondersoek. Die ondersoek van die dier onder narkose het geen verdere afwykinge getoon nie. Geen vergrote limfkliere kon palpeer word nie.

DIAGNOSE

Die histopatologiese ondersoek het getoon dat die mukosa van die rektum erg geinfiltreer is deur limfosiete wat selfs deur die spierlae van die tunika muskularis gestrek het en hulle verplaas het. Op grond van die mikroskopiese bevindings is 'n diagnose van 'n limfosietiese limfosarkoom gemaak.

BEHANDELING

Aangesien slegs dié massa in die rektum tydens die ondersoek gevind is, is daar besluit om die area te bestraal en dit op te volg met chemoterapie.

Radioterapie

Die radioterapie het 8 dae geduur en het bestaan uit 2 bestralings van 800 Rads fotone elk met 'n 6 MeV Siemens versneller. Die bestraling is ventraal toegedien oor 'n area van 180 mm by 90 mm op die buik, met die dier onder algemene narkose om posisionering te vergemaklik.

Chemoterapie

Tydens die eerste bestraling is 4 mg/kg metielprednisiloon (Depo Medrol, Upjohn) binnespiers, en 1 mg vincristine sulfaat (Pericristine, Petersen) binne-aars toegedien. Behandeling met 1 mg vincristine sulfaat (Pericristine, Petersen) binne-aars, 10 mg doksorubisien hidrochloried (Andriblastina, Chemfarma Laboratories) binne-aars, 250 mg siklofosfamied (Endoxan, Noristan) binne-aars, en 15 mg bleomisiensulfaat (Blenoxane, Bristol) binnespiers is tegelyke tyd met die tweede bestraling toegedien.

Antibiotika terapie is toegepas om sekondêre infeksies te beheer.

Eers 6 dae nadat die laaste behandeling toegepas is, het die hond newe-effekte begin toon. Die simptome was vomisie, aptytverlies, dehidrasie en effense diaree. Vomisie het 2 dae geduur en die verlies van eetlus 4 dae. Die newe-effekte is suksesvol tydens hospitalisasie simptomaties behandel met anti-emetika, aarvoeding en tonikums.

Sedert die eerste behandeling het die parsing en die rektale prolapse nie weer voorgekom nie. Nadat die newe-effekte van die behandeling oorkom was, het die hond ongekompliseerde herstel getoon. Die oorlewingstyd op hierdie stadium sedert eerste diagnose is 302 dae. Op die oomblik lyk die hond klinies gesond en leef 'n normale lewe.

BESPREKING

Limfosarkoom van die dermkanaal van die hond is 'n skaars gewas. Die posisie in die rektum is blykbaar raar, veral as 'n enkele letsel. Die letsel is as primêr beoordeel omdat die hond, buiten die probleme in die rektum, geen kliniese tekens van kanker getoon het nie. Volgens Theilen & Madewell[1] toon honde met die dermkanaalvorm van limfosarkoom kakeksie, vomisie, diaree, tekens van wanvoeding en gewigsverlies. Met abdominale palpasie kan vergrote mesenteriese limfkliere en vergrote massas gevoel word. Geen van bogenoemde simptome was teenwoordig nie. Hierdie pasiënt het slegs parsing getoon met gevolglike herhaaldelike prolapse weens die verdikking in die rektum.

Aangesien die eienaar van die hond ongeveer 150 km vanaf die kliniek gewoon het, is die behandelingsregime vir praktiese doeleindes so eenvoudig as moontlik gehou. Die newe-effekte as gevolg van die behandeling was minder as wat verwag is en die vomisie en aptytverlies wat die ergste simptome was, is hoofsaaklik toegeskryf aan die chemoterapie. Oormatige haarverlies het nie voorgekom nie.

BEDANKINGS

Ons dank aan Drs A. Frazer en A. de Vos van Ladybrand wat die geval na ons verwys het en die Superintendent van die Nasionale Hospitaal Bloemfontein, om te publiseer.

VERWYSING

1. Theilen G.J.; Madewell B.R. 1979 Veterinary Cancer Medicine. Lea Philladelphia p 241-250

SHORT COMMUNICATION KORT MEDEDELING

RESPONSES OF UNANAESTHETISED AND PENTOBARBITONE-ANAESTHETISED SHEEP TO A LETHAL DOSE OF SUCCINYLDICHOLINE

C. BUTTON and MARIA S.G. MÜLDERS*

ABSTRACT: Button C.; Mülders M.S.G. **Responses of unanaesthetised and pentobarbitone-anaesthetised sheep to a lethal dose of succinyldicholine.** *Journal of the South African Veterinary Association* (1983) **54** No. 1, 63-64 (En) Department of Physiology, Pharmacology and Toxicology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

A lethal dose of succinyldicholine was administered to 3 unanaesthetised and 4 pentobarbitone-anaesthetised, non-ventilated sheep. When compared to the unanaesthetised sheep, the anaesthetised sheep had smaller increases in arterial and central venous blood pressure, blood glucose and plasma catecholamines. It was concluded that the difference in response between the groups could largely be ascribed to conscious perception of asphyxia in the awake group with resulting fear.

Key words: Succinyldicholine, sheep, anaesthesia, game cropping.

INTRODUCTION

The controversy surrounding the use of succinyldicholine (succinylcholine, SDC) for the cropping of game animals in the Republic of South Africa has been discussed in detail in an earlier report[1]. Massive catecholamine release was described in conscious, non-ventilated calves paralysed by SDC. A question unanswered by this report was whether the catecholamine release was an expression of fear or whether it was largely an autonomic reflex. In the present trial we attempted to eliminate the element of fear in sheep by anaesthetising them before injecting a paralysing dose of SDC. The responses of these sheep were compared with those of a group of SDC-paralysed but unanaesthetised sheep.

MATERIALS AND METHODS

The subjects were 2 groups of Merino sheep, all approximately 1 year old. Two days before each trial began, silastic catheters were implanted in the carotid artery and jugular vein using a technique described previously[1]. Unfortunately, the catheters pulled out of one sheep, leaving 7 in the trial. The first group, comprising 4 sheep, were anaesthetised with pentobarbitone sodium to stage III, plane III anaesthesia. Blood pressure was recorded on a physiological recorder (Siemens-Elema Mingograf, Model 62) as follows: the sheep were placed in right lateral recumbency and pressure transducers (Statham P50) were connected by means of 3-way valves to the arterial and venous catheters. An electrocardiogram was recorded using the same recorder. After a stabilization period of approximately 10 minutes control blood pressures and electrocardiograms were recorded and venous blood samples were taken for plasma glucose and catecholamine determinations by previously described methods[1]. Next, 3 mg/kg body mass of succinyldicholine chloride (Scoline, Glaxo) was injected into the gluteal muscle mass while blood pressure and electrocardiogram were recorded continuously. Additional blood samples were drawn at 2 and 5 minutes after the SDC injection for glucose and catecholamine determinations. The recorder was switched off at about 10 minutes after the SDC injection, by whch time the sheep had died.

The remaining 3 sheep were subjected to the same procedure as above except that they were given no anaesthetic and were manually restrained in lateral recumbency. They were then given the SDC injection and blood samples were drawn at the same times as above.

RESULTS

All sheep died as a result of the 3 mg/kg injection of SDC. Increases in both systolic and diastolic blood pressure (Fig. 1) and in mean central venous pressure (Fig. 2) were greater in the unanaesthetised than in the anaesthetised group of sheep. Similarly, blood glucose

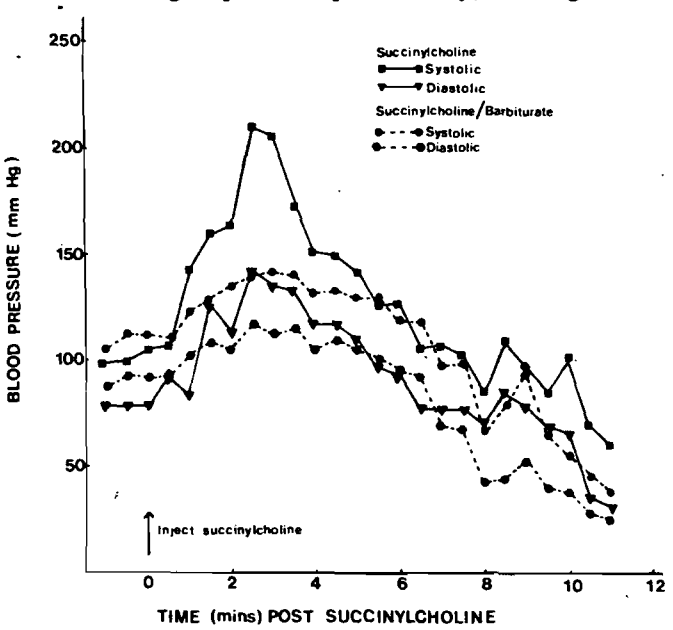

Fig. 1: Systolic and diastolic blood pressures in unanaesthetised and in pentobarbitone-anaesthetised, non-ventilated sheep after injection of succinylcholine.

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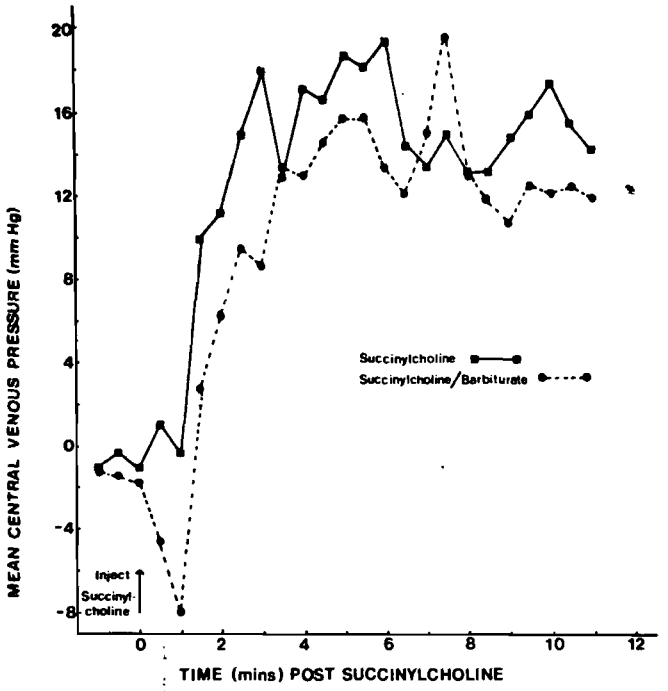

Fig. 2: Mean central venous blood pressure in unanaesthetised and in pentobarbitone-anaesthetised, non-ventilated sheep after injection of succinylcholine.

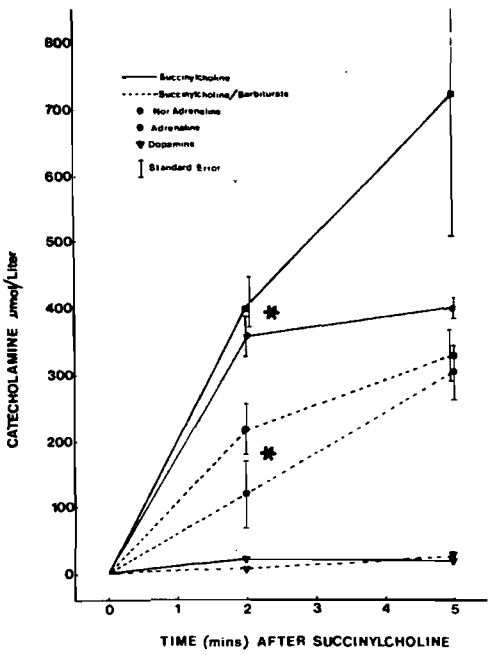

Fig. 4: Mean ± standard error of plasma nor-adrenaline, adrenaline and dopamine in unanaesthetised and in pentobarbitone-anaesthetised, non-ventilated sheep after injection of succinylcholine. The asterisks denote probably significant differences (P<0,1).

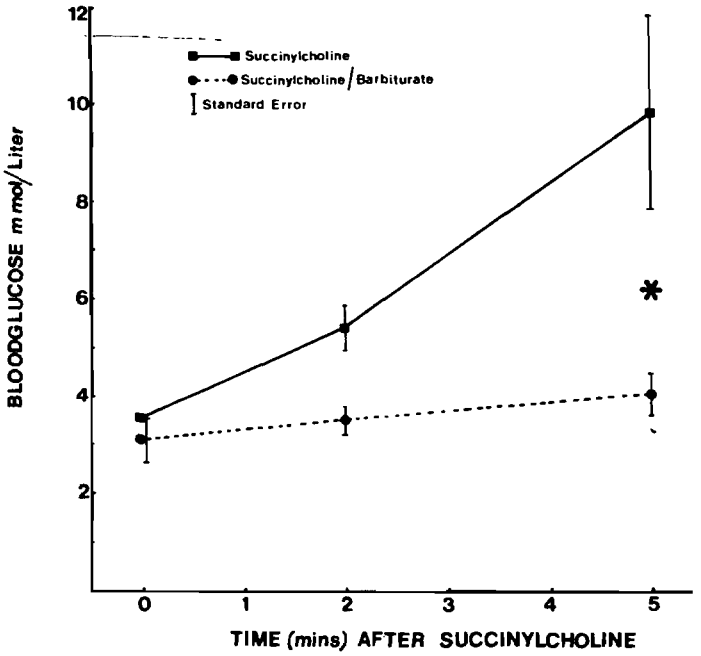

Fig. 3: Mean ± standard error of blood glucose in unanaesthetised and pentobarbitone-anaesthetised, non-ventilated sheep after injection of succinylcholine. The asterisk denotes a probably significant difference (P<0,1).

concentrations at 2 and 5 minutes post SDC injection were greater in the unanaesthetised group (Fig. 3). The difference at 5 minutes was probably significant (P = <0,1; paired t test[2]). Similarly, the plasma nor-adrenaline and adrenaline concentrations in the unanaesthetised group were greater at 2 and 5 mintues than the anaesthetised group (Fig. 4). The differences in nor-adrenaline and adrenaline in the groups at 2 minutes were probably significant (P = <0,1).

DISCUSSION

The inability to communicate directly with animals forces one to resort to indirect methods when trying to determine an animal's mental state. The inability to ventilate while conscious is, in man, a terrifying experience and it would be reasonable to assume that the same applies to other mammals. In this trial an attempt was made to gauge the degree of asphyxia-related fear in an unanaesthetised group of sheep by comparing plasma catecholamines and related parameters (blood glucose, blood pressure) in these sheep to the same parameters in a group of sheep in which conscious experience of fear had been abolished by general anaesthesia.

The increase in plasma catecholamines and glucose and blood pressure in the anaesthetised group can be ascribed to reflex autonomic activity in response to asphyxia. The greater increases in the unanaesthetised group can probably be ascribed to an awareness of the asphyxial process and of the resulting fear response. This study supports our findings in calves and underlines reservations felt in some quarters concerning continued use of SDC for killing or immobilizing game.

ACKNOWLEDGEMENTS

The autors thank Prof. H J Bertschinger for performing the catecholamine assays and Dr D.G.A. Meltzer for his help with the statistical analyses.

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SHORT COMMUNICATION **KORT MEDEDELING**

THE EFFECT OF NUTRITIONAL STRESS ON THE PLASMA PROGESTAGEN LEVELS AND EMBRYONIC MORTALITY IN TWIN PREGNANCIES OF MARES

C.H. VAN NIEKERK*, J.C. MORGENTHAL* and CYNTHIA J. STARKE**

ABSTRACT: Van Niekerk C.H.; Morgenthal J.C.; Starke C.J. **The effect of nutritional stress on the plasma progestagen levels and embryonic mortality in twin pregnancies of mares.** *Journal of the South African Veterinary Association* (1983) **54** No. 1, 65-66 (En) Department of Human and Animal Physiology, University of Stellenbosch, 7600 Stellenbosch, Republic of South Africa.

Two thoroughbred mares bearing twins as diagnosed by rectal palpation, were subjected to nutritional stress by drastically reducing the daily ration. This resulted in a marked reduction in plasma progestagen levels and the death of either one or both of the twin embryos.

Key words: Pregnant mare, twins, nutritional stress, progestagens, embryonic death.

INTRODUCTION

Although the incidence of twinning as recorded in practice is only 1-5 %, observations on slaughtered mares have shown that as many as 20 % of all pregnancies begin as twins[1]. It is, therefore, possible to assume that resorption of one of the twin embryos occurs in the majority of such pregnancies. It is also significant that the abortion or birth of twins are mostly seen in maiden and barren mares while the incidence of twinning in mares with foals at foot is considerably smaller[2]. This observation is consistent with our own experience in this respect, as well as the tendency for twins to be more prevalent in studs where a high plane of nutrition is maintained.

The fact that a drastic decrease in quantity and quality of feeding can induce a large percentage of early embryonic resorption was described as long ago as 1965 by Van Niekerk[3], and this observation was subsequently applied successfully by Merkt & Klug[2], to bring about the resorption of one of the embryos in twin pregnancies. After positively diagnosing twin pregnancies by rectal examination between 20 and 47 days of pregnancy, they reduced the ration by about half for 14 days, and out of 16 mares so treated, 11 eventually produced one foal each, 4 aborted or produced twins and the remaining mare lost both embryos during treatment.

Recently van Niekerk & Morgenthal[4] found that stressful conditions like pain, infectious diseases, emotional disturbances such as weaning and the administration of exogenous corticosteroids cause a severe fall in plasma progestagen concentration in pregnant mares, and suggested that such circumstances could play a major role in the occurence of pregnancy failure in the thoroughbred mare.

The purpose of this experiment was to determine whether the application of controlled nutritional stress, in an attempt to reduce twins to singletons, would have a similar depressive effect on plasma progestagen concentration as other stressful conditions.

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MATERIALS AND METHODS

The two cases of twinning on which this report is based were encountered in a thoroughbred stud where every mare is routinely examined per rectum for pregnancy on Days 20, 45 and 60 of gestation.

Mare A was barren during the preceeding season while Mare B had a foal at foot at the time of treatment. In both cases the presence of twins was diagnosed on Day 45 of pregnancy, which was confirmed on Day 68 in the case of Mare A and Day 55 with regard to Mare B. Both mares received a ration of oat hay ad lib., supplemented daily by 8 kg of a balanced concentrate containing 16 % protein. Treatment comprised the complete withdrawal of the concentrate for a period of 10 days starting on Days 78 and 55 with respect to Mare A and B respectively, with oat hay remaining available ad lib.

Jugular vein blood was collected weekly for at least 2 weeks before the beginning of treatment, and daily thereafter for the first 4-5 days. This was followed by a decreased frequency of blood collection, as indicated in Fig. 1. Unconjugated progestagens were determined by radioimmunoassay as previously described[4].

RESULTS AND DISCUSSION

At the beginning of treatment the progestagen concentration of Mare A was more than 4 times higher than that of Mare B, whose hormone levels were considered to be within the expected limits that are usually to be found in mares carrying singletons at that stage of pregnancy (J.C. Morgenthal, unpublished results). It is our experience that while mares with twins are generally inclined to show considerably higher levels of plasma progestagens than those with single foetusses, it only becomes apparent after 100 days of pregnancy in the majority of cases.

The plasma progestagen concentration of both mares dropped sharply after withdrawal of the concentrate component of their ration (Fig. 1). Weekly rectal examinations revealed that Mare A lost one foetus during this period of decreasing hormone levels within 14 days of the beginning of treatment. The event was followed by a transient rise in hormone concentration, after which a progressively downward trend ensued over

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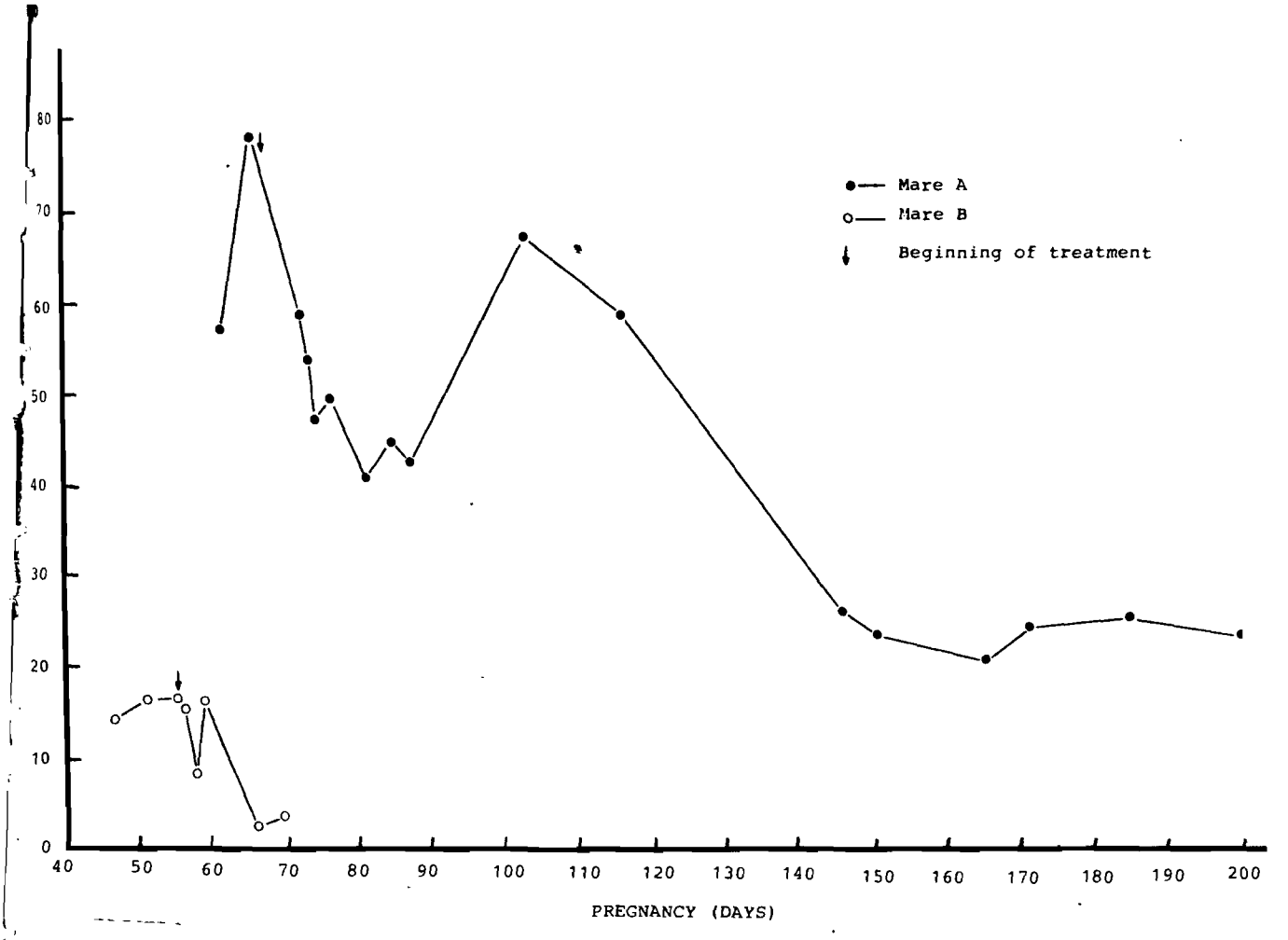

Fig. 1: The effect of nutritional stress on the plasma progestagen concentrations of two mares with twin pregnancies.

about the next 40 days until it finally consolidated at levels that are considered normal for a mare with a single conceptus. Mare A eventually gave birth to a single foal at normal term.

In Mare B the hormone concentration responded similarly to treatment by dropping rapidly to a very low level of 2 ng/ml 11 days after concentrate withdrawal, during which she lost both conceptuses.

These findings indicate that nutritional stress has the same effect on the progestagen concentration of the pregnant thoroughbred mare as the other kinds of stress that were reported earlier[4], and confirm that it can induce embryonic resorption in the process. It is apparent, however, that the treatment applied in this experiment was too drastic for a twinning mare suckling a foal, where the resultant stress is compounded by the physiological demands of lactation which is instrumental in the death of both conceptuses.

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SHORT COMMUNICATION **KORT BERIG**

THE DANGER OF IMMUNISING BOERGOATS AGAINST HEARTWATER

B. GRUSS*

ABSTRACT: Gruss B. **The danger of immunising Boergoats against heartwater.** *Journal of the South African Veterinary Association* (1983) 54 No. 1, 67-68 (En) P.O. Box 12573, 0110 Onderstepoort, Republic of South Africa.

Ten 6-toothed Boergoat does and 215 Boergoat kids were injected by the intravenous route with the Onderstepoort heartwater blood vaccine and then temperatured daily. On Day 10, all the does had rectal temperatures in excess of 40°C and were treated with oxytetracycline intravenously, while the kids received an intramuscular injection of the antibiotic. One kid died from heartwater on Day 11. On Day 15, 7 does had a febrile reaction and all the does and kids were treated as before. Two does died of heartwater on Days 16 and 17 respectively, while 12 kids developed heartwater on Day 17 and 6 kids on Day 18. All were treated for heartwater. A total of 2 does (20%) and 16 kids (7,4%) died during the experiment.

Key words: Heartwater, goats, *Cowdria ruminantium*, dipping, pyrethroid dips, immunisation.

INTRODUCTION

The Boergoat is considered a good source of red meat in the coastal belt of the Eastern Cape and bushveld of the immediate interior. The animals are well adapted to this region, especially in areas of dense bush where Angora goats do not thrive owing to the entanglement of their hair in the bush which has caused many animals to die from hunger and thirst.

The Boergoat is also an excellent symbiont with cattle ranching in the coastal bushveld, where the grass increment is higher than that in the valley bushveld, as they do not compete with cattle for their food while at the same time, bush encroachment is checked. A further advantage is that the red meat production is thus increased by a considerable amount. These animals are not only more resistant to the inclement weather than Angora goats, but their breeding season is throughout the year, in contrast with that of the Angora goat which breeds only in the late summer and autumn.

Unfortunately, however, these animals have been found to be highly susceptible to heartwater, and prior to the advent of the pyrethroid dips, great losses were experienced by farmers who farmed with Boergoats. This discouraged many farmers from running Boergoats. For this reason it was decided to attempt to immunise a group of Boergoats in the Uitenhage district with blood infected with *Cowdria ruminantium* by the method described by Thomas & Mansvelt[1].

MATERIALS AND METHODS

Ten 6-toothed Boergoat does which had aborted or had been marked for culling were selected from a flock comprising a total of 250 does and 215 kids ranging in age from 3 – 6 weeks. They were ear-tagged and considered as the pilot group. The does had all been previously inoculated against enterotoxaemia, *Pasteurella multocida* and *P. haemolytica* and, immediately before commencement of the experiment, they were drenched with morantel citrate (Thelmesan, Repvet). Only kids over 4 weeks of age had been inoculated against enterotoxaemia and *Pasteurella* spp. and drenched with Lintex (Bayer) for tape worms.

For immunization purposes it was decided to use blood infected with the Ball 3 strain of *C. ruminantium* which is issued by the Veterinary Research Institute, Onderstepoort. The frozen heartwater blood was obtained on a Friday afternoon from the Veterinary Research Institute, packed in an insulated carton of dry ice and transported by road to Port Elizabeth over the weekend. The blood was received at noon on the following Monday, when the carton was replenished with dry ice and then placed in a commercial deep freezer at about -20°C from the Monday until the Thursday when the blood was administered to the animals concerned.

The 10 does in the pilot group each received 5 ml and the kids 2½ ml of the thawed blood intravenously (iv).

The does and 215 kids were then placed with the rest of the flock in the bush. They were dipped in a belly dip containing a pyrethroid and organophosphate (Sumifleece, Shell chemicals) every 10 days. The dipping regime commenced 2 months before the trial was instituted and continued throughout its duration. The whole flock was kraaled at night. At about 05h30 every morning for 18 days, the 10 does, i.e. those in the pilot group, were temperatured and examined for the presence of ticks, and thereafter were allowed to join the others in the bush. All animals that died during the course of the trial were necropsied and squash smears of their hippocampi were prepared according to the method of Purchase[2] and examined microscopically for the presence of *C. ruminantium*.

RESULTS

Ten days after infection all the does had rectal temperatures in excess of 40°C. They were all given oxytetracycline (Dabicycline 150 mg/ml, Chemveld) intravenously at a dosage rate of 10 mg/kg while the kids received the same antibiotic at the same dosage rate, but by the intramuscular (im) route. On Day 11, post infection, the temperature of all the does had returned to normal, but one kid showed typical signs of heartwater and died the same day. Unexpectedly on Day 15, 7 of the does once again manifested fever reactions as before, and the does and kids were again treated with the antibiotic. On Day 16 one doe died of heartwater

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and another on Day 17. Twelve kids were sick on Day 17 and were treated with oxytetracycline, and an antiepileptic drug, clonazepan (Rivotril, Roche) at a dosage rate of 0,5 ml by the iv route and 0,5 ml by the intramuscular route. Nine kids died of heartwater that day. On the following day, 8 other kids had heartwater and were treated as before, but 7 died that day. After Day 18, there were no more losses and the daily temperaturing was stopped. During the period of the experiment there were no losses in the rest of the flock. The diagnosis of heartwater was confirmed in all cases that died at necropsy and by examination of the hippocampal smears. Only a few sporadic deaths from heartwater occurred during the 2 months prior to commencement of the trial.

To sum up: 2 does in the pilot group (20%) and 17 (7,4%) of the 215 kids died from heartwater.

DISCUSSION

It is evident from the results of this experiment that any attempt to immunise Boergoats with the present "infect and cure" method may be costly and disastrous. All these goats were born and lived in an enzootic heartwater area. Considerable losses had occurred among this specific flock prior to the advent of the pyrethroid dips. It was originally expected that very few of the does would react to the immunisation with the Ball 3 strain of *C. ruminantium,* and such high losses were totally unexpected. These results are totally different from the results obtained with similar experiments in Angora goats where very few of them reacted to infection with the Ball 3 strain and no losses occurred (B. Gruss 1981. Unpublished work).

It can be concluded that the Boergoat is one of the most susceptible ruminants to heartwater, that some are refractory to treatment, and that with our present knowledge, Boergoats should be protected from ticks by the use of a pyrethroid dip, rather than by immunisation against heartwater. The Boergoat may even be an excellent model for research in the development of a safe vaccine.

ACKNOWLEDGMENTS

I thank Mr. C.J. Pietersen of the farm "Doornkom", Uitenhage for his assistance in this experiment, as well as for carrying the losses with the author; and Shell Chemicals, Port Elizabeth for donating the Sumifleece dip.

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BOOK REVIEW — BOEKRESENSIE

VETERINARY APPLIED PHARMACOLOGY AND THERAPEUTICS

G.C. BRANDER, D.M. PUGH, R.J. BYWATER

4th edn. Baillière Tindall – Cassel. London SW1P 1SB. 1982 pp VII and 582, figures 34, numerous diagrams, Price R58,50 (ISBN 0 7020 08710)

This 4th edition of Brander and Pugh's well-known text is aimed primarily at the veterinary student. The book comprises 4 introductory chapters and 19 chapters devoted to systematic pharmacology. There are chapters on the autonomic nervous, alimentary, respiratory, circulatory, haematologic, urinary, endocrine, dermal, and central nervous systems. Chapters on vitamins and metabolic diseases of ruminants are also included. There are an additional 16 chapters in the chemotherapy section of the book, covering the usual antibacterials, antibiotics, antifungals, growth promoters, pesticides, anthelmintics, coccidiostats, antiseptics, disinfectants, antitrypanosome and antiprotozoal drugs. The 3 concluding chapters on pharmacy: prescription writing, dispensing and legal obligations will have relatively little value to students outside of the U.K. There are 3 appendices. The first, on Latin abbreviations will be of historical value in South Africa; the second adequately covers weights and measures; and the third consists of notes on the treatment of poisoning.

Although the authors have included much new material in this edition, 3 general criticisms can be levelled at their work. Firstly, undue emphasis has sometimes been placed on older and less frequently used drugs at the expense of modern and more commonly used drugs. For example, xylazine HCl is dealt with in about half a page and the side-effects are sketchily detailed. In contrast, chloral hydrate, which is seldom used nowadays, has 2 full pages devoted to it. Secondly, the authors have not made full use of current veterinary pharmacological literature. Their description of the toxicity of phenylbutazone in horses, for example, is incomplete. A number of papers have been published on the use of arteriodilators in congestive heart failure, but no mention is made of this use. The third criticism is that there are a number of major omissions. The benzodiazepine group of drugs to which diazepam belongs is not mentioned at all, nor is any reference made to the lethal neurotoxicity of the diamidines, phenamidine and diminazine. The well described effects of the oxytetracycline vehicles polyvinylpyrrolidone and propylene glycol are not mentioned in the chapter on tetracyclines. Long-acting oxytetracycline formulations, available for the past 3 years, are also not mentioned.

Despite the above criticisms, the book does have merit. The chapters on sex hormones, anabolic steroids and penicillins are good. The book is nearly 3 times the price of its predecessor which was published in 1979. It could serve as a supplementary text for veterinary students but unfortunately does not contain all the information they require.

C. Button

TO THE EDITOR **AAN DIE REDAKSIE**

MONENSIN POISONING IN SHEEP

Recently a number of cases of monensin poisoning in sheep were diagnosed and experimentally confirmed at the Section of Toxicology, Onderstepoort, Veterinary Research Institute. Monensin (Rumensin or Coban, Elanco) was included in the sheep feed as a growth stimulant, but due to mixing errors at feed mills and by farmers, gross overdosage sometimes resulted. Up to 40 times the recommended concentration of 22 ppm in feed have been colorimetrically determined[2].

Monensin is an ionophor-antibiotic, produced by the fungus, *Streptomyces cinnamonensis*. According to Pressman and Fahim[3] the monovalent carboxylic ionophors such as monensin, are rigid lipid soluble compounds, which form dynamically reversible complexes with monovalent cations such as K^+ and Na^+. The ionophors then act as vehicles for transporting the cations along their concentration gradients across cell membranes. The resultant changes in ion concentrations and electrical potentials have profound effects upon cellular function and metabolism and account for the interesting pharmacological and toxicological properties of the ionophors.

Lasalocid and salinomycin are other ionophors currently marketed in this country.

Monensin, under the trade name "Coban" is registered as a coccidiostat for broilers at approximately 100 ppm in the feed. "Rumensin" is the trade-name for monensin when it is used as a growth promotant in cattle feed (15-33 ppm) and sheep feed (15-22 ppm). Although monensin is not registered for use in rabbits, Fitzgerald[1] found it effective against *Eimeria stiedai* at 0,005 % to 0,01 % in their feed. Feed refusal occurred with 0,02 % monensin in their feed. Horses are very sensitive to monensin and may be endangered when sharing grazing and feeding troughs with cattle. The LD_{50} for horses is 3-4 mg/kg, making them 10 times more sensitive than cattle. Monensin is not registered for use in pigs either, but in this case, it was found that a growth promotant effect was achieved with a 100 ppm in the feed, while 200 ppm caused feed refusal and 500 ppm resulted in deaths (K Passmoor 1982, P.O. Box 98, Isando 1600, personal communication).

As monensin at twice the recommended level causes feed refusal in all animals including sheep, it was seen as a built-in safety mechanism against poisoning. However, the occurrence of field cases of monensin poisoning has refuted this supposition.

A typical case history was as follows: The sheep in question were fed monensin-containing cubes as a grazing supplement. At the time of the outbreak they were accustomed to the cubes, having been exposed to them for more than a month. A day after a new batch of the same cubes had been made available to them, the whole flock became apathetic and did not graze. Only a few of them reluctantly ate of the cubes. On the 2nd day, many of them passed loose black mucoid to watery, sometimes blood stained faeces, a number were mildly bloated and many walked with a stiff-legged gait as if they were suffering from laminitis. Feed refusal was a very prominent feature. They started dying from the third day and this went on for about 10 days. Ruminal pH was within normal limits (6-6,8).

When feed cubes free of monensin was put down beside the refused batch, the sheep ate them with gusto

The salient post-mortem features were hydropericardium, hydrothorax, ascites, lung oedema, enlarged liver and spleen, and haemorrhagic enteritis.

Histopathology revealed necrosis especially of myocardium and intestinal mucosa, as well as a nephrosis.

The initial sudden deaths can easily be mistaken for pulpy kidney, while the clinical signs of subacute cases appears similar to those of acidosis, with only the normal ruminal pH indicating the contrary. Feed refusal may be mistaken for anorexia.

The specimens to be taken for toxicological examination would be the incriminated feed (at least 500 g) with the appropriate label and batch number, as this is essential for pinpointing a badly mixed batch. For pathology, formalinized specimens of the heart apex, right and left ventricular wall and septum, as well as lung, liver, kidney, skeletal muscle (e.g. *M. semimembranosus*) and small intestine (especially the ileum) are of value.

The occurrence of monensin poisoning, emphasizes the importance of proper accurate mixing at the feed mill and on the farm. It also points to the fact that feed refusal is not an infallible safety factor.

To conclude, it must be stressed that this letter is not aimed at discrediting valuable products such as monensin and other ionophors. The intention is merely to elucidate monensin poisoning in sheep and to emphasize the hazards of using improperly mixed feeds. Used as recommended, monensin is not dangerous.

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TO THE EDITOR — AAN DIE REDAKSIE

ELECTRON MICROSCOPIC STUDY OF A SQUAMOUS CELL CARCINOMA ON THE EYELID OF A HORSE

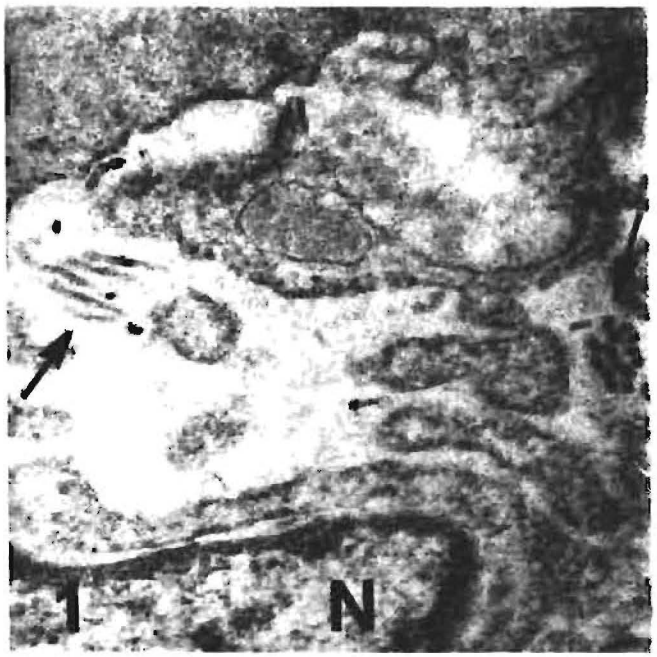

Fig. 1. Tubulofilamentous structures within cisternae of rough endoplasmic reticulum (↑) Nucleus (N). x 43,400.

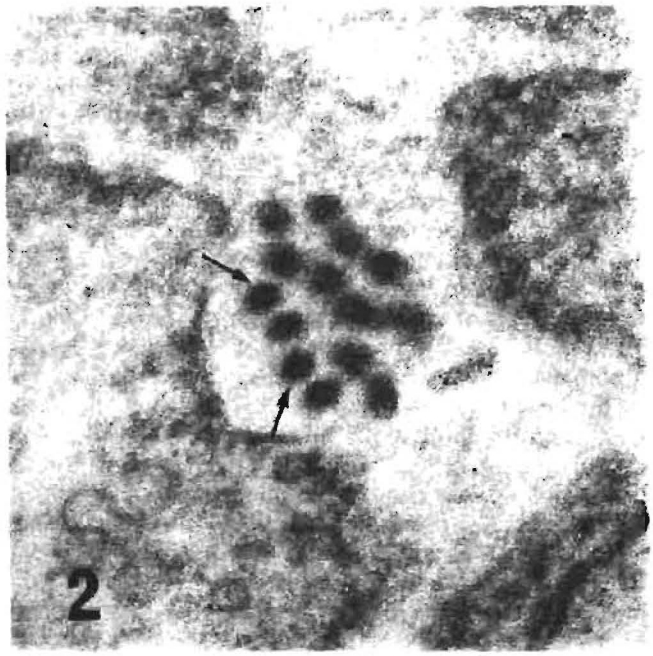

Fig. 2. Tubular elements showing limiting membrane (↑) x 122,600.

A small sample of a squamous cell carcinoma on the eyelid of a horse was fixed in 4% glutaraldehyde in cacodylate buffer, post-fixed in 1% osmium tetroxide, dehydrated with acetone and embedded in Araldite using a method modified from that of Rowden & Lewis[3]. Thin sections, post-stained with uranyl acetate and lead citrate were examined in an Hitachi HU 11B electron microscope at 75Kv. Polymorphonuclear leukocytes, monocytes, lymphocytes, plasma cells, an occasional mast cell, fibroblasts, melanocytes and epithelial cells were identified. Also present were large elongated cells with eccentric irregularly shaped nuclei within which the chromatin was clumped (mainly along the margins); there were sparse but normal-appearing mitochondria and a small Golgi region. The extensive cytoplasm was finely granular in appearance with very few free ribosomes but large juxta-nuclear distended cisternae of rough endoplasmic reticulum containing granular material and tubulofilamentous elements (Fig. 1) were a distinguishing feature of these unidentified cells. The circular particles have an electron-dense core and in appropriate sections (Fig. 2) appear to be limited by an outer membrane, these may be cross sections of the fibrillar structures or be derived from them by a budding-off process. The average diameter of both types is approximately 33-36 nm and in the long sections show no evidence of periodicity. What seem to be similar structures have been found in cancers of the prostate and breast by Tannenbaum & Lattimer[4] and within the glandular lumen of a case of human prostatic carcinoma by Dmochowski & Horoszewicz.[1] Smaller but identically appearing structures have been found by Grausz et al.[2] in the glomerularendothelium of 29/30 patients with systemic lupus erythematosus.

Although virus-like appearance (a possibility suggested by Tannenbaum & Lattimer, and Grausz et al.) the nature of the structures is unknown but hopefully similar observations may have been made by your readers.

I would like to thank Dr J.H. Mason of the Serum laboratories, S.A. Institute for Medical Research who supplied me with the material. The investigation was funded in part by the Poliomyelitis Research Foundation and the Department of Health and Welfare.

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TO THE EDITOR — AAN DIE REDAKSIE

ONKOTERAPIE IN HUISDIERE

)or die publikasie: *Onkoterapie in Huisdiere* deur .S.J. Odendaal en J.D.E. Cronje, Jaargang 53 nr. 3 982, kortliks die volgende:

In hulle berekening van die oorlewingsyfer gebruik ıulle die werklike lewensduur van die hond en kat, ıaamlik 12 jaar teenoor die mens se verwagte lewens- luur van 77 jaar.

'n Verhouding dus van 1:6,4 waar dit 1:4 moet wees, ıangesien die werklike lewensduur van die mens niks neer as ongeveer 47,5 jaar tel nie.

Met 'n oorlewingsyfer van 5 jaar vir mense behoort lit dan vir honde en katte 1,25 jaar te wees. Die addisionele 170 dae op hulle berekende 285 dae kan maklik verhaal word uit die 75 % van diere waar lewe met genadedood beëindig is.

Graag verneem ek of daar 'n verskil is in die oorlewingsyfer vir mense in die groep tot 'n ouderdom van sê 48 jaar en die groep ouer as 48 jaar?

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BOOK REVIEW — BOEKRESENSIE

THE UGANDA WATERBUCK

C.A. SPINAGE

1st edn. Academic Press, London 1982 pp XVI and 334, Figures 65, Plates 52 ISBN 0-12-657720-X. Price £24.

Dr Spinage has been involved in research and management and as a consultant on the ecology of African game for some 20 years. This is reflected in the text of his book which has its origin in a doctoral study he undertook on the social organization of the waterbuck in 1964. Waterbuck have been the subject of 3 further studies for degree purposes since then, those of H.J. Herbert (1972) at the Mammal Research Institute, Pretoria University, on the population dynamics in the Sabi-Sand Wildtuin and E.M.N. Elliott (1976) at Cambridge University, on their ecology in a newly enclosed area in Kenya being covered adequately in Spinage's review. Unfortunately Spinage was unaware of D.A. Melton's (1978) thesis at the Mammal Research Institute on "Ecology of waterbuck in the Umfolozi Game Reserve", parts of which have only been published this year. This was a pity because it is probably the most comprehensive study on waterbuck yet undertaken and covers several aspects reviewed by Spinage.

This shortcoming aside, Dr Spinage should be complemented for including most of the published research from southern Africa, so often a deficiency in books emanating from East Africa.

Although Spinage has tended to give the impression that behaviour is the most important aspect dealt with in the book, this is misleading. Only 3 of the 12 chapters relate entirely to ethology with the emphasis on territoriality. In the remaining chapters he covers the classification of waterbucks, his study area and methods used, growth and senescence, reproduction, parturition and maternal behaviour, population structure and factors affecting survival, population density, food supply and habitat preference and daily life.

In the very last chapter on Territorial Concepts and Function, Spinage also discusses territorial organisation in the Uganda kob, lechwe, puku, reedbuck, oribi, and the Alcelaphines, including topi, bontebok and hartebeest. The choice here seems to have been somewhat arbitary and it would have been preferable had he expanded the discussion on the kob, lechwe and puku, about which we know so little, particularly as they are also species associated with wetlands. Moreover, he omitted to include important research on puku in Zambia, by Wendy Rees and on lechwe in Botswana by D. Williamson. Again, this criticism is qualified because the first papers from these studies have only recently been published.

This is an excellent contribution to the literature on African mammals and, as such, will be widely welcomed. It will prove invaluable to any mammalogist studying our larger ungulates and Dr Spinage is to be congratulated on his continuing productivity despite his other commitments. Although the veterinarian may find the book disappointing with only 9 pages devoted to disease and parasites, one suspects this may be a reflection of the state of the science as far as veterinary medicine is concerned. But there is much more in this book to interest the veterinarian and all those interested in African big came will benefit by reading it.

J.D. Skinner

BOOK REVIEW BOEKRESENSIE

APPLIED ANIMAL REPRODUCTION

H.J. BEARDEN and J. FUQUAY

Reston Publishing Co. Inc., Virginia 1980 pp XIV and 337, Figures 142, Tables 57 (ISBN 0-8359-0249-8).

This is a well written easy-to-understand text aimed at the undergraduate student in Animal Science. It follows a clear concise path to bring the basics of applied animal reproduction home to the reader. It may serve as a reference to students in Animal Nursing with limited application for students in Veterinary Science.

The book is divided into 5 sections and only deals with cattle, horses, pigs and sheep. Section 1 deals with anatomy and physiology and covers the female and male reproductive systems, the hormones of reproduction and the factors associated with the natural regulation of reproduction in these species. Section 2 describes the various reproductive processes including the oestrous cycle, spermatogenesis, ovigenesis, fertilization, gestation, parturition and lactation. It also contains a chapter on male mating behaviour and one on semen and its components.

The third section covers artificial insemination (AI) including the history of AI, semen collection and evaluation, semen preservation and handling and AI-techniques. Section 4 deals with managemental procedures aimed at improved reproduction and contains chapters on the alteration or control of reproductive processes, reproductive management and pregnancy diagnosis. There are also chapters on environmental management and nutritional management in this section.

The last section deals very briefly with anatomical and inherited causes of reproductive failure, the physiological and psychological causes of such failure as well as the infectious diseases responsible for reproductive failure.

H.M. Terblanche

BOOK REVIEW BOEKRESENSIE

BORDER DISEASE OF SHEEP: A VIRUS-INDUCED TERATOGENIC DISORDER

R.M. BARLOW and D.S.P. PATTERSON (Ed.)

Advances in Veterinary Medicine. Supplement to Journal of Veterinary Medicine No. 36. Verlag Paul Parey, Berlin 1982 pp 90+43 illustrations and 9 tables ISBN 3-489-64416-6

This collaborative review on Border disease in sheep is an excellent example of results that can be achieved when a multidisciplinary approach to a disease problem is intiated. A short historical background to the disease is given and the various clinical syndromes are described as an introduction to the monograph. Detailed pathological descriptions of the lesions in the skin, fleece, nervous system and placenta are given. Various other chapters deal succinctly with the virology, neurochemistry, epidemiology, immunity and the economic impact of the disease. In a number of these chapters an indication is also given as to future research aims to elucidate certain aspects of the disease, e.g. a better understanding of the pathogenesis of Border disease. Another feature of the research into this condition is that it may be of great significance in elucidating the biology of similar viral infections in man. It is a pity that a monograph of this excellence is not available for each and every of the many economically important diseases in the veterinary field. This review is highly recommended to researchers and pathologists in veterinary science.

A.L. Lange