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TYDSKRIF VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING

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TYDSKRIF VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING

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BINNESPIERSE WEEFSELREAKSIE EN RESIDUE IN SLAGBEESTE NA TOEDIENING VAN LANGWERKENDE OKSITETRASIKLIEN FORMULASIES

INGE-MARIÉ PETZER*, J.J. VAN STADEN* EN W.H. GIESECKE*

ABSTRACT: Petzer Inge-Marié; van Staden J.J.; Giesecke W.H. **Tissue reaction and residues in slaughter cattle after administration of longacting oxytetracycline formulations.** *Journal of the South African Veterinary Association* (1984) **55** No. 3, 107-111 (Afr). Section of Food Hygiene, Veterinary Research Institute, P.O. Box 12502, 0110 Onderstepoort, Republic of South Africa.

Samples from 40 slaughter cattle were investigated over 10 weeks for determining macroscopic tissue reaction following intramuscular administration of Liquamycin/LA (Pfizers) into the thigh and Terramycin/LA (Pfizers) G 333 into the neck respectively. Antibiotic residues in the lesions were assessed under ultra violet light and by microbiological means. Antibiotic residues were still detected after 7 weeks in the thigh and 5 weeks in the neck. One week old lesions in the thigh consisted mainly of a local necrotic centre, up to 120 mm in diameter, with a haemorrhagic zone and surrounded by oedema and petechia in adjacent muscle. This was gradually replaced by mainly fibrotic tissue as the weeks went by and eventually by local abscess formation still apparent 10 weeks later. Much the same pattern was seen in the neck but of milder degree and shorter duration – 6 weeks. Alarming is the fact that only in one case out of 40, a lesion could be detected in the carcass on the slaughter line, the others could have passed fit for human consumption in an abattoir.

Because the manufacturer suggests a 28 day withholding period before slaughter and after administration of the product, results of this investigation raise several questions on format, completeness and contents of such directions for use and their implications for meat inspection, meat hygiene and public health.

INLEIDING

Suid-Afrika is 'n uitgestrekte land met relatief min veeartse en baie verskillende veesiektes. Sekere veemiddels is daarom kragtens Wet 36/1947 sonder voorskrif aan die boer beskikbaar. Oksitetrasiklien, beskikbaar in kort- en langwerkende formulasies word baie algemeen in vee gebruik aangesien dit 'n wyespektrummiddel is en baie bekende siektes kan genees.

Die langwerkende middel word deur boere verkies aangesien dit as eenmalige inspuiting gebruik kan word en sodoende tyd en arbeid bespaar. Die middels word gewoonlik formuleer vir intramuskulêre toediening en moet dus weefselverdraagsaamheid toon, d.w.s. binne veearts-aanvaarbare perke van weefselirritasie en ongemak in die dier.

Baie data is beskikbaar oor weefselbeskadiging veroorsaak deur kortwerkende oksitetrasiklienes soos gesien in werk gedoen deur Immelman et al[2], Nouws[3], Van Schothorst & Peelen-Knol[7], Svenden[6], Rasmussen & Hogh[5] en Gilbert et al[1]. Daarinteen is min inligting beskikbaar oor weefselbeskadiging a.g.v. die langwerkende oksitetrasiklien formulasies.

Ongeveer 80 % van skape wat tydens die produksie van hartwaterentstof met langwerkende oksitetrasiklien behandel is, het by slagting absessasie van die dy getoon (Oberem, 1981; ongepubliseerde data). Dit het die vermoede laat ontstaan dat erger en langerdurende weefselbeskadiging daarop volg as wat algemeen aanvaar word. Die langwerkende middel word as twee produkte, identies in formule, versprei – een as etiese produk in terme van Wet 101 van 1965 (Liquamycin/LA; Pfizer) en die ander as geregistreerde veemiddel onder Wet 36 van 1947 (Terramycin/LA; Pfizer; G 333).

MATERIAAL EN METODES

Eksperimentele diere

Veertig volwasse slagbeeste, verdeel in 4 groepe van 10 elk, is in die ondersoek gebruik. Hulle het verskil in ras, ouderdom en geslag terwyl hulle almal klinies gesond en goed gespier was met 'n gemiddelde massa van 450 kg.

*Seksie Voedselhigiëne, Navorsingsinstituut vir Veeartsenykunde, Posbus 12502, 0110 Onderstepoort, Republiek van Suid-Afrika.

Produkte ondersoek

1. Produk 1

Naam: Liquamycin/LA (Pfizer)
Inhoud: Die formulasie bevat 200 mg oksitetrasiklien per ml basis

Gebruiksaanwysings:

"Liquamycin/LA inspuitbare Oplossing is ontwerp vir binnespierse inspuiting as 'n enkele dosis van 1 ml/10 kg liggaamsmasa by beeste, skape, varke en bokke, wat 20 mg oksitetrasiklien per kg liggaamsmassa verskaf. Onderhuidse toedienings sal dieselfde uitwerkings as die binnespierse roete verskaf, maar swelling by die plek van inspuiting mag voorkom. By jong varkies van minder as 10 kg liggaamsmassa, word die onderhuidse roete van toediening verkies. Geen langdurige uitwerking word verkry as Liquamycin/LA Inspuitbare Oplossing binne-aars toegedien word nie.

'n Enkele inspuiting van 1 ml per 10 kg liggaamsmassa vir die behandeling van gevoelige besmettings sal gewoonlik voldoende wees. Herhaalde behandeling 48 – 72 uur na die oorspronklike inspuiting kan toegedien word indien nodig.

Tydelike gelokaliseerde swelsels by die punt van inspuiting mag waargeneem word.

Beeste – 1 ml met 10 kg liggaamsmassa. Binnespierse inspuitings moet diep in die vleisagtige deel van die spier toegedien word.

Voorsorgmaatreëls:

- Gebruik slegs by beeste, skape, varke en bokke.
- Vermy bevriesing
- Bewaar onder 30 °C
- Die vleis van behandelde diere mag nie vir menslike verbruik binne 28 dae aangewend word nie en in geval van melk nie binne 5 dae na die laaste behandeling nie.

2. Produk 2

Naam: Terramycin/LA (Pfizer) G 333
Inhoud: Die formulasie bevat 200 mg per ml oksitetrasiklien per ml basis.

0038-2809/84/03/0107-0111

Tabel 1: OPSOMMING VAN PRODUK, BEESTE EN TERAPEUTIESE ADMINISTRASIE ONDERSOEK

	1: Liquamycin/LA (Pfizer)	2: Terramycin/LA (Pfizer) G 333
Aantal beeste	3 groepe van 10 beeste	1 groep van 10 beeste
Posisie van toediening	Kaudale aspek van die dy, ± 250 mm distaal van die *Tuber ischiadus* in lyn van lg. na die *Tuber tarsalis*	Weerskante, in die middelste derde van die nek
Naalde gebruik	Regter dy: 17 × 25 mm Linker dy: 17 × 38 mm	17 × 50 mm
Middel per inspuitplek	Maksimum van 20 ml	Maksimum van 10 ml
Gemiddelde totale toediening per dier	40 ml (2 × 20 ml)	40 ml (2 × 20 ml)
Dag van behandeling	0	0
Dae van slagting (aantal diere per groep geslag)	7 (1) 14 (1) 21 (1) 28 (1) 35 (1) 42 (1) 49 (1) 56 (1) 63 (1) 70 (1)	7 (2) 14 (2) 21 (1) 28 (1) 35 (1) 42 (1) 49 (1) 56 (1) 63 (0) 70 (0)

Gebruiksaanwysings:

"Terramycin/LA Inspuitbare Oplossing is ontwerp vir binnespierse inspuiting as 'n enkele dosis van 1 ml/10 kg liggaamsmassa by beeste, skape, bokke en varke, wat 20 mg Terramycin per kg liggaamsmassa verskaf. Onderhuidse toediening sal dieselfde uitwerking as die binnespierse roete verskaf, maar swelling by die plek van inspuiting mag voorkom. By jong varkies van minder as 10 kg liggaamsmassa, word die onderhuidse roete van toediening verkies. Geen langdurende uitwerking word verkry as TERRAMYCIN/LA Inspuitbare Oplossing binneaars toegedien word nie.

Waarskuwing:

- Gebruik slegs in beeste, skape, bokke en varke
- Bewaar onder 30 °C
- Moet nie behandelde diere binne 28 dae na die laaste behandeling vir menslike gebruik slag nie
- Melk van behandelde koeie moet nie vir menslike verbruik binne 5 dae na die laaste behandeling gebruik word nie."

Ontwerp van die ondersoek

Produkte 1 en 2 is in spiere onderskeidelik van die dy en nek van die beeste ingespuit. Die 10 diere van elke groep is op dieselfde dag behandel en daarna is weekliks 1 bees per groep, volgens 'n vooraf bepaalde program, geslag. Besonderhede word in Tabel 1 verskaf.

Kliniese waarnemings

Na toediening van die produkte is die diere daagliks dopgehou vir enige tekens van swelling by die area van toediening of enige tekens van ongemak wat die diere toon a.g.v. die inspuiting.

Na-slagtingondersoek

Alle karkasse is onderwerp aan 'n roetine ondersoek soos voorgeskryf in staande regulasies van Wet 87 van 1967. Die oppervlakte van die karkas om en by die inspuitplekke is veral noukeurig ondersoek vir enige letsel of verandering wat verband kon hou met die inspuiting. 'n Groot spierporsie, insluitende die toedieningsarea, is daarna uit elke karkas gesny.

Laboratoriumondersoek

1. Patologies-anatomiese ondersoek van die vleis

Die subkutane oppervlakte van die spierporsies is

Fig. 1: Makroskopiese oppervlakte letsel sigbaar – een week na administrasie in die dy (in 2,5 % van gevalle).

Fig. 2: Letsel, onsigbaar vanaf oppervlakte, twee weke na administrasie in die dy. Met insnyding is die lokale area van nekrotiese weefsel, edeem en petechia intramuskulêr en subkutaan gevind.

Fig. 3: Drie weke na administrasie in die dy – intermuskulêre nekrose, geel verkleur met 'n hiperemiese rand asook echimosis in aanliggende fibrotiese weefsel sigbaar. Milde edeem is nog teenwoordig.

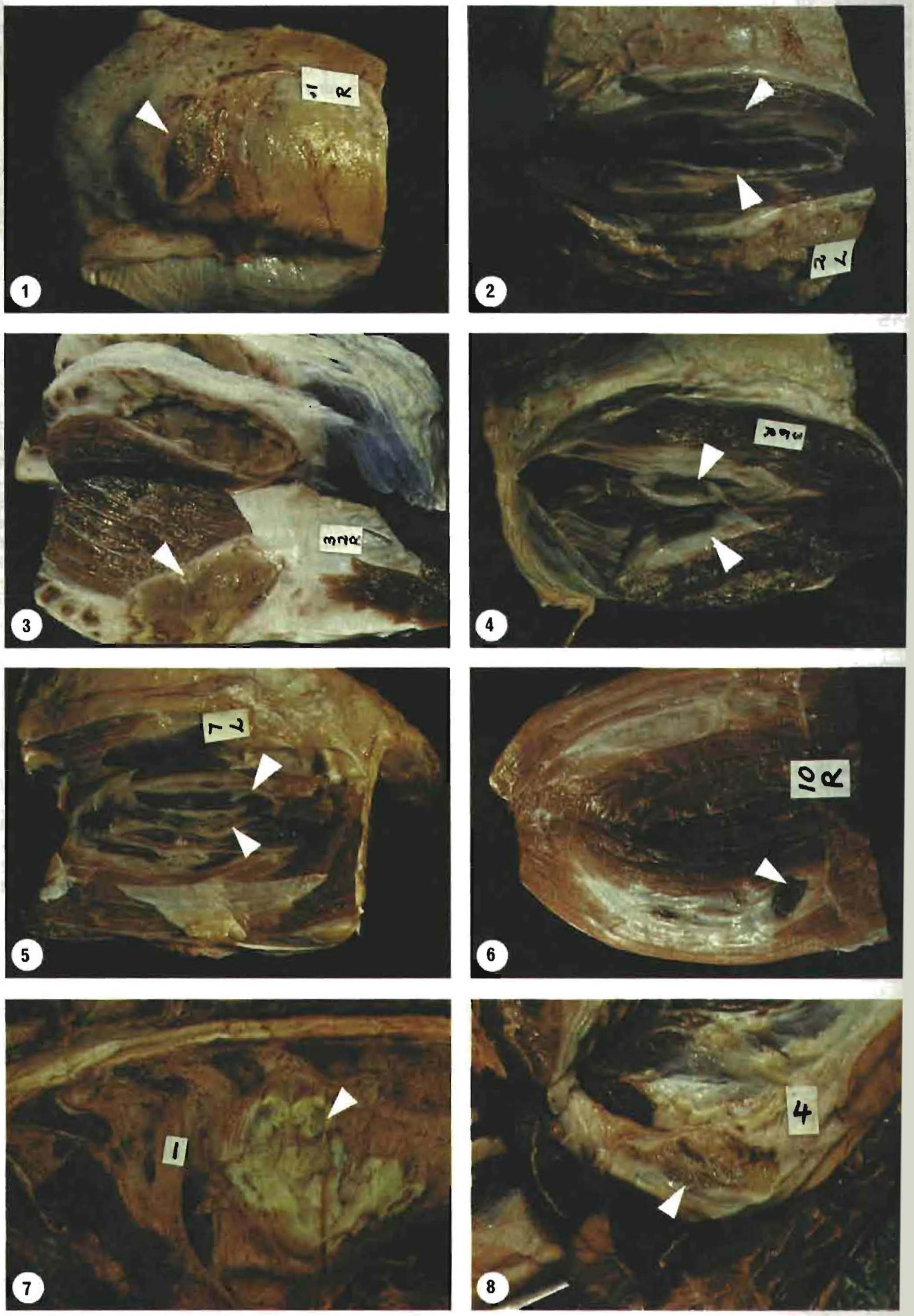
Fig. 4: Lokale intramuskulêre fibrose en absessasie vyf weke na administrasie in die dy.

Fig. 5: Sewe weke na administrasie in die dy – intramuskulêre absessasie omring deur fibrose nog baie prominent teenwoordig.

Fig. 6: Intramuskulêre fibrose met absesse tien weke na administrasie in die dy.

Fig. 7: Week een na administrasie in die nek – 'n lokale intramuskulêre nekrose, verkleuring, edeem en petechiale bloedings is teenwoordig.

Fig. 8: Vier weke na administrasie in die nek – milde fibrose en edeem teenwoordig.



fotografeer en daarna is insnydings in die veselrigting, deur die areas van die inspuiting gemaak. Snitvlakke is ondersoek vir enige veranderinge, bevindings is opgeteken en snitvlakke is fotografeer. Die area met die mees opsigtelike letsel is uitgesny vir die bepaling van tetrasikliene in die letsel.

2. Bepaling van tetrasiklienresidue

2.1 Fluoressensiemetode

Die teenwoordigheid van tetrasikliene is bepaal deur weefselondersoeke vir autofluoressensie onder langgolflengte ultraviolet (UV) lig.

2.2 Mikrobiologiese metode

Die vlak van tetrasikliene in die letsel is bepaal d.m.v. 'n skyfdiffusietegniek algemeen in gebruik vir die bepaling van antimikrobiese residue in melk. Veranderde weefsel is versnipper en 1g is daarna in 9 ml steriele distilleerde water suspendeer. Na ± 15 minute, by kamertemperatuur, is die suspensie vir 'n kort tyd geroer en 1 ml daarvan is gebruik om 'n serie van 1:2 verdunnings voor te berei in steriele distilleerde water. Elke verdunning is soos volg getoets:

'n Steriele papierskyf (Whatman AA, 13 mm deursneë) is in die verdunning geweek, oormatige vloeistof is verwyder deur die skyf oor die kant van die proefbuis te laat gly voordat dit op die oppervlakte van 'n vaste voedingsbodem, Bacillus No. 3 agar, geplaas is wat kort van te vore op die oppervlakte inokuleer is met 'n 24 uur oue kultuur van *Bacillus stearothermophilus var calidolactis*. Die organisme besit 'n minimum sensitiwiteit vir oksitetrasiklien HCl (0-HCl) ekwivalent aan ± 0,25 – 0,5 ug 0-HCL/ml van die substraat. Die plate is daarna oornag by 55 °C inkubeer, waarna die deursneë van enige inhibisiesones om die skywe gemeet is vir evaluering ooreenkomstig 'n standaardkurwe, geskep vir die ondersoek.

Standaardkurwe vir bepaling van vlak van tetrasiklien volgens deursnit van remsone

1 ml van Produk 1 is gebruik om in steriele distilleerde water 'n serie van 1:2 verdunnings te maak. Elk daarvan is ondersoek volgens bg. skyfdiffusiemetode ter bepaling van die groottes van die remsones wat veroorsaak word deur die verskillende tetrasiklienkonsentrasies. Ooreenstemmende vlakke van tetrasiklienkonsentrasies en die deursneë van remsones het gedien vir die berekening van 'n standaardkurwe geskep volgens:

$y = a + b \ln X$

waar y = deursnee (mm) van remsone

a = 32, 65 b = 2,79 en X = mg 0 – HCl/ml

RESULTATE

Kliniese bevindings

Produk 1

Op Dag 1 het al die diere merkbare swellings op beide dye getoon. Die veldbeeste, gewoonlik nie mak, het bly staan totdat mens baie naby hulle gekom het. Met aanraking van die inspuitplek het hulle pyn getoon. Die swelling en pynsimptome het geleidelik afgeneem en wel vinniger in die linkerdy. Na 7 dae het 6 van die 30 diere nog 'n geringe swelsel in die regterdy getoon.

Produk 2

Op Dag 1 was daar 'n matige maar waarneembare swelling by die inspuitplek langs die nek by 2 van die 10 diere; na 36 uur het al die beeste 'n effense styfheid van die voorbene en nek getoon wat veral opvallend was wanneer hulle draai. Op Dag 2 was geen swelling of ongemak meer sigbaar by enige van die beeste nie

Na-slagting

Met die uitsondering van een geval kon geen oppervlakkige weefselverandering by die inspuitplekke waargeneem word nie. Die uitsonderlike geval was een waar Produk 1 toegedien is: 'n duidelike area van lokale subkutane weefselnekrose, omring deur 'n akute inflammatoriese reaksie, het hier vertoon.

Laboratoriumondersoek

1. Patologies-anatomiese ondersoek van vleis

1.1 Produk 1

Die produk het, onafhanklik van die diepte van administrasie, duidelike subfasiale, intramuskulêre en/of intermuskulêre nekrose veroorsaak, assosieer met ooreenstemmende lokale inflammatoriese edeem en ietwat proliferatiewe fibrose; tydens Week 2 het die edemateuse veranderinge feitlik verdwyn terwyl fibrose toegeneem het; tydens die opeenvolgende weke het die nekrotiee letsel geleidelik verklein en het vanaf Week 5 'n neiging getoon om klein absesse, omring deur fibrotiese verandering van fluktuerende omvang, te vorm; tydens Week 10 het weefselletsels verder verklein maar net 2 van 6 spiermonsters was normaal terwyl die oorblywende nog absessasie en milde fibrotiese weefselveranderinge getoon het.

1.2 Produk 2

Die produk het duidelike intra- en intermuskulêre lokale nekrose by die inspuitplek veroorsaak met inflammatoriese veranderings in omliggende weefsel. Die letsel het op dieselfde wyse ontwikkel as met Produk 1, behalwe dat die neiging tot absessasie vanaf Week 3 begin het. Die laaste abses is 6 weke na toediening gevind; dit was omring deur 'n klein fibrotiese area. Vanaf Week 7 kon geen weefselverandering waargeneem word nie.

2. Tetrasiklienresidue

2.1 Fluoressensiebepaling van antibiotiese residue

Produk 1

Letsel wat onder ultraviolet lig ondersoek is, was positief tot die 7de week (Tabel 2).

Produk 2

Letsel wat tot Week 6 positief (Tabel 2).

2.2 Mikrobiologiese bepaling

Produk 1

Veranderde weefsel het bepaalbare tetrasiklienresidue tot Week 7 na toediening getoon. Die resultate was teenstrydig aangesien die tetrasiklien nie eweredig in die letsel versprei was nie (Tabel 3).

Tabel 2: GETAL MONSTERS MET FLUORESSENSIE A.G.V. OKSTITETRASIKLIENRESIDUE IN WEEFSELLETSELS VAN BEESTE GESLAG 1 – 10 WEKE NA INSPUITING

Produk	Diepte van toediening	Weke na behandeling/teenwoordigheid van tetrasikliene									
		1	2	3	4	5	6	7	8	9	10
1: Liquamycin/LA (Pfizer)	25 mm (in regterdy)	3/3*	3/3	3/3	3/3	2/3	1/3	0/3	0/3	0/3	0/3
	38 mm (in linkerdy)	3/3	3/3	2/3	2/3	2/3	1/3	1/3	0/3	0/3	0/3
2: Terramycin/LA (Pfizer) G 333	50 mm (in nek)	2/2	2/2	1/1	1/1	1/1	1/1	0/1	0/1		

* 3/3 = 3 monster uit 3 is positief vir oksitetrasiklien

Tabel 3: VLAKKE VAN TETRASIKLIENRESIDUE IN WEEFSELLETSELS VAN BEESTE GESLAG 1 – 10 WEKE NA INSPUITING

	Posisie van inspuiting	Weke na inspuiting/Deursnee (mm) van inhibisie sone (met 1:1 verdunning)									
		1	2	3	4	5	6	7	8	9	10
1: Liquamycin/LA (Pfizer)	Regterdy	26	27	24,5	18	15,5	–	22	–	–	–
	Linkerdy	26	23	29	–	12	12	–	–	–	–
2: Terramycin/LA (Pfizer) G 333	Nek	*	*	*	22,5	19	–	–	–	–	–

* Waardes nie bepaal

Produk 2

In veranderde weefsel is tetrasiklienresidue tot Week 5 in bepaalde konsentrasies opgewys (Tabel 3).

BESPREKING EN GEVOLGTREKKING

Navorsing met kortwerkende oksitetrasiklienprodukte toegedien aan kalwers, varke en konyne, het getoon dat makroskopies-sigbare letsels 6 dae en antibiotiese residue in die letsel tot 60 uur na toediening voorkom. Die verhouding van nekrose en fibrose het gewissel, afhangende van die draersubstans in die middel. (Immelman et al[2], Rasmussen & Hogh[5] en Nouws[3]).

Die studie dui aan dat die toestand egter veel erger is in die geval van die langwerkende produk. Kliniese simptome van swelling en ongemak het gewissel van 2 tot 9 dae, afhangende van die posisie, hoeveelheid en diepte van toediening. Die graad van patologies-anatomiese veranderinge van die inspuitplek het mettertyd geleidelik afgeneem en was na 6 tot 10 weke nie meer sigbaar nie. Die letsel was so geleë dat alhoewel spesiale aandag geskenk is aan die plekke van inspuiting, slegs 1 uit 40 gevalle (2,5 %) in die loop van die amptelike roetine karkasinspeksie waargeneem kon word. Beide die patologiese-anatomiese verandering asook antibiotiese redisue was nog teenwoordig na verstryking van 28 dae: die aanbevole tydperk volgens die "Gebruiksaanwysings". Die letsel is versteek en die kanse om diere raak te sien wat ingespuit is en selfs voor die weerhouperiode verstryk het, geslag word, is skraal. Hierdie feit kan lei tot groot probleme met gebruik van beide produk 1 en 2 t.o.v. antibiotiese residue, vleisinspeksies en volks-gesondheid.

Hierdie probleme word waarskynlik ook deur ander langwerkende antibiotika-formulasies veroorsaak en ernstige aandag sal dus aan die evaluering van derglike produkte in teikendiere gegee moet word.

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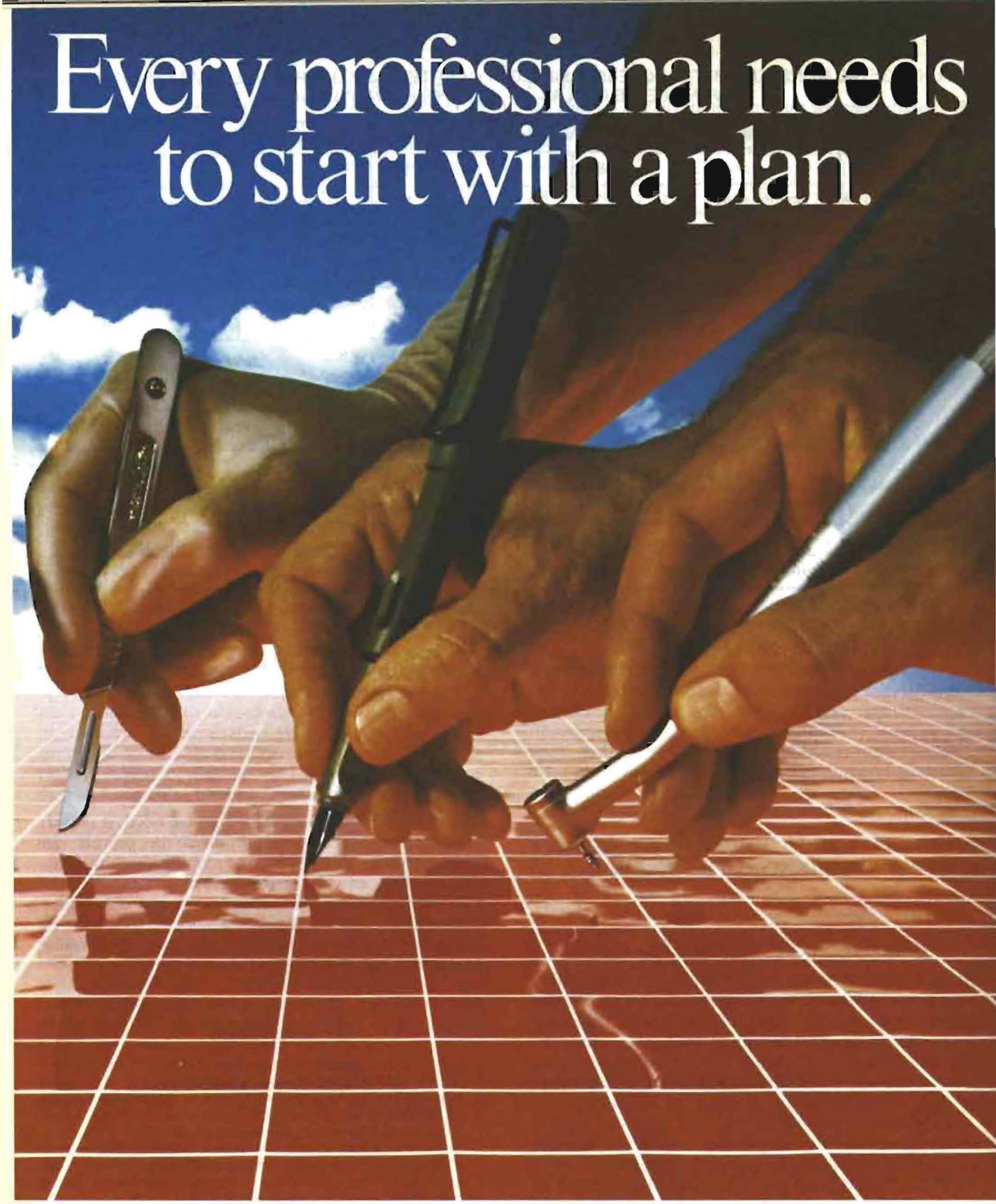

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BOOK REVIEW **BOEKRESENSIE**

NOMINA ANATOMICA

5th ed. Williams & Wilkens, Baltimore/London 1983 pp XXIX + 162 ISBN 0-683-06550-5 Price R30,00

Apart from containing the complete list of human anatomical terminology (approved by the Eleventh International Congress of Anatomists) the Nomina Histologia and the Nomina Embryologica are also included in this volume.

With the exception of directional terms like 'cranial' and 'caudal' which replace 'anterior' and 'posterior', our own Nomina Anatomica Veterinaria has its historical roots firmly planted in human terminology.

As can be expected the Anotomy Section comprises a meagre 86 pages compared to the 150 pages of the NAV which has to cater for the variations in anatomical patterns amongst our domestic animals.

M.M.S. Smuts

BOOK REVIEW **BOEKRESENSIE**

DISEASES OF SHEEP

W.B. MARTIN

Blackwell Scientific Publications, Oxford, London, Edinburg, Boston, Melbourne. 1983 pp X11 + 282 Figs 54 Price £27,50 (ISBN 0-632-01008-8)

The Moredun Institute, which has been engaged in research on sheep diseases since its establishment in 1925, has built up a vast store of knowledge on a wide variety of sheep diseases. The research papers emanating from this Institute have always been marked by thoroughness and profundity. In "Diseases of Sheep", Dr W B Martin, Director of Moredun, has collected the contributions of 22 of his research workers on pertinent topics and also of 20 other eminent workers in their respective fields. This is and authorative comprehensive work containing up-to-date information on a wide range of sheep diseases. Admittedly, the information in this book is directed particularly at the sheep industry in Britain and Europe, but students, practitioners and research workers in South Africa will find much of the information applicable to the local disease situation.

B.C. Jansen

BOOK REVIEW **BOEKRESENSIE**

APPLIED VETERINARY HISTOLOGY

WILLIAM J. BANKS

1st ed. Williams and Wilkens, London 1982 pp XV + 572 Figs 883 (108 colour photographs on 8 plates) Price $41,00, (ISBN 0-683-00410-7)

This book is a vastly improved and extended version of the original rather concise edition of Histology and Comparative Organology: A Text atlas. The improvements include the remarkable expanded knowledge in the field of ultrastructure, a much expanded histophysiological approach integrated with morphological considerations, all the necessary species differences in the histological structure of domestic animals, the principles of tissue regeneration and repair and lastly an important chapter on normal and pathological exfoliative cytology which is of great practical importance in clinical diagnoses.

The book is divided into five sections viz general principles of histology, cytology, histology of tissues, organology and exfoliative cytology. It gives sufficient detail on the various aspects, is well written, concise and easy to follow. It is one of the very few histological texts which is most suitably adapted to the requirements of the veterinary student. With 883 illustrations and 108 colour photographs of cells and tissues the text is most effectively illustrated. Many of the black and white figures are three dimensional sketches which are so important in conveying a correct understanding of tissue and organ sections seen under the microscope.

It is a book which I can wholeheartedly recommend to all students of the veterinary, medical and zoological sciences.

W.H. Gerneke

ABSTRACTS

ABSTRACT: Lange, A. Lucia, Brown, J.M.M. & Maree, Charlotte C., 1983. **Biochemical studies on a suspected lysosomal storage disease in Abyssinian cats.** *Onderstepoort Journal of Veterinary Research.* **50,** 149-155 (1983).

Blood lipid analysis was performed on the serum of 2 normal kittens and 1 adult cat on serum from 3 affected kittens.

Thin layer chromatography was done on tissue extracts of various organs from clinically affected kittens and unaffected unrelated kittens of a similar age, and on serum from carrier cats, affected kittens, related unaffected and unrelated kittens. Spleen end lymph node cell cultures were prepared from 1 affected kitten and the growth medium and cell cultures were analysed for lipids. A lecithin-like phospholipid was identified in the serum of an affected kitten, a carrier cat and a related unaffected kitten. This substance was produced by the liver of affected kittens and also by macrophage-like cells in spleen cell centures prepared from the spleen of a kitten with signs of the disease.

ABSTRACT: Newsholme, S.J., Kellerman, T.S., Van der Westhuizen, G.C.A. & Soley, J.T., 1983. **Intoxication of cattle on kikuyu grass following army worm *(Spodoptera exempta) invasion.* Onderstepoort Journal of Veterinary Research, 50,** 157-167 (1983).

Clinical features and pathological and mycological findings in a field outbreak of intoxication in dairy cattle grazing kikuyu grass are reported. The outbreak followed invasion of the grass by the army worm (*Spodoptera exempta*).

Clinical signs included drooling of saliva, depression, apparent inco-ordination, sunken eyes, ruminal distension and atony, recumbency, moderate diarrhoea and "sham drinking". Seventy-seven cows (64 %) were clinically affected over a period of 12 days. Of these, 37 died.

Necropsies performed on 4 affected cattle revealed necrosis of the epithelium of the forestomach, which was consistently more severe in the omasum. Light microscopy showed extensive necrosis of the epithelium of the forestomach with associated fibrinopurulent inflammation. The *stratum spinosum* and *s. granulosum* were selectively involved, but the *s. basale* was generally preserved. Electron microscopical examination of ruminal and omasal epithelium from 2 of these cattle revealed cytopathological features in the *s. spinosum* and *s. granulosum* which were consistent with stages in an acute, anoxic type of injury.

Mycological examination of the pastures revealed sparse growth of a mixed fungal population, which included *Myrothecium verrucaria.* There was no evidence of heavy fungal infestation.

Previous evidence that *M. verrucaria,* or other fungi, may be involved in the aetiology of kikuyu grass poisoning of cattle in New Zealand is addressed. It appears improbable that any of the fungi isolated in this investigation could played an important role in the aetiology of this outbreak.

ABSTRACT: Car, M., 1983. **The influence of water-level fluctuation on the drift of *Simulium chutteri* Lewis, 1965 (Diptera, Nematocera) in the Orange River.** *Onderstepoort Journal of Veterinary Research,* **50,** 173-177 (1983).

In July 1982, the invertebrate drift at Marksdrift comprised 98,7 % *Simulium chutteri;* 0,75 % Chironomidae; 0,3 % Ephemeroptera; 0,15 % Copepoda, and 0,1 % Trichoptera. Simuliid eggs were found in only 6 out of 75 samples.

A single water-level reduction of 57 cm (54 %) resulted in a more than sixfold increase of *S. chutteri* larvae in the drift and a more than 50 % decrease of 1st and 2nd instar larvae in the drift after the water had returned to its original level. Larvae found lying in pools after the water-level had dropped belonged mainly to instars 5-7, 70 % of them showing symptoms of starvation after 3 days when the river had risen again.

The drift of simuliid head capsules decreased when the larval drift increased, as fewer simuliid larvae moulted when they had been disturbed.

The low drift of eggs and the presence of very few pupae and adults indicated that most of the *S. chutteri* population was in the larval stage and that July was therefore an ideal month for water-level manipulation. Its main effect was achieved by irritating larger larvae and thus preventing them from resettling.

ABSTRACT: Scialdo-Krecek, Rosina C., 1983. **Studies on the parasites of zebra. II. *Cylicostephanus longiconus n.* sp. (Nematoda: Strongylidae) from the mountain zebra, *Equus zebra hartmannae* (Matschie, 1898).** *Onderstepoort Journal of Veterinary Research,* **50,** 169-172 (1983).

A new species of nematode, *Cylicostephanus longiconus,* was collected from mountain zebra. *Equus zebra hartmannae* (Matschie, 1898), on the Kelpie farm in the Khomas Hochland, South West Africa/Namibia.

These nematodes have 1 large dorsal and 2 small subventral teeth in the oesophageal funnel and submedian papillae with very long tips. The males have a very well-developed dermal collar and genital cone.

ABSTRACT: Boomker,J., Horak,I.G., Gibbons,Lynda M. & de Vos V., 1983. ***Haemonchus contortus* from the vaal ribbok, *Pelea capreolus,* and the bontebok, *Damaliscus dorcas dorcas,* in the Bontebok National Park.** *Onderstepoort Journal of Veterinary Research,* **50,** 179-181 (1983).

During a survey of the parasites of antelope in the Bontebok National Park, Swellendam, Cape Province, specimens of *Haemonchus contortus* with exceptionally long spicules were recovered from 5 out of 8 bontekok, *Damaliscus dorcas dorcas,* and 3 out of 5 vaal ribbok, *Pelea capreolus,* but not from 4 springbok, *Antidorcas marsupialis.* Typically, *H. contortus* has spicules 0,466 ± 0,085 mm long, but those recovered from vaal ribbok had spicules 0,581 ± 0,02 mm long and were recovered in large numbers from this antelope only. This indicates that the nematode is probably a definite parasite of vaal ribbok, and its occurrence in bontebok must be regarded as accidental.

BLOOD SELENIUM LEVELS OF SHEEP IN SOME DISTRICTS OF THE NORTHERN ORANGE FREE STATE: THE BULTFONTEIN AREA

J.A. ERASMUS*

ABSTRACT: Erasmus J.A. **Blood selenium of sheep in some districts of the Northern Orange Free State: The Bultfontein area.** *Journal of the South African Veterinary Association* (1984) **55** No. 3, 115-116 (En). Veterinary Laboratory, P.O. Box 625, 9500 Kroonstad, Republic of South Africa.

Blood selenium levels of apparantly healthy, adult sheep from the Bultfontein area, as determined by neutron activition, varied between 0,165 and 0,500 μg/ml. Due to the absence of anaemia, a symptom which could be taken as one of the earliest signs of chronic selenosis, these values suggest the intake of high but non-toxic amounts of selenium.

Keywords: Selenium, sheep, grazing.

INTRODUCTION

Originally the biological significance of selenium was confined to its toxic effects, but later surveys indicated that larger geographical areas were affected by deficiency rather that by excess[6]. Using blood selenium values as a measure of selenium intake[10], the situation in the Northern Orange Free State (NOFS) appears to vary from deficient in some of the eastern districts, sufficient in the central districts and high in the Bultfontein area[5]. With reference to the latter area the median blood selenium concentration of 13 clinically healthy sheep was 0,360 μg/ml in contrast to the 0,165 μg/ml for sheep in the Kroonstad district[5].

A more complete survey as to the blood selenium status of sheep in the Bultfontein area was conducted. This data as well as possible effects of the suggested increased selenium intake by sheep in that area[5], are discussed.

MATERIALS AND METHODS

Eleven farms in the Bultfontein area were randomly selected. On these properties blood samples of 115 clinically healthy sheep were taken from the jugular vein into heparinized vacutainer tubes (Radem Laboratory Equipment, Wynberg). A 1,0 ml volume from each sample was freeze dried. Blood selenium as determined by neutron activation on the freeze dried samples[5], were finally expressed as μg Se/ml.

The whole blood samples were also used for the determination of packed cell volume (PCV) and blood haemoglobin (Hb). PCV was determined by centrifuging samples for 5 min. in a Runne microhaematocrit centrifuge (Labotec Pty. Ltd., Johannesburg). Hb was determined spectrophotometrically after the addition of Zap-oglobin (Coulter Electronics, Halfway House) to 20 ml of a 1/501 dilution of whole blood in Isoton (Coulter Electronics, Halfway House).

Normal ranges for blood selenium, PCV and Hb were read directly from cumulative, relative frequency curves[5] which were constructed from the raw data.

RESULTS

All results are given in Figs. 1, 2 and 3 and summarized in Table 1.

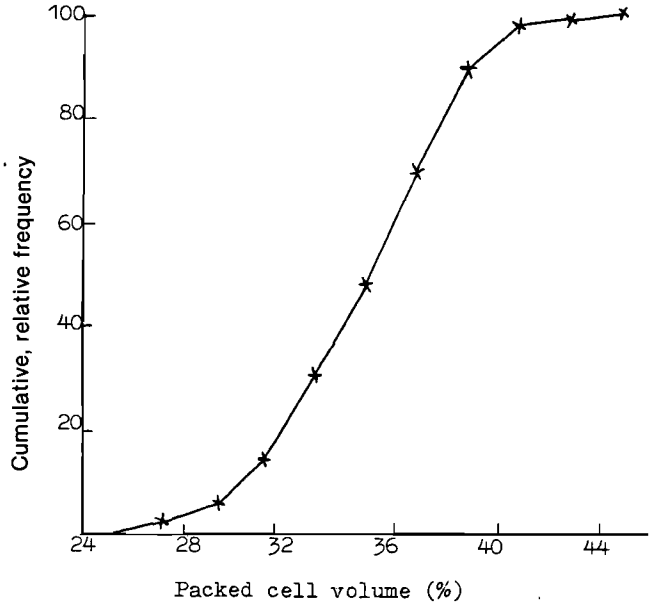

Fig. 1. Cumulative relative frequency curve for the packed cell volume of sheep in the Bultfontein district.

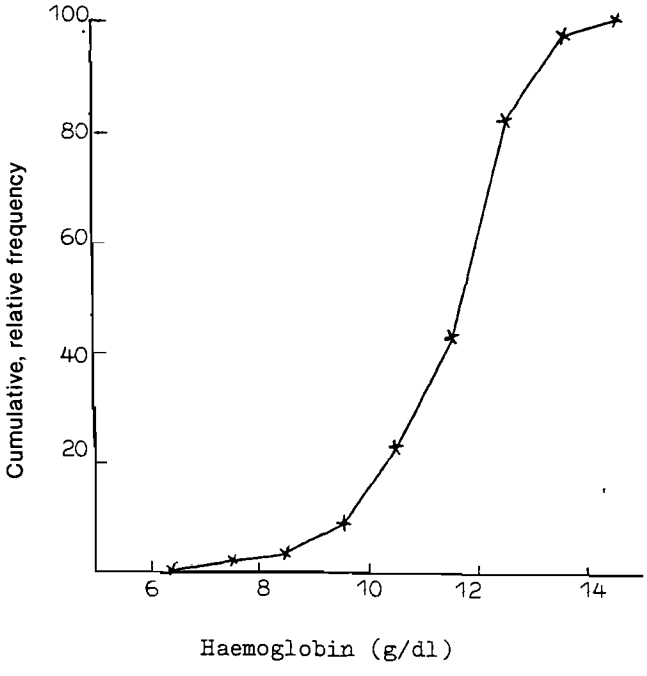

Fig. 2. Cumulative relative frequency curve for blood haemoglobin of sheep in the Bultfontein district.

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Table 1: RANGES FOR BLOOD SELENIUM, PACKED CELL VOLUME AND HAEMOGLOBIN FOR CLINICALLY HEALTHY SHEEP FROM THE BULTFONTEIN AREA COMPARED WITH NORMAL RANGES GIVEN IN THE LITERATURE

Determination	Figure shown by median (50%)	Ranges			Range cited in the literature
		80%	10% lower	10% upper	
Selenium (μg/ml)	0,320 (n* = 115)	0,165-0,500	0,050-0,165	0,500-0,750	0,090-0,305 Median = 0,165 [5]
Packed cell volume (%)	35,2 (n = 103)	31 – 39	26 – 30	39 – 42	24 – 46 [1] Avarage = 38 [8]
Haemoglobin (g/dl)	11,6 (n = 115)	9,6 – 12,9	7,2 – 9,6	12,9 – 13,9	8 – 15,0 [1] Average = 12 [8]

*n = number of observations

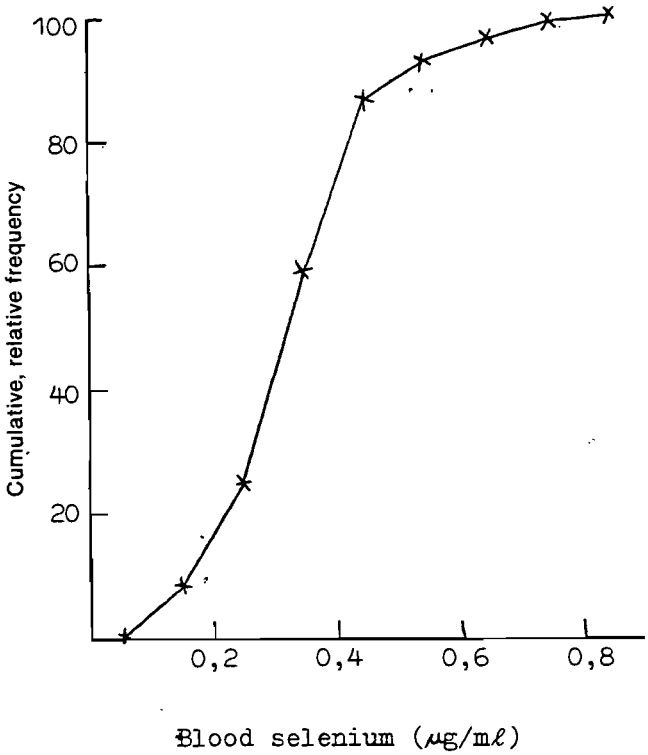

Fig. 3. Cumulative relative frequency curve for blood selenium of sheep in the Bultfontein district.

DISCUSSION

Depending on the magnitude and continuety of intake, a steady rise in tissue selenium develops over a period of weeks of months until saturation levels are reached[9]. At this point excretion of selenium in the urine and the faecal matter begins to keep pace with absorption[3]. Selenium is thus not continuously cummulative in the tissues. Retained selenium is partly incorporated into sulpher containing proteins such as selenocystine and selenomethionine, replacing sulpher normally present, while the balance of the selenium occurs as more labile compounds[7 9]. When an animal gets transferred from a seleniferous to a non-seleniferous diet, it's tissue selenium levels decline[9].

Sheep selected for this particular survey were all dependant on natural grazing consisting mainly of smaller perenniel Karoo shrubs. As the protein content of this type of nutrient is expected to remain fairly constant throughout the year[4] and as plant selenium is mainly bound to its protein fraction[2], the selenium intake by these sheep should have been fairly constant. If maximum tissue saturation levels were already reached, clinical illness due to chronic selenosis with blood levels up to 3 μgSe/ml[1] could occur in at least some of these sheep. The median selenium level found during this survey however was about 10 times less than that of sheep suffering from chronic selenosis.

One of the earliest indications of chronic selenium poisening is an anaemia which is characterized by the depression of blood Hb levels[1]. In the Bultfontein sheep the ranges for both PCV and Hb compare favourably with the norms suggested by Blood, Henderson & Radostits[1] as well as Schalm, Carrol & Jain[8]. Notwithstanding high blood selenium concentrations, these sheep could thus also be described as haematologically normal.

The median blood selenium concentrations for sheep in both the Bultfontein surveys were of the same magnitude, but about 2-3 times more than the median value for sheep in the Kroonstad district[5]. These increased values could however not be taken as indicative of chronic selenosis. Therefore the normal range for blood selenium of sheep in the NOFS, consedering data from the Kroonstad[5] and the Bultfontein areas should be taken as 0,090 – 0,500 μg/ml.

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THE EFFECT OF THAWING TEMPERATURE ON POST-THAW LONGEVITY OF FROZEN BOVINE SEMEN

R.O. GILBERT*

ABSTRACT: Gilbert R.O. **The effect of thawing temperature on post-thaw longevity of frozen bovine semen.** *Journal of the South African Veterinary Association* (1984) **55** No. 3, 117-118 (En). Department of Genesiology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

Bovine semen, diluted with a skimmed milk, 10 % egg yolk, 7 % glycerol extender and frozen in 0,25 ml French straws, was thawed in water at 5 °C, 20 °C or 35 °C for 20 seconds and then incubated at 37 °C. Specimens were examined periodically for motility. Semen thawed at 35 °C maintained motility for longer than that thawed at lower temperatures. Attepts to duplicate these findings *in vivo* were inconclusive but tend to correlate *in vitro* findings.

Key words: artificial insemination, bovine, frozen semen, thawing temperature.

INTRODUCTION

The rapid progress of artificial insemination technology and new methods of semen packaging have caused some confusion in semen handling practices in the field. This work was undertaken to determine whether any differences in longevity (as evidenced by spermatozoan motility) could be brought about by different thawing temperatures.

MATERIALS AND METHODS

In experiment I, semen from one ejaculate of each of 5 bulls was diluted in a skimmed milk-egg yolk (10 %) – glycerol (7 %) extender, frozen in 0,25 ml French straws ("Cassou" fine straws, IMV, France), and stored in liquid nitrogen. Several straws of semen from each bull were then thawed in water 35 °C, 20 °C or 5 °C for 20 s. Unopened straws were then transferred to a water bath at 37 °C. At 1, 3, 6, 9 and 25 h ater thawing one or more straws were examined for spermatozoan motility using a phase contrast microscope with a warm stage maintained at 37 °C. The percentage of spermatozoa displaying progressive motility, local motility or no motility were estimated by the author and an experienced technician and recorded. Specimens were randomized so that neither examiner could identify them. In experiment 2, one ejaculate of each of two bulls was frozen in the same way as above. Three straws of semen of each bull were thawed at 35 °C, 20 °C and at 5 °C. Nine di-oestrous heifers were inseminated with semen from each bull. Four, six or nine hours post-insemination, cervical mucus was aspirated from each heifer and examined microscopically as above. The percentage of spermatozoa displaying any motility was recorded. (Viscosity of cervical mucus prevented clear progressive motility).

Table 1: MOTILITY OF SPERMATOZOA OF VARIOUS BULLS AT VARIOUS INTERVALS AFTER THAWING. (PERCENTAGE PROGRESSIVELY MOTILE : PERCENTAGE LOCALLY MOTILE : PERCENTAGE IMMOTILE)

Bull	Interval after thawing (h)	Thawing temperature (°C)		
		35	20	5
I	1	55:10:35	45:10:45	35:10:55
	3	50:10:40	40:10:50	0:25:75
	6	30:15:55	15:15:70	0:10:90
	9	25:10:65	10:10:80	0:10:90
	24	0:10:90	0: 0:100	0: 0:100
II	1	30:10:60	30:10:60	25:10:65
	3	20:10:70	5:10:85	5:10:85
	6	5: 5:90	5: 5:90	5: 5:90
	9	0: 0:100	0: 0:100	0: 0:100
	24	0: 0:100	0: 0:100	0: 0:100
III	1	50:10:40	45:10:45	45: 5:50
	3	45:10:45	40:10:50	30:10:60
	6	35:10:55	10:10:80	20:10:70
	9	20:10:70	5: 5:90	0: 5:95
	24	0: 0:100	0: 0:100	0: 0:100
IV	1	45:45:40	35:15:50	20:20:60
	3	50: 5:45	40:10:50	20:10:70
	6	5:25:70	5:10:85	0: 5:95
	9	0:10:90	0: 5:95	0: 0:100
	24	0: 0:100	0: 0:100	0: 0:100
V	1	30:10:60	10:10:80	0: 0:100
	3	10:10:80	0: 0:100	0: 0:100
	6	0:10:90	0: 0:100	0: 0:100
	9	0: 5:95	0: 0:100	0: 0:100
	24	—		

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Table 2: PERCENTAGE OF MOTILE SPERM RECOVERED FROM CERVIX OF INSEMINATED HEIFERS AT VARIOUS INTERVALS AFTER INSEMENATION

Bull No	Interval after insemination (h)	Thawing temperature (°C)		
		35	20	5
V	4	30%	0%	0%
	6	No sperm recovered	0%	0%
	9	0%	0%	No sperm recovered
VI	4	60%	60%	20%
	6	No sperm recovered	10%	0%
	9	No sperm recovered	0%	No sperm recovered

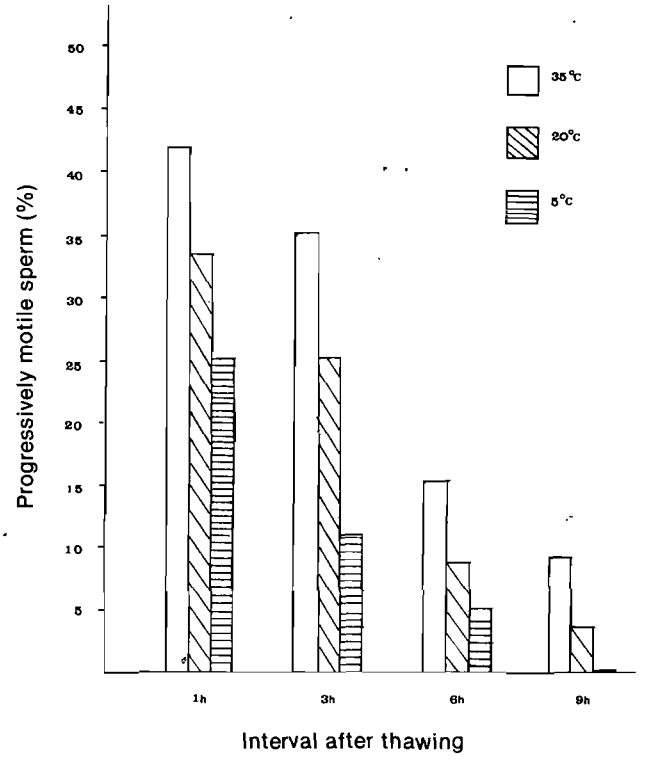

Fig. 1: Mean motility after in vitro incubation of spermatozoa thawed at 35, 20 or 5 °C.

RESULTS

The results of Experiment 1 are shown in Table 1 and Fig. 1. It is noteworthy that the initial difference in motility following thawing at different temperatures was small.

After a period of incubation, however, differences become more pronounced and in each case, semen thawed at 35 °C showed better longivity than semen thawed at 20 °C which, in turn was better than that thawed at 5 °C.

The results of Experiment 2 are shown in Table 2. Sperm recovery from the cervices of these heifers was poor, but in both cases, 35 °C thawing appeared to be superior in terms of sperm survival.

DISCUSSION

Modern changes in semen processing, especially the change in South Africa in the past five years from 0,5 ml to 0,25 ml French straw has focussed attention on semen handling techniques. The high surface to volume ratio of the 0,25 ml straw permits a more even thaw and a higher sperm recovery rate after thawing (B. Cassou, 1979, personal communication, IMV, L'Aigle, France). This same characteristic makes the 0,25 ml straw more susceptible to poor handling.

Previous studies, have found the optimal thawing rate to differ with diluent, packaging system and rate of freezing[1,2]. At present, the South African market is serviced almost entirely by semen diluted in milk-egg yolk-glycerol extender and frozen in 0,25 ml French straws. The present study indicates that optimal spermatozoal survival for such semen is obtained by thawing wat 35 °C in preference to lower temperatures. Higher temperatures and shorter exposure may well be more beneficial, but are not practical in the field especially under South African conditions of owner-insemination.

ACKNOWLEDGEMENTS

This study was undertaken while the author was in the employ of Taurus Artificial Insemination Co-operative, Private Bag 5, 1675 Irene, Republic of South Africa.

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THE STARCH DIGESTION TEST IN THE HORSE

S.R. VAN AMSTEL*, F. REYERS* and P.A. COLLY**

ABSTRACT: Van Amstel S.R.; Reyers F.; Colly P.A. **The starch digestion test in the horse.** *Journal of the South African Veterinary Association* (1984) **55** No. 3, 119-120 (En). Department of Medicine, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

Variable results were obtained when the starch digestion test was carried out on a suspected case of malassimilation in a horse. In order to re-evaluate this test, 15 starch digestion tests were carried out using 4 horses. Results showed a marked variation in the increase in plasma glucose levels between individual horses and especially between poor and good condition horses when the dose of starch was based on body mass. Results also suggest that a fixed dose of starch may give more consistent results.

Key words: Starch digestion test, horse.

INTRODUCTION

Intestinal malabsorption is well documented in the horse[1]. Maldigestion, however, resulting from exocrine pancreatic insufficiency, has not been recognised in this species[2]. Although the oral glucose tolerance test (OGTT) has been shown to be a reliable indicator of the small intestinal absorptive capacity[4], the question still remains open regarding the possible presence of maldigestion when using this test. A starch digestion-absorption test which can eliminate this problem has been described[3], but variable results were obtained by us when this test was used on a suspected case of maldigestion in a horse. An attempt to evaluate some of the influences on this test as an indicator of intestinal digestive and absorptive capacity was carried out.

MATERIALS AND METHODS

Four Thoroughbred horses (A, B, C and D), aged between 2-8 years were used in the test. A clinical examination, urine analysis, faecal flotation and haematology were carried out on each of the horses. They all appeared to be in good health but two (A and B) were in fairly poor body condition but not emaciated. Their weights were A, 395 kg; B, 420 kg; C, 460 kg and D, 465 kg.

The starch digestion tests were conducted with a minimum of 2 d between tests. The horses were starved for 16-18 h prior to starch administration. Eleven tests were carried out using a commercial soluble starch (Maizena CPC South Africa (Pty) Ltd. Durban) at a dosage rate of 2 g/kg body weight mixed in 4 ℓ of water. In another 4 tests a standard dosage of 900 g per animal was used mixed in the same amount of water.

Fasting blood samples were taken before starch administration. After dosing the starch, blood samples were taken at $^1/_2$ h intervals for 3 h. Blood was collected in Varley's fluoride preservative[7]. At the end of each test the plasma was separated and glucose determinations carried out using the GOD-PAP method of Trinder[6] adapted for use on the auto-pacer (Chemetrics Co, USA).

RESULTS

The results of the tests (n = 7) carried out using a dosagerate of 2 g/kg are shown in Fig. 1. The analysis of variance[5] showed firstly that there was a significant difference ($p < 0,001$) between the mean glucose levels at different half-hourly intervals. Secondly there was also a significant difference ($p < 0,001$) between animals within the half-hourly intervals. Lastly it showed that the difference between animals at half-hourly intervals is significant at 1½, 2½ and 3 h ($p < 0,05$) and at ½, and 2 h ($p < 0,01$), but not at Time 0 (fasting glucose).

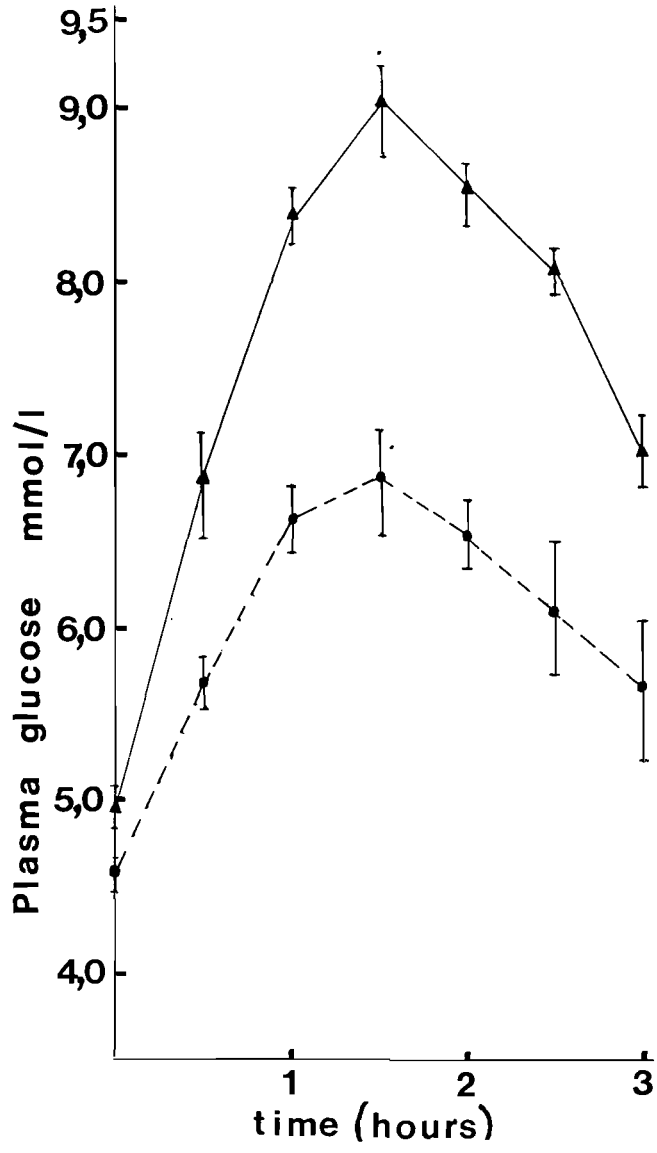

Fig. 1: The mean plasma glucose concentrations ±standard error in Horses A and B (n = 4) (– – – –) and C and D (n = 3) (————) at ½ hourly intervals. Starch dose: 2 g/kg bodymass. Vertical lines indicate the range.

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The results of the tests (n = 4) using a standard dose of 900 g of starch is shown in Fig. 2. It shows that there was no significant difference in the glucose levels between the horses.

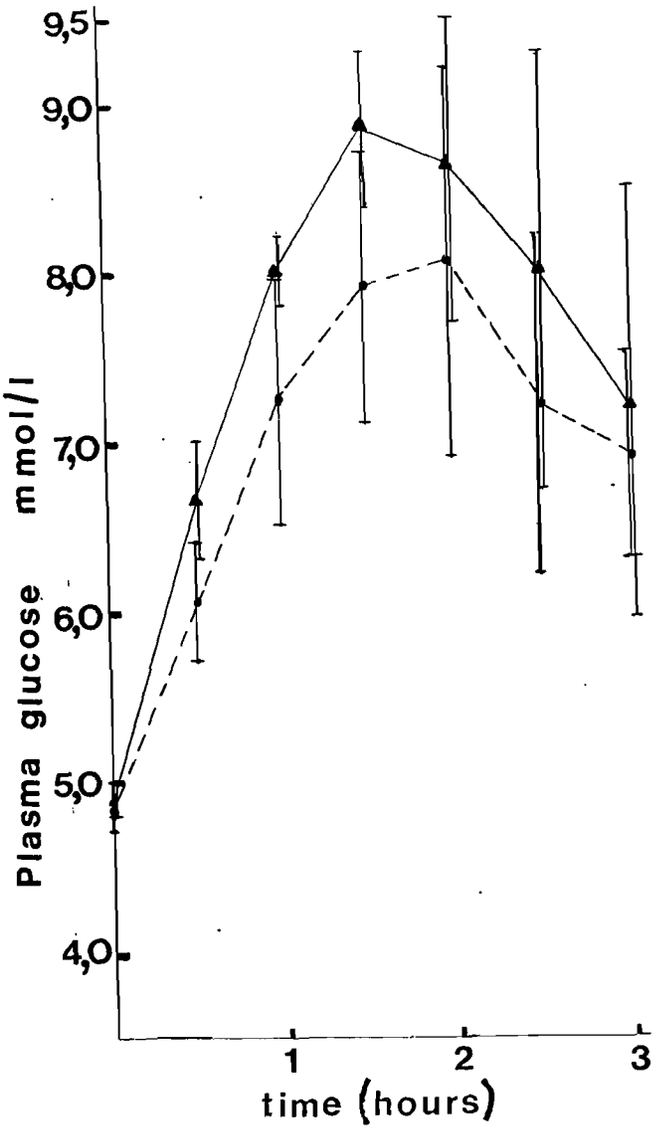

Fig. 2: The mean plasma glucose concentrations ± 1 standard error in Horses A and B (n = 2) (– – – –) and C and D (n = 2) (————) at ½ hourly intervals. Starch dose: 900 g. Vertical lines indicate the range.

CONCLUSIONS

Two of the horses in the trial (A and B) were in fairly poor condition, and the other two (C and D) were in good condition. Analysis of variance showed that there was a significant difference ($p < 0,01$) between mean glucose levels at half-hourly intervals between the "good" condition and "poor" condition horses, with the glucose means for the "good" condition horses exceeding that of the "poor" condition horses. This difference was not significant ($p < 0,05$) at Time 0 and 3 h. This, latter finding may suggest that administration of the dosage of starch on a body-mass basis (eg 2 g/kg) will disadvantage a poor condition horse in terms of the extent of the elevation of blood glucose values. In other words they are underdosed with respect to their gut absorptive area and/or their plasma volume. This problem seems to be overcome when a standard dose of starch is used. However, one starch assimilation trial on each horse gives insufficient data for a statistically valid conclusion and the final proof must wait until more work has been done.

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A NEW CAUSE OF CATTLE MANGE IN SOUTH AFRICA: *PSORERGATES BOS* JOHNSTON

P.T. OBEREM* and F.S. MALAN**

ABSTRACT: Oberhem, P.T.; Malan, F.S. **A new cause of cattle mange in South Africa : *Psorergates bos* Johnston.** *Journal of the South African Veterinary Association* (1984) **55** No. 3, 121-122 (En). Section of Entomology, Veterinary Research Institute, P.O. Box 12582, 0110 Onderstepoort, Republic of South Africa.

A herd of Bonsmara bulls in the Eastern Transvaal was found to be suffering from mange. Scrapings were made and the mites that were collected was identified as *Psorergates bos.*

Key words: *Psorergates bos,* cattle, Republic of South Africa.

INTRODUCTION

Psorergatic acariasis of sheep, *Psorergates ovis* Womersley, was first recorded in South Africa by Fiedler & du Toit[2] in 1954 and has subsequently been found in all 4 provinces in the country.

Zumpt[7] listed several other species of the genus *Psorergates* that have been found in South Africa, for example *P. cercopitheci* Zumpt & Till from vervet monkeys; *P. hystrici* Till from the crested porcupine, and *P. oettlei* Zumpt & Till from the multimammate rat. Psorergatic acariasis is also known to occur in other parts of the world on various species of bats and rodents[3] and primates[4 6]. It was, however, unkown in domestic cattle until Johnston[3] described a new mite, *P. bos*, from cattle in USA. Mange of cattle caused by this mite was later found to be fairly widely distributed in the USA[5].

CASE REPORT

The discovery was made on the farm, Weltevreden, 25° 34' S and 31° 10' E, of a Bonsmara stud breeder in the Nelspruit District of the Eastern Transvaal lowveld. Twenty stud bulls were kept in a small camp and regularly handsprayed against ticks with the recommended concentration of Flumethrin (Bayticol Bayer, Reg No G 489 Act 36/1947). These bulls slowly developed symptoms of pruritis and alopecia, and later even showed a loss in live mass. The lesions varied in severity from a mild focal loss of hair to large areas of alopecia. Dermal desquamation was apparent where the hair was absent. The lesions were distributed over the whole body, especially on the dorsal third, including the head, neck, shoulders and back. The worst lesions, however, were seen on the rump and ischial areas. (Fig. 1 & 2).

Numerous skin scrapings were made with a scalpel, using glycerine as a lubricant. The mites found were digested in KOH, dehydrated and mounted on glass slides in Eukitt (mounting reagent, D. Kindler, Freiburg West Germany) under coverslips.

IDENTIFICATION

P. bos is almost identical in appearance to *P. ovis*, the Australian sheep itch mite, but it is smaller and the form of its ventral tibial setae is different (Fig. 3)..

Fig. 1 & 2: Typical distribution of skin lesions caused by *P. bos* infestation

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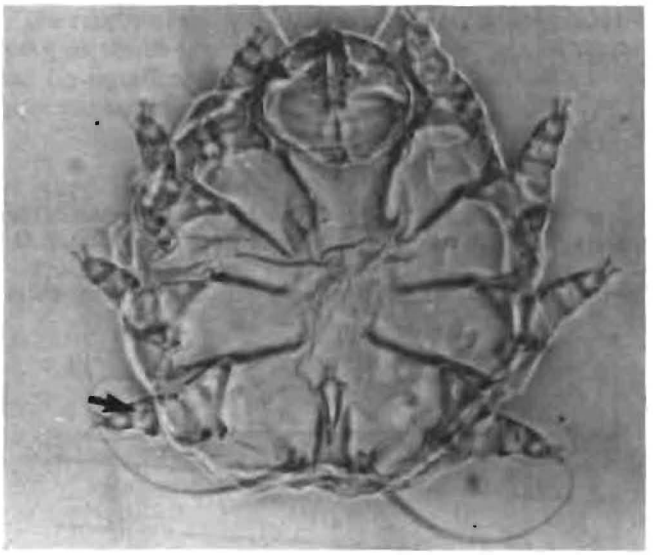

Fig. 3: Ventral view of *P. bos*. Arrow indicates position of ventral tibial seta.

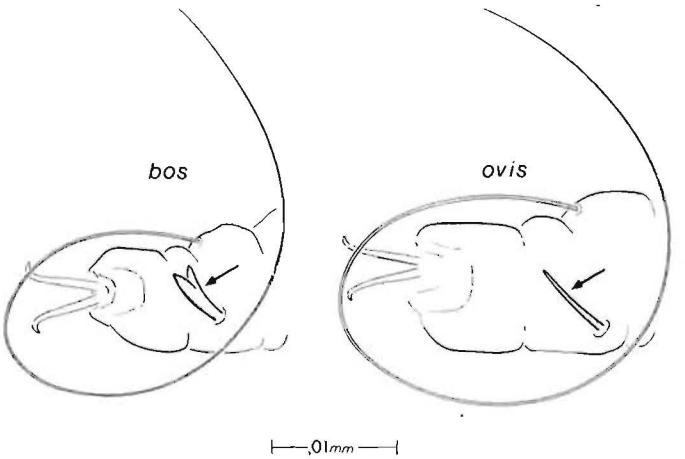

Fig. 4: Left: Stout distally bifid ventral tibial seta of *P. bos*
Right: Spine-like ventral tibial seta of *P. ovis*

Fain[1] gives 177 – 180 μm as the range in length (including gnathosoma) of females of *P. ovis* from South Africa. According to Johnston[3] *P. bos* ranges from 135 – 145 μm in overall length.

P. ovis[1,3] and *P. bos* differ consistently in the form of the ventral tibial setae of all the legs[3]. In *P. ovis* this seta is spin-like whereas in *P. bos* it is stout and bifid distally (Fig. 4).

TREATMENT

Ivermectin (Ivomec, injectable, MSD, Reg No. G 541 of Act 36, 1947) at the recommended dose of 1 ml/50 kg subcutaneously resulted in dramatic recovery of the affected animals. Pruritis and scratching ceased and their hair grew again.

DISCUSSION

The lesions seen on these bulls were far more severe than those described by either Johnston[3] or Roberts & Meleney[5]. According to these authors pruritis was not seen to be a constant feature in contrast to the obviously severe itching reported here.

It is always extremely difficult to collect *Psorergates* mites in the field because they are so small. This is probably why *P. bos* has only now been found for the first time in South Africa. Only very careful examination of cattle in future will make it possible to determine the distribution and importance of this mite in South Africa and elsewhere.

ACKNOWLEDGEMENTS

The technical assistance of Mrs Jackie Matthee is gratefully acknowledged. The authors also wish to thank Mr Rudi Meiswinkel for the illustrations.

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VOLVULUS VAN DIE DUNDERM IN 'N HOND

J.S.J. ODENDAAL*

ABSTRACT: Odendaal J.S.J.; **Volvulus of the small intestine in a dog.** *Journal of the South African Veterinary Association* (1984) **55** No. 3, 123-124 (Afr). 152 Benade Drive, Fichardt Park, 9322 Bloemfontein, Republic of South Africa.

A case of primary volvulus of the small intestines in a dog is presented. This condition is described as rare in the dog. The surgical treatment and positive results are discussed.

Key words: Volvulus, dog, surgical treatment.

INLEIDING

Volvulus is 'n toestand waar 'n deel van die dermkanaal om die lang as van die mesenterium roteer. Omdat die mesenterium se voue die langste is by die jejenum en ileum, vind rotasie gewoonlik in hierdie area plaas. So 'n rotasie kan die bloedvoorsiening in die mesenterium belemmer en dermkanaalobstruksie veroorsaak met gevolglike gasaansameling en timpanie van die dermkanaal. Afhangende van die graad van afsluiting, kan volvulus 'n akute toestand wees wat vinnig tot dood kan lei.

Twedt en Wingfield[2] beskryf 'n toestand van gastriese dilatasie-volvulus in die hond wat hoofsaaklik op die rotasie van die maag dui. Alhoewel hierdie toestand ook as 'n volvulus beskryf word, stem dit ooreen met torsie van die maag.

Volgens Larsen en Bellenger[1] is volvulus van die dermkanaal 'n raar toestand in honde. Die toestand kom veral in aktiewe honde voor. Die simptome kan insluit skok, depressie, anoreksie en vomisie.

GESKIEDENIS

'n Vierjaar-oue Dobermanteef is ingebring vir konsultasie. Die klagte was dat die hond skielik lusteloos geword het en geen kos wou inneem nie. Hierdie verandering was vir die eienaar dadelik opvallend omdat die hond 'n baie lewendige geaardheid gehad het en 'n gulsige eter was. Die eienaar het geen vomisie waargeneem nie.

KLINIESE ONDERSOEK

Tydens die ondersoek is uitsetting van die buik waargeneem. Die timpanie kon duidelik met perkussie vasgestel word, maar was nie so erg soos gewoonlik met torsie nie. Die hond was depressief en het met haar kop na onder gestaan. Die slymvliese was kongestief. Torsie van die maag is oorweeg maar die hond het nie die tipiese akute simptome van torsie getoon nie. Die buik was nie so pynlik nie en die hond het nie respirasieprobleme getoon soos met torsie waar die gasgevulde maag gewoonlik teen die diafragma stoot nie. Die respirasie was feitlik normaal, die harttempo effens verhoog (ongeveer 80) en die koors normaal. Obstruksie deur 'n vreemde voorwerp is ook oorweeg maar 'n behoorlike buikondersoek was as gevolg van die timpanie nie moontlik nie.

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BEHANDELING

Aangesien besef is dat die toestand chirurgiese behandeling regverdig, is voorbereidings direk vir eksploratiewe laparotomie getref. Skokbehandeling is onmiddelik toegepas.

Na verdowing is die hond in 'n ventro-dorsale posisie geplaas en die buik is voorberei vir 'n aseptiese operasie. 'n Snit van ongeveer 200 mm is in die *linea alba* gemaak. 'n Oormatige gasgevulde dermkanaal het dadelik uit die buik gestoot. Die dermwande was dun en feitlik deurskynend.

Weens die gasgevulde dermkanaal wat aanhoudend uitstoot, kon 'n oordeel oor wat presies die oorsaak van die probleem was nie dadelik gevel word nie. Daar is dus besluit om die gas uit die derms te laat ontsnap deur middel van 'n enterotomie. Eers nadat die meerderheid gas ontsnap het en die enterotomie geheg is, kon 'n diagnose van volvulus gemaak word. Die maag was in posisie en die volgende prosedure is gevolg om die aard van die volvulus vas te stel: Die pilorusklep is as landmerk gebruik en toe is met die voorvinger en duim, die dermkanaal kaudaal gevolg. Die duodenum en pankreas was nog in posisie maar hoe verder daar kaudaal beweeg is, hoe meer het die dermkanaal gedraaid voorgekom. Deur in een rigting te beweeg kon die presiese rotasie van die dermkanaal vasgestel en reggestel word. Die dunderm het teen die wysers in gedraai en is met die wysers reggedraai. Die ondersoek met die vingers is deurgevolg tot by die ileosekale klep. Die kolon was weer in posisie. Die rotasie was ongeveer 180 °. Nadat die dermkanaal reggestel is, is die oorblywende gas vir kontroledoeleindes met die vingers vanaf die pilorusklep reg deur die dermkanaal tot by die anus uitgestoot. Die buik is geheg en die hond is vir nog 'n paar uur op aarvoeding gehou.

RESULTATE

Die hond het die eerste 2 dae na die operasie swaar herstel en het aan ernstige depressie gely asook 'n totale verlies van aptyt. Selfs skoon vars water het geen aftrek gekry nie. Vitamiene ("Vitoplex", Centaur), lewerbeskermers ("Bykahepar", BykGulden) en 5 % dekstro-saline ("Viaflex") is binne-aars as ondersteunende behandeling toegedien.

Teen die einde van die 3de dag het sy self begin water inneem en op die 4de dag het sy die eerste keer self 'n bietjie begin eet. Geen vomisie het tydens hierdie tyd voorgekom nie. Die volgende dag het die hond beter geëet, water gedrink, geen nadelige simptome getoon nie

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en meer lewe begin toon. Die hond is toe ontslaan en 10 dae later is die steke verwyder. 'n Kontroleondersoek is uitgevoer en die pasiënt het in alle opsigte weer 'n normale lewe gevoer. Ses maande na die operasie het daar nog geen herhaling van die toestand voorgekom nie.

BESPREKING

Gedurende dertien jaar van privaatpraktyk was dit die enigste geval van primêre volvulus in 'n hond wat gevind is, terwyl torsie van die maag vergelykenderwys, redelik gereeld voorkom.

'n Interessante verskynsel is dat vomisie nooit gerapporteer of waargeneem is nie. Dit is egter moontlik dat die eienaar net nie vomisie opgelet het nie. Dalk het 'n vroeë aanbieding van die geval of die graad van afsluiting in die dermkanaal nie op daardie stadium vomisie gestimuleer nie. Die relatief-stadige herstel het ook opgeval, nieteenstaande vroeë behandeling. Dieselfde simptome van erge depressie en totale aptytverlies het egter nog 2 dae geduur. Die herstel nadat eetlus herwin was, was vinnig en sonder komplikasies.

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BOOK REVIEW

BOEKRESENSIE

DISEASES OF THE HORSE
A Handbook for Science and Practice

O. DIETZ AND E. WIESNER

Original title: Handbuch der Pferdekrankheiten für Wissenschaft und Praxis, Translated by Turner, A.S. (Fort Collins, Colo) XLIV + 1196p., 505 Fig., partly in colour, 43 Tab, bound, 1984 Price Sfr. 648-/DM561-/US $ 280-75 (ISBN 3-8055-3497-3)

These three volumes have been well-translated from the German version which was published in 1982. They cover an extremely wide field, Part 1 starting with general considerations including examination of the horse and working through various sections such as clinical pathology, anaethesiology, nutrition and digestion, exercise physiology, doping, shock therapy and dressings and bandages. Part 2/I Special Part deals with diseases of the various organ systems including sections on reproductive diseases and breeding practices and diseases of the endocrine and nervous system, the eye, ear, skull, spine and pelvis. Part 2/I Special Part is a continuation of Part 2/I and deals with musculo-skeletal conditions and all the infectious diseases. The latter are divided into diseases of each body system. Parasitism and antiparasitic management is dealt with in one section as are various metabolic disorders, poisonings and evironmental injuries. There is a fairly comprehensive section covering diseases of newborn foals plus a section on inherited defects; the volume closes with vices of the horse and the index for all 3 volumes.

Certain sections are more comprehensive and up-to-date than others and despite having been published first (in German) in 1982, the majority of references (appearing usually at the end of each section) in some sections were up to early 1970's, with few more recent than 1977 and very few in the 1980's. The majority of references are European and are printed in German or other European language although some European, American and other authors better-known to us are listed. The German approach to certain subjects, e.g. colic, with special reference to importance of Strongyle larval migration, I found vague but the surgical approach to treatment of colics was covered in some depth and well illustrated with diagrams. The sections on infectious diseases and poisonings are not geared specifically to the tropics and subtropics; for instance, horsesickness and other orbivirus diseases are not mentioned.

I think this will be a valuable text especially for student libraries for the gleaning of wide general and basic knowledge. Enquine practitioners might consider it as a compact, wide but fairly superficial and in parts outdated reference source for their bookshelves.

J.H. Williams

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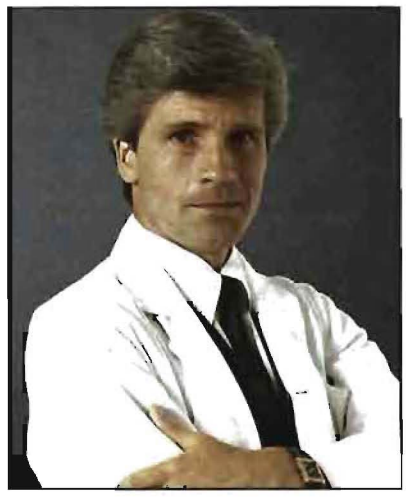

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BOOK REVIEW **BOEKRESENSIE**

SMALL ANIMAL DERMATOLOGY

GEORGE H. MULLER, ROBERT W. KIRK and DANNY W. SCOTT

3rd Edn. W.B. Saunders Company, West Washington Square, Philadelphia PA 19105. Also London, Toronto Mexico City, Rio de Janeiro, Sydney and Tokyo. 1983 pp XV and 889, illustrations 495 (including 71 colour plates) and 78 tables. Price R122,76 (ISBN 0-7216-6609-4).

The third edition is essentially a new book. The addition of D.W. Scott as co-author has contributed significantly to the sections on histopathology, neoplasia, endocrine and immunological disorders. Many new skin disorders are discussed.

The authors devote chapters each to skin structure and function, dermatohistopathology, diagnostic methods and therapy. This is followed by the chapters on bacterial, viral, fungal and parasitic skin diseases, all notable for their completeness. The section on immunological skin disease is really excellent and deals with cutaneous immunology as well as with hypersensitivity, autoimmune and immune-mediated disorders. Of the same high standard is the chapter on endocrine cutaneous disorders. Congenital and hereditary skin defects, acquired alopecias, pigmentary abnormalties, keratinization defects, psychogenic dermatoses, environmental and nutritional skin diseases are each dealt with in detail while miscellaneous disorders are grouped in a separate chapter. The section on skin neoplasia is complete and deals with primary and secondary skin neoplasms as well as the non-neoplastic tumours. As in the previous editions there is a section on comparative dermatology where some skin diseases of small animals are compared to its counterpart in human medicine. The last chapter, devoted to the chronology of veterinary dermatology is followed by a handy appendix on dermatologic drugs and equipment – some of which may be known under different trade-names locally, and an excellent glossary.

The book should be of immense value to the small animal clinician and is an absolute must for every teacher in veterinary medicine and pathology as well as those interested in veterinary dermatology. It will be a sound investment for any student in veterinary science who will benefit from the illustrations, which are of high quality and standard, and the fairly concise and easily comprehensible text. The retention from previous editions of the schematic indications of the distribution of the lesions on the patient in the various diseases remains an asset as does the adequate reference lists given on each topic to assist further reading. The book is very well indexed and the reader should not experience any difficulty in locating a specific subject.

I.B.J. van Rensburg

BOOK REVIEW **BOEKRESENSIE**

KRANKHEITEN DES PFERDES EIN LEITFADEN FÜR STUDIUM UND PRAXIS

HANNS-JÜRGEN WINTZER

Verslag Paul Parey 1982 pp 558, Price DM 196 (ISBN 3-489-60416-4)

This multi-author book of 558 pages was planned to fill an obvious gap in German veterinary literature, namely the lack of a comprehensive scientific guide to the diseases of horses. Twelve authors from the Universities of Berlin, Utrecht, Berne, Vienna and Saskatchewan collaborated to produce the volume.

As the Editor, Professor Dr Hanns-Jürgen Wintzer points out in his foreword, there are numerous texts devoted to surgical diseases of horses, whereas there is a conspicuous lack of works on modern concepts of internal diseases and infertility. Consequently, most of the book is devoted to internal medicine and reproductive disorders; the remaining 131 pages contain concise description of the signs, diagnosis and treatment of surgical conditions – a truly monumental task, as any surgeon will agree.

The scope of this book obviously dictated the style and presentation, but it would be wrong to dismiss it as a mere "guide" to the diseases, as the title might suggest. The authors have succeeded in packing an enormous amount of information between the covers. High quality photographs illustrate many of the sections – there are 212 plates in black-and-white plus 24 high quality colour pages, each of which has 6-8 different prints.

One chapter of 65 pages is devoted to bacterial, viral and protozoal diseases. While this may appear to suggest sub-minimal coverage of very wide fields, basic facts are given regarding the signs, diagnosis and therapy of the various conditions.

The chapters on the urinary system and on metabolic diseases would have benefitted from more detailed descriptions of diagnostic techniques and findings. The section on the diagnosis of pregnancy in mares does not include the use of ultrasonic scanning.

This is a useful and praiseworthy book for practitioners and students with a knowledge of German. It more than succeeds in its aim of providing, as the subtitle indicates, a guide for the study and practice.

A. Littlejohn

MANIFESTATIONS OF BOVINE PARAFILARIASIS

P.M. KRETZMANN, H.G. WALLACE and D.B. WEAVER*

ABSTRACT: Kretzmann P.M.; Wallace H.G.; Weaver D.B. **Manifestations of bovine parafilariasis.** *Journal of the South African Veterinary Association* (1984) **55** No. 3, 127-129 (En). Division of Veterinary Services, Department of Agriculture, P.O. Box 206, 3680 Cato Ridge, Natal, Republic of South Africa.

Three bovine carcases displaying inter- and intramuscular, intra-abdominal and intra-thoracic lesions attributable to *Parafilaria bovicola* infestations were selected for further inspection at Cato Ridge abattoir. The distribution of these lesions in the carcase is described. The condition was confirmed by the presence of eosinophils in scrapings and histological sections of the lesions and by the recovery of the parasite.

Key words: bovine, parafilariasis, inter-muscular, intra-muscular, intra-abdominal, intra-thoracic.

INTRODUCTION

The lesions caused by *Parafilaria bovicola* in the subcutis of infested bovines was first described in South Africa by Pienaar & Van den Heever[3]. Subsequently various authors have expounded on the macroscopic pathology seen and the distribution of these lesions throughout the carcase[1 2 4-7 8]. Van den Heever et al[4] described the frequency and distribution of 129 parafilarial lesions on beef carcases on horizontal and vertical planes. They found that the lesions appeared in the greatest numbers on the shoulders, withers and thoracic areas and declined in frequency in cranial and caudal directions. On a vertical plane the lateral and dorsal aspects of the carcase were found to be more prone to infestation that the ventral third of the carcase. This phenomenon is confirmed by observations at this abattoir.

Initial reports[3 4] described the subcutaneous migration of the parasite but no mention was made of damage to deeper structures. Viljoen et al[7] recorded that some of the fibres of the superficial muscles bordering on inflamed areas in the subcutis showed degenerative and regenerative changes. More recently Wallace et al[8] related that the superficial lesions may be accompanied by the same greenish-yellow discolouration of the fascia between the muscles or less frequently within the muscle tissue itself and occasionally as greenish-yellow granulomas of up to 10 mm in diameter in the musculature. They mention, too, that lesions have been detected in the sub-pleural tissue, the mediastinum, the sub-peritoneal tissue and in the perirenal fat.

The aim of this article is to describe the distribution and extent of lesions seen in three beef carcases naturally infested with *Parafilaria bovicola.*

MATERIALS AND METHODS

The method of inspection at this abattoir has been described by Wallace et al[8].

Three beef carcases (all oxen, graded "Super") were selected at secondary inspection for further examination. The selection was based on the manifestation of the condition and the presence of adult bovicola on the carcase. The worst affected sides of each carcase were systematically deboned and examined for the presence of lesions. Scrapings were made of suspected lesions, stained with Stévenels stain (made up according to the 6th edition of Biological Staining Methods, by G.T. Gurr) and examined microscopically for the presence of eosinophils.

Histological sections were made of all intra-muscular, intra-abdominal and intra-thoracic lesions detected. The sections were stained with Haematoxylin/Eosin and the micro-pathology was examined for eosinophil infiltration as well as for the presence of parasites and evidence of migratory tracts.

One adult parasite from each carcase was collected and identified.

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RESULTS

Carcase No. 1

This carcase showed severe extensive superficial lesions, characteristic of those caused by *P. bovicola* infestation[1-8]. These lesions presented as greenish-yellow pigmentary changes in the subcutis. The texture was oedematous and varied from slimy to soapy in nature. A metallic odour prevailed on close olfactory examination.

Extensive involvement of the inter-muscular fascia was especially prominent on the left side of the neck and withers. Muscular tissue bordering on these lesions showed pale degenerative changes to a depth of ± 20 mm. The medial aspect of the *M.serratus dorsalis* and the ventro-lateral aspect of the *M.rhomboideus* were most prominently affected.

Sections of muscle tissue revealed that an extensive eosinophilic infiltration was the prominent feature with a moderate lymphocyte and macrophage influx. Hyaline degeneration of the muscle fibres together with vacuolation and lysis was associated with the cellular influx. This micropathology is concommitant with the description given by Viljoen & Coetzer[7].

One adult worm was found in the subcutis of the neck.

Carcase No. 2

The extensive superficial lesions, as described in carcase No. 1, were also prominent in this carcase. These lesions extended into the facia between the muscle groups of the neck on the right side of the carcase. Intra-muscular lesions were detected on the medial aspect of the *M.serratus dorsalis,* on the lateral aspect of the *M.rhomboideus* and on the dorso-lateral aspect of the *M.trapezius.*

Likewise the inflammation was present in the fascia underneath the *M.obliquus externus abdominis* caudal to the last rib. The characteristic myopathy was also seen in those aspects of this muscle in contact with the lesions in the fascia.

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The lesions in the fascia in this region extended into a greenish-yellow discolouration ± 100 × 100 mm in size underneath the parietal peritoneum. The adjacent perirenal fat displayed the characteristic pathology which extended bilaterally from the pelvic rim to the cranial aspect of the kidneys where it was especially prominent. The lesion was within the kidney parenchyma were evident.

Scrapings from the subperitoneal and adipose tissue showed a marked accumulation of eosinophils. Sections of the fat tissue showed this accumulation of eosinophils especially within the connective tissue surroundig the adipose cells. They were especially prominent around the blood vessels within the trabeculae.

One adult worm was found in the subcutis of the neck.

Carcase No. 3

Once again the severe subcutaneous lesions were prominent.

Intra-muscular lesions, as described in carcase No. 1 and 2, were detected in the *M.intercostale externi* (dorsal part of the second and third intercostal spaces) and on the lateral and ventral aspects of the *M.longissimus dorsi* muscle at this level. This was on the right side of the carcase. Also on this side of the carcase the lesions within the fascia extented underneath the *M.latissimus dorsi* and deep within the fascia surrounding the *M.longissimus dorsi* on a level with the caudal border of the scapula. This lesion extended into a greenish-yellow discolouration ± 300 by 200 mm in size underneath the parietal pleura. This discoloration was especially prominent on the dorsal aspect of the thoracic cavity. The greenish-yellow oedematous lesion was present in the mediastinum with mediastinal lymph node involvement. The lymph nodes were enlarged, a pale greenish colour and oedematous. The pericardium on the right side was also oedematous in texture and showed the charactaristic greenish-yellow pigmentation. The lesion was also adherent to the visceral pleura on the lung surface at two foci, ± 2 cm in diameter.

One degenerate granuloma ± 10 mm in diameter was present in the subcutis just caudal to the border of the scapula.

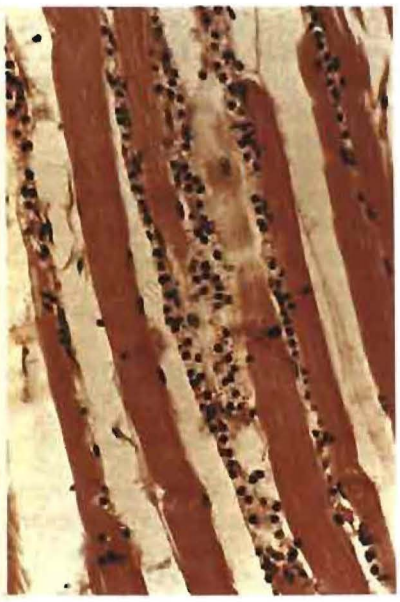
A section through this granuloma showed a gravid female worm surrounded by a fibrous capsule. Microfilariae were present within the worm, some of which had penetrated into the surrounding host tissue. The typical granulomatous reaction has been previously described[3 6].

DISCUSSION

It is recognised that the majority of lesions attributable to *P. bovicola* infestation are superficial and localised and necessitate removal of the affected tissue only. The rest of the carcase is then passed as fit for human consumption. Not infrequently, however, the superficial lesions are so extensive as to involve the majority of the carcase surface. These carcases invariably display intermuscular lesions of varying intensity within the facia and are often accompanied by lesions in the muscle adjacent to the affected fascia. Sub-peritoneal, abdominal, sub-pleural and thoracic lesions cannot be regarded as rarities although their intensity is often limited to small localised areas. Lesions in the pelvic cavity have also been detected.

It is these latter manifestations of parafilariasis that often necessitates total carcase condemnation.

Internal lesions attributable to *Parafilaria bovicola* infestation have also been detected at City Deep and



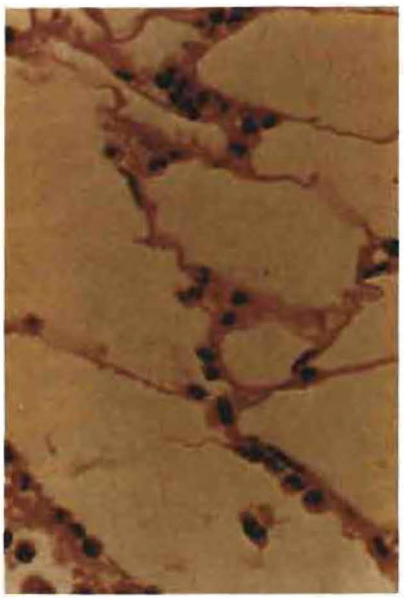


Fig. 1: A section through an intra-muscular parafilarial lesion. Note the eosinophil accumulation and the hyaline degeneration and lysis of the muscle fibres. X400

Fig. 2: A section through the lesion in the peri-renal adipose tissue sampled from carcase No. 2. Note the eosinophil accumulation. X800

Fig. 3: A section through the gravid female worm within the granuloma described under carcase No. 3. Note the microfilariae. X320

Pretoria abattoirs (Dr. G Vogelzang, 1983, Division of Veterinary Services, personal communication).

The economic importance of this condition cannot be underestimated. Weavet et al[9] state that affected animals slaughtered at Cato Ridge abattoir originate from virtually all districts within the province of Natal and Kwa-Zulu and some surrounding areas including some areas from which it was not previously recorded.

Wallace et al[8] recorded a 585 % increase in compounded total carcase condemnations for parafilariasis at this abattoir from the year July 1979 / June 1980 to July 1981 / June 1982. In July 1979 / June 1980 this condition was responsible for the condemnation of 0,054 % of the total cattle slaughtered during that year whilst in July 1981 / June 1982, the figure increased to 0,370 %. This statistic remains at a high level. For the year July 1982 / June 1983, 632 beef carcases were condemned for parafilariasis at this abattoir (0,303 % of the total cattle slaughtered) an amount representative of a considerable loss to the meat industry.

ACKNOWLEDGEMENTS

The authors wish to express their gratitude to: Dr P. Oberem of the Veterinary Research Institute, Onderstepoort, for the photographs; Mrs D. Hook for the typing of the manuscript; and the Allerton and Cato Ridge members of the Laboratory Liaison Committee (especially Dr J. Kitching and Mrs Anne Selmer-Olsen) for their enthusiasm and assistance with the compilation and criticism of this article. In addition, the authors acknowledge the role played by Maybaker (SA) (Pty) Ltd., in making the publication of the photographs possible.

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BOOK REVIEW BOEKRESENSIE

THE ACUTELY TRAUMATIZED SMALL ANIMAL PATIENT

T.H. BRASMER

1st ed. W.B. Saunders, Philadelpia 1984 pp. xi + 162, Figs. 59, Price R62,50, (ISBN 0-7216-1917-7)

The author states that his book is intended for practitioners, who, by necessity, have to deal with trauma cases without the technical support available in an academic institution.

The first chapters of the book deal with stress and the physiologic response to trauma. The latter is highly comprehensive and contains previously unpublished experimental data of the author on the effects of blood loss. The chapter on the examination of the trauma patient is short and straightforward. The last five chapters deal with the ABCDE of trauma: Airway, Bleeding, Central Nervous System, Digestive System and Excretory System. In each chapter a review is given of the conditions that may occur, without going into excessive detail. The author gives his approach to the fifferent problems. The surgery indicated for the repair of certain conditions such as pneumothorax is well described and illustrated. For other conditions only the surgical principle is given, which makes it necessary for the unexperienced surgeon to consult other textbooks. Each chapter is ended with a short list of references, often taken from the human literature.

The overall impression that one gets when reading this book, is that the author knows what he is talking about. The 35 years of clinical experience of the author, combined with a sound knowledge of the pathophysiology of trauma, contribute to the high standard of this book. It is highly recommendable to small animal practitioners and interested students. The price of this book however is not in proportion to its small size and relatively small number of illustrations.

F.J.M. Verstraete

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CASE REPORT **GEVALVERSLAG**

A CASE OF A HIGHLY INVASIVE CARCINOMA OF A SALIVARY GLAND IN A CROSSBRED DOG

G.J. LOUW* and SELMA J.E.M. VAN SCHOUWENBURG**

ABSTRACT: Louw G.J.; van Schouwenburg Selma J.E.M. **A case of highly invasive carcinoma of a salivary gland in a crossbred dog.** *Journal of the South African Veterinary Association* (1984) **55** No. 3, 131-132 (En). Department of Anatomy, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

A 13 year-old male crossbred dog was presented with a unilateral swelling of the facial region, severe pain when the mouth was opened, and chronic otitis. Upon palpation it appeared that the right mandible had been fractured. At necropsy the head revealed a large tumour which had invaded the mandible and had caused areas of osteolysis. Histopathology revealed a solid highly anaplastic carcinoma of one of the serous salivary glands.

Key words: dog, carcinoma, serous salivary gland, mandible, otitis.

Fig. 1: Right lateral view of the head of the dog. The cheek has been incised to expose to tumour (T).

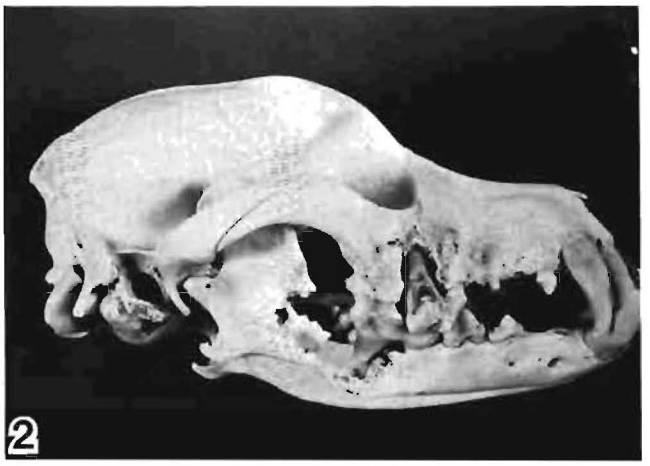

Fig. 2: Right lateral view of the skull, showing the destruction of bone.

HISTORY

A 13 year-old crossbred Terrier-type male dog was presented for clinical examination. The owners had noticed that the right side of his head was very swollen, that his appetite was steadily decreasing, and that he was salivating excessively. The dog had a history of chronic bilateral otitis which alway responded well to treatment, but constantly recurred.

CLINICAL SIGNS

Attempts to open the dog's mouth for examination caused severe pain and therefore general anaesthesia was administered in order to enable the oral cavity to be thoroughly explored. Externally all the lymph nodes and salivary glands at the angle of the right mandible were enlarged. An extensive tumour was visible caudally on the right side within the oral cavity, which enveloped the upper and lower premolars and molars, and extended into the pharynx (Fig. 1). It seemed that the mandible had been fractured at the site of the tumour. It was then decided to perform euthenasia on the dog.

PATHOLOGY

Specimens of the tumour were collected in 10 % buffered formalin for histopathology. The skull was boiled clean and defatted for examination. The right ramus of the mandible had undergone necrosis and lysis of the bone with resultant loss of the molar teeth (Fig. 2 – 4).

The maxillary bone on the right had undergone lysis around the roots of the fourth premolar tooth and the first molar tooth (Fig. 5). Osteolysis was also evident on the ventral surface of the right bulla tympanica and the bony part of the eustachian tube, resulting in exposure of the middle and inner ear to the parotid region (Fig. 6). These lesions would explain the recurrent chronic otitis.

Histopathology revealed the mass to be highly

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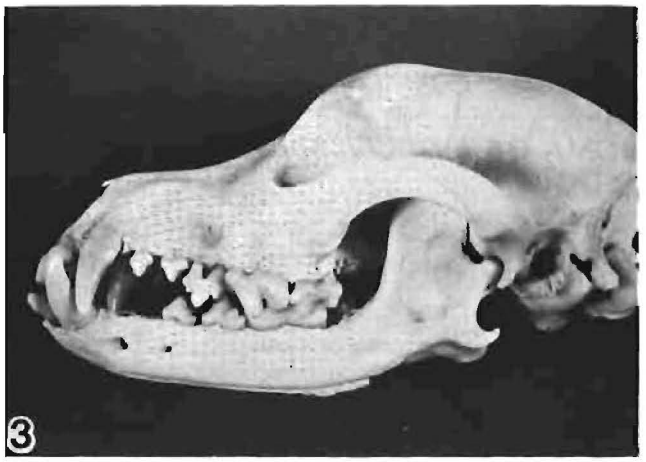

Fig. 3: Left lateral view of the skull, which was unaffected by the tumour.

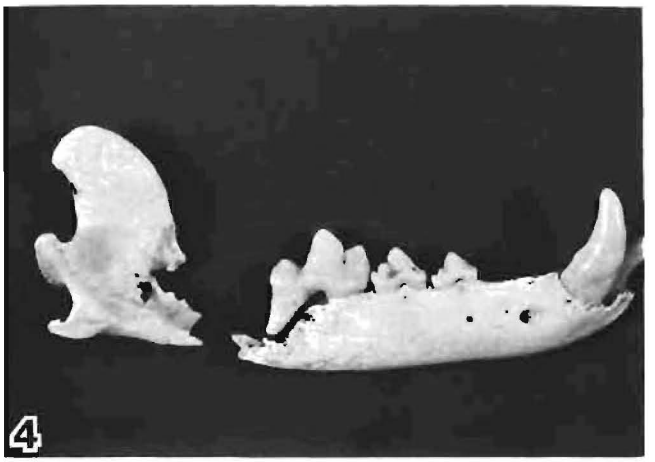

Fig. 4: The right mandible, showing lysis of the bone and loss of teeth.

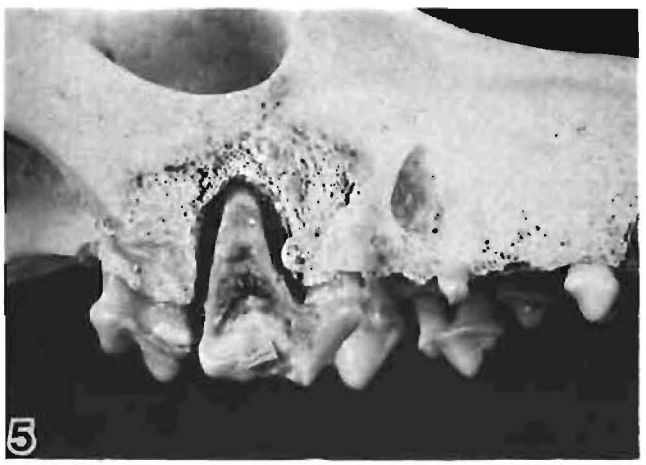

Fig. 5: The right maxillary region, showing the exposed roots of the fourth premolar tooth and the first molar tooth.

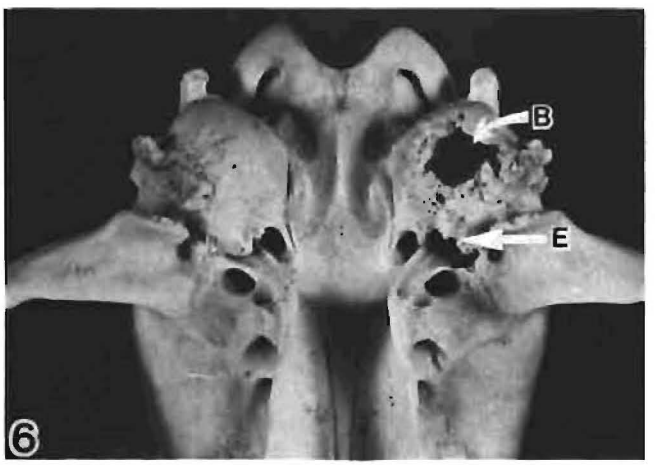

Fig. 6: Ventral view of the skull, showing destruction of the floor of the bulla tympanica (B) and a portion of the osseous part of the eustachian tube (E).

anaplastic solid carcinoma of one of the serous salivary glands. There were emboli in the blood vessels and extensive necrosis within the carcinoma, and a considerable formation of connective tissue in reaction to the tumour cells.

DISCUSSION

According to Jubb and Kennedy[1] neoplasms of the salivary glands are rare in all animals species, but occur with greater frequency in the dog and the parotid is more often involved. These tumours are almost exclusively in aged animals, the majority grow rapidly and are painful. The carcinomas are classified as squamous, mucoepidermoid, or adenomatous. It is often difficult to appreciate the malignancy of the adenomatous type.

The histology of salivary gland tumours is widely diverse. According to Jubb and Kennedy[1] there is often a pseudocystic dissolution histologically in which tumour cells with clear vescicular cytoplasm rupture to form cyst-like spaces, which contain secretions.

ACKNOWLEDGEMENTS

We wish to express our appreciation to Dr L. Lange, Department of Pathology, for her assistance in interpreting the histopathology; to Sr A. Kruger, Department of Anatomy, for preparing the sections for histopathology; to Mrs H. Smit for the photography; to Mrs M. van Aswegen for typing the manuscript; and to Mr M. Ramatshela for the preparation of the skull.

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CASE REPORT GEVALVERSLAG

DISPROVAL OF AN APPARENT GOAT X SHEEP HYBRID

U. MÄRKI and D.R. OSTERHOFF*

ABSTRACT: Märki U.; Osterhoff D.R. **Disproval of an apparent goat × sheep hybrid.** *Journal of the South African Veterinary Association* (1984) **55** No. 3, 133-134 (En). Department of Zootechnology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

Samples of whole blood of a Boergoat buck (60,XY), and ewe (54,XX) and her heterosexual twins were cytogenetically examined. The female offspring had hair and seemed to be a cross between the two animals. However, both twins possessed 54 chromosomes, including 6 large submetacentric autosomes, whose R- and G-banding patterns were identical to those of the ewe.

Key words: Chromosomes, goat, sheep, R- and G-banding.

INTRODUCTION

Detailed descriptions of successful matings between goat and sheep have been lacking until recently. Eldridge et al.[2] gave brief morphological and cytogenetical description of a female goat which gave birth to twins, one of which was female of odd appearance carrying both hair and wool. The lymphocyte cultures of the odd female showed that all metaphase spreads had 57 chromosomes including 3 large submetacentrics as would be expected in a goat by sheep cross. Unfortunately no other reliable report could be found which mentioned crosses of the two different species.

The present paper presents the results of a cytogenetical examination of an apparent goat × sheep hybrid.

MATERIALS AND METHODS

Heterosexual twins were born of an ewe after an observed mating with a Boergoat buck at Bloemspruit in July 1983. The male twin looked like his mother while the female one had hair and pointed to a cross.

Peripheral blood was obtained from the Boergoat buck, the ewe and the twins. Peripheral blood lymphocytes were cultured by the method of Moorhead et

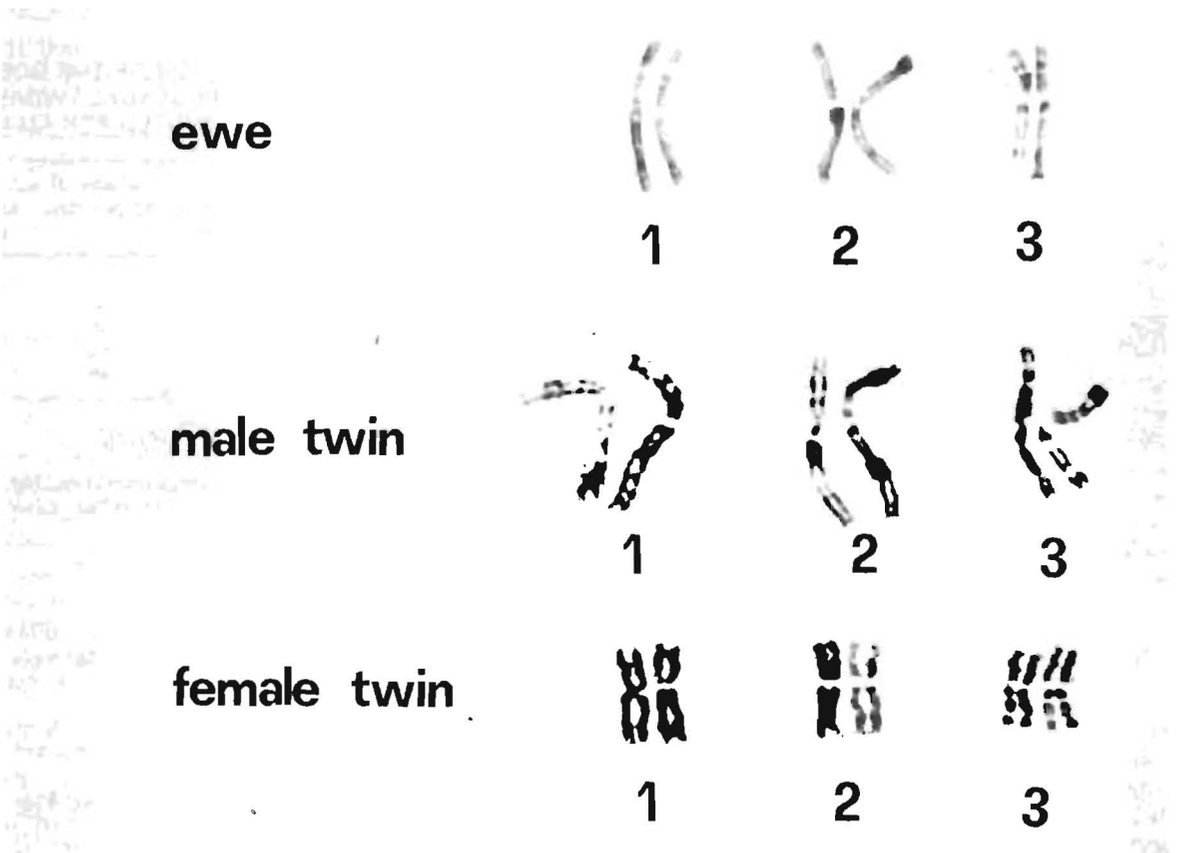

Fig. 1: G-banded submetacentric autosomes of the ewe twins

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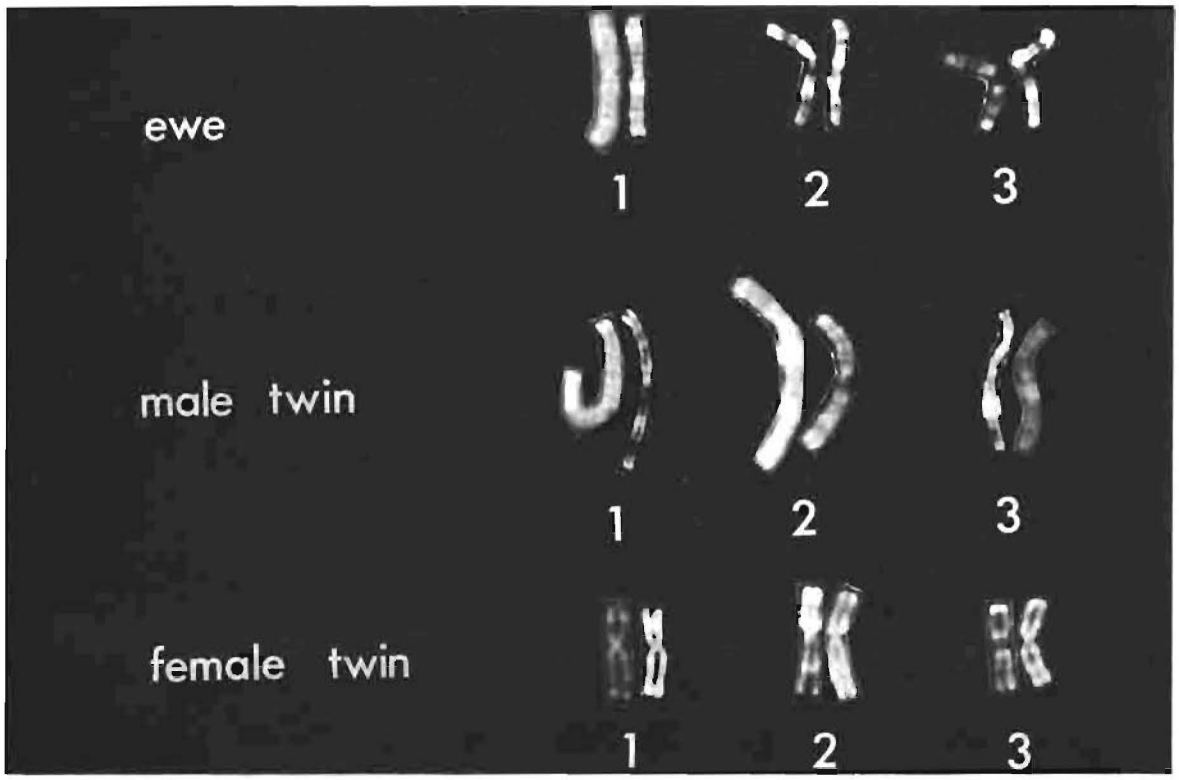

Fig. 2: R-banded submetacentric autosomes of the ewe and twins

al.[4]. The preparations were analyzed by conventional Giemsa staining and also by a modified Giemsa-banding technique[5] and R-banding technique[1]. The Giemsa staining was used for counting of chromosomes while the G- and R-banding patterns of the submetacentric chromosomes were used for analysis of occured translocations in the offspring.

RESULTS

The results of the karyotypic analysis of the Boergoat buck, the ewe and the heterosexual twins are summarized in Table 1. The Giesma stained mataphase of the parents do not show any differences to the standard chromosome number and shape of goat and sheep respectively. The heterosexual twins have the same karyotype as their mother, 54 chromosomes, including 6 large submetacentric autosomes. The Giesma-stained metaphases of the offspring cannot exclude occured translocations leading to submetacentric chromosomes. The G- and R-banded submetacentric chromosomes in Fig. 1 and Fig. 2 possess the same G-banding pattern as the standard G-banded karyotype of the sheep, *Ovis aries*[3] and the R-banding pattern of the mother. Beyond all doubt, the father of the offspring is a ram and not, as suspected a Boergoat.

DISCUSSION

It was very easy to analyze cytogenetically the apparent goat × sheep hybrids because the number and shape of the chromosomes of these two species differ quite considerably. Both twins possessed 54 chromosomes per lymphocyte cell, including 6 large submetacentric chromosomes and both showed the same banding pattern as the chromosomes of the mother. Any translocations could be excluded.

Although the number of chromosomes differs with 6 between goat and sheep, Eldridge et al.[2] found a goat × sheep hybrid with 57 chromosomes including 3 large, submetacentrics as would be expected in a goat (58 acrocentric autosomes) by sheep (52 autosomes, including 3 pairs of submetacentrics) cross.

Table 1: KARYOTYPIC ANALYSIS OF THE BOERGOAT BUCK, EWE AND HETEROSEXUAL TWINS (30 LYMPHOCYTE CELLS COUNTED PER ANIMAL)

	No. cells with		Shape of autosomes	
	54 chromosomes	60 chromosomes	acrocentric	submetacentric
Boergoat buck		30	58	
Ewe	30		46	6
♂ twin	30		46	6
♀ twin	30		46	6

ACKNOWLEDGEMENTS

The authors thank Dr P. Steyn from Bloemfontein and Mr A. Pienaar from Bloemspruit for the opportunity to collect blood and Mrs H.H.C. Pretorius for technical assistance.

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IDENTIFICATION OF HELMINTHS IN RUMINANTS AT NECROPSY

R.K. REINECKE*

ABSTRACT: Reinecke R.K. **Diagnosis of helminths in ruminants at necropsy.** *Journal of the South African Veterinary Association* (1984) 55 No. 3, 135-143 (En). Faculty of Veterinary Science, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

The shortcomings of faecal worm egg counts as a method of diagnosis are discussed. The best method of making a diagnosis of helminthosis is a necropsy. The equipment necessary and technique of carrying out an autopsy are described in detail. Emphasis is laid on the common nematodes which inhabit the gastro-intestinal tract and their relative sizes are illustrated. A brief description of the common nematodes, cestodes and trematodes of domestic ruminants adequate for a preliminary diagnosis is given in tabular form. Full host parasite lists and the number of worms of the most common species necessary to cause clinical signs and even death are tabulated. Emphasis is laid on microscopical examination to recover and diagnose helminths.

Key words: Nematodes, trematodes, ruminants, diagnosis, necropsy, lethal worm burdens.

INTRODUCTION

The basic ecological concept is that almost every animal in a flock or herd is infested and the environment is always contaminated[2]. This means that all animals are passing helminth eggs in the faeces onto the pasture every day, with the possible exception of a few days after treatment – some veterinarians regarded faecal worm egg counts as a reliable method of diagnosis, without taking the variation in egg production and the other factor into consideration[3].

Female worms lay eggs at a different rate and the common strongyle worms may be grouped as follows[3]:

Eggs/Female/Day	Genus	Definition
5 000 - 10 000	*Oesophagostomum* and *Haemonchus*	Very high egg production
3 000 - 5 000	*Chabertia* (Very erratic in the author's experience)	High egg production
600 - 800	*Bunostomum* and *Gaigeria*	Medium egg Production
200	*Cooperia, Ostertagia* and *Trichostrongylus*	Low egg production
50 or less	*Nematodirus*	Very low egg production

All these figures are approximate. Where there are only a few females present they lay more eggs compared with females under conditions of overcrowding. The age and immune status of the host, physical consistency of faeces etc, – all influence worm egg counts.

Larval cultures

Very few, if any, veterinarians ever make a faecel culture and with the exception of the eggs of *Cooperia, Bunostomum, Gaigeria* and *Nematodirus* which have a characteristic shape, the other genera can only be identified by harvesting infected larvae from faecal cultures and thereafter converting the worm egg counts to the respective genera. Unfortunately this is a slow process and takes 6-7 days before all the infective larvae are available for diagnostic purposes.

The significance of egg counts

It is obvious that an empirical decision on the significance of egg counts traps the veterinarian into making mistakes. Examples are *Haemonchus* or *Oesophagostomum* where 5 000 eggs per gram is a very light infestation whereas it is probably fatal in animals with *Ostertagia* or *Trichostrongylus*. The absence of *Nematodirus* eggs in faecal egg counts by no means indicates that this worm is absent because eggs are present for a few weeks only in suckling lambs, are seldom found in weaned sheep and rarely if ever, in older wethers or ewes.

There is only one good method of diagnosing worms and that is to perform a thorough necropsy.

Fig. 1 Aparatus for necropsy lying on a heavy duty PVC ground sheet.
A, pipette (25 ml) with plastic tube; B, bowel scissors; C, pruning shears; D, butcher's knife; E, ladle; F, plastic hose with shower rose; G, twine; H, iodine solution (45%); I, stainless steel tray painted black; J, measuring cylinder (100 ml); K, detergent; L, formalin; M, 1ℓ flask graduated from 100 – 500 ml; N, honey jar graduated from 10 – 50 ml; O, 14 ℓ bucket graduated at 10 ℓ and 5 ℓ; P, special sieving bucket with sieve attached (apertures 150μm) (Helena Smit, University of Pretoria)

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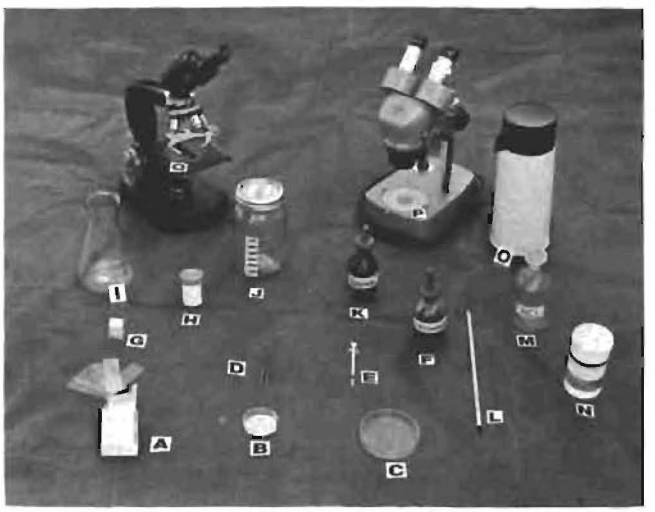

Fig. 2 Apparatus for recovery, identification and counting of worms.
A, microscope slides; B, petri dish with 2 mm squares; C, petri dish with parallel lines 8 mm apart; D, forceps with fine points; E, 1ml syringe with a fine needle; F, lactophenol; G, coverslips; H, sample bottle (45 ml); I, Ehrlenmeyer flask 500ml; J, glass jar 1 ℓ graduated from 100-500 ml; K, iodine solution 45%; L, thermometer 0-100°C; M, HCl (commercial); N, pepsin scales; O, thermos flask 1 ℓ; P, student stereoscopic microscope (Vickers); Q, compound microscope (Helena Smit, University of Pretoria)

Necropsy

Equipment (Reinecke[5]) (Fig. 1 and 2)

- Ground sheet heavy duty polyvinyl chloride
- Butcher's knife
- Long-handled pruning shears (800 mm)
- Bowel scissors (210 mm)
- Tray stainless steel painted flat black (45x27x6 cm) or pie dish (pyrex type) (230x130x50 mm)
- String or twine (2-5 mm diameter)
- Butcher's saw (600 mm)
- Buckets (14 ℓ) heavy duty (2 or 3)
- Iodine concentrated solution. Prepare by adding hot water to 70-90 g KI and then slowly dissolve 40 g I crystals in the solution. Finally add water to a final volume of 1 ℓ
- Formalin – 10% solution i.e. 4% formaldehyde solution
- Sieve(s) Fine stainless steel or copper mesh (150 μm apertures) or if 3rd stage (L_3) or 4th stage (L_4) larvae need to be recovered apertures of 38 μm. The diameter of the sieve is 22cm
- A high quality plastic pipe is inserted into the base of a 14 ℓ bucket and the pipe is machined to the following dimensions:
  60 mm high, outer diametre 205 mm, inner diametre upper part 190 mm, lower inner diametre 200 mm. Fourteen mm from the inner lower edge of the pipe a ledge or seat is machined which makes a water-tight fit for the Endicott (type) sieve when inserted upside down. The pipe is glued into the base with a silicon sealer and on the outer surface of the pipe 3 suitcase type clips have been riveted and 3 stainless extensions

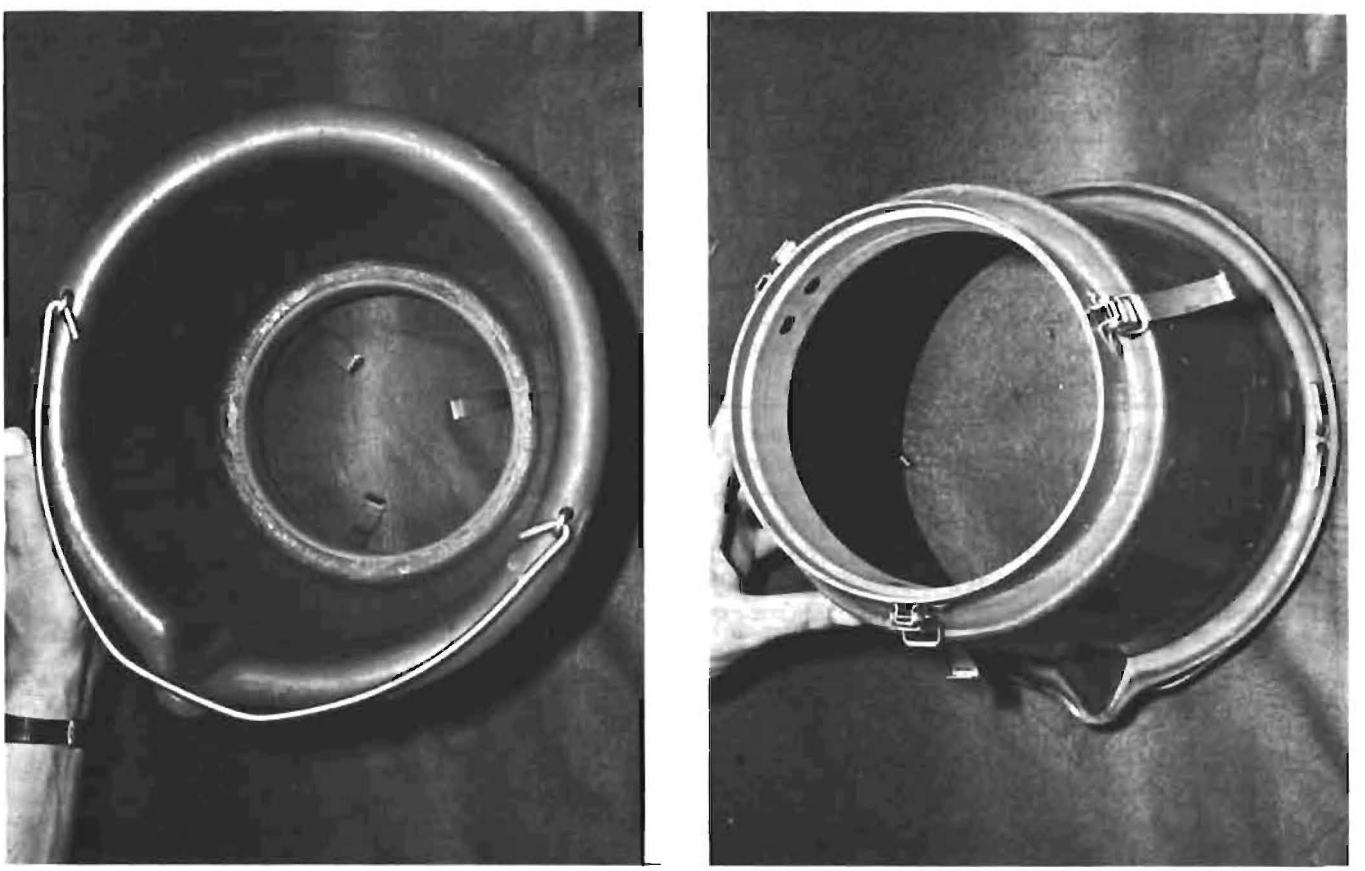

Fig. 3 Special bucket with a machined plastic pipe inserted in the base to allow the sieve to be placed in it for sieving; Upper and lower views (Helena Smit, University of Pretoria)

(60 x 15 mm) have been attached which clamps the sieve firmly in position when in use (Fig. 3)
- Carpenter's vice
- Disposable syringe 1 ml with a fine (26 gauge) hypodermic needle. The tip of the needle is pressed firmly on a steel or glazed surface and the tip of the needle burred to make a hook. This is an efficient tool for picking the worms out of the ingesta and transferring them to a slide or plastic bottle. An alternative is a fine pointed forceps (110 mm).
- Ehrlenmeyer flask (500 ml)
- Wide-mouthed glass jars (1 ℓ capacity) with screw caps
- Wide-mouthed glass jars (180 ml capacity) with screw caps
- Plastic or glass specimen bottles (45 ml capacity) flat bottomed with screw caps
- Microscopes student stereoscopic and compound
- Counting chamber a petri dish (80 mm) is placed on lined paper. With a ruler and diamond pencil score lines approximately 8 mm apart on the upper surface of the petri dish using the lined paper as a guide.
- Microscopic glass slides
- Cover slips (22 mm diametre) or for large worms rectangular cover slips (22 x 14 mm)
- Lactophenol solution consisting of lactic acid 20 g (16,5 ml) carbolic acid 20 g, glycerine 10 g (8 ml) and distilled water 20 g (20 ml)
- Surface acting detergent
- Plastic hose with shower rose (or hose attachment) and adaptor for taps
- Metal spoon or ladle
- Glass pipettes (25 ml) with rubber or plastic tube attached to the narrow end
- Pepsin scales
- Hydrochloric acid concentrated (commercial grade)
- Measuring cylinder graduated plastic or glass (100 ml)
- Thermos flask wide-mouthed (1 ℓ or 2 ℓ capacity)
- Thermometre graduated 0-100°C
- Petri dish graduated marked on lower surface of base in 2 mm squares
- Standard textbook either Levine[4] or Reinecke[5] or Soulsby[6] for identification of adult worms

Before carrying out a necropsy mark one of the buckets (with a felt tip pen) at the 5 and 10 ℓ mark by filling to the level with water and marking to outer surface at the appropriate level. Similarly, a 1 ℓ glass jar should be marked from 100 to 500 ml level and a 180 ml glass jar at the 50, 100 and 150 ml levels respectively.

The following procedures should be followed to perform a proper necropsy:-

Remove the left limbs and open the carcass with the butchers knife, cut the thorax with the long-handled shears and remove the ribs on the left side of the carcass. With the butchers knife continue cranially along the throat removing the musculature overlying the trachea, oesophagus and larynx. Open the muscles between the jaws of the mandible and pull the tongue through the jaws of the mandible, pull it ventrocaudally, cut through the hyoid bones and free the larynx, trachea and oesophagus. Make an incision through the dorsal aspect of the thorax freeing the heart and aorta from the spine, cut through the diaphragm and with firm pressure on the tongue and trachea pull the viscera from the carcass in a ventral direction. Cut the anus loose from the surrounding skin, open the pelvis with the pruning shears and/or the butchers saw and free the rectum from the surrounding tissue cranially and remove the entire gastrointestinal tract and pluck from the carcass placing it on the ground sheet.

Important lesions during necropsy should be noted viz. anaemia, hydrothorax, ascites, cachexia as well as any cestode larvae (Hydatids) in the lungs and liver or Cysticercus tenuicollis in the abdominal cavity. Grey lungs (sheep and goats) a black liver (sheep and cattle) or hobnailed liver (goats) are indications of *Schistosoma mattheei* (Bilharzia). If the mesanteric fat is not congealed, search the mesenteric blood vessels for *S.mattheei* and in cattle white connective tissue bands indicate chronic *S.mattheei* infestation. Thickened bile ducts are caused by liver flukes (*Fasciola* spp.)[5] Also search for *Stilesia hepatica* which may cause dilation of the bile ducts. Place the liver, lungs and trachea and heart on a pan for detailed examination at a later stage.

Remove a long bone (femur of humerus) saw down its length and examine the exposed bone marrow (see below).

Open the skull and remove the brain in sheep and look for the larval stage of *Taenia multiceps* (Coenurus cerebralis). Saw the skull in 2 parts down the centre line exposing the nasal cavities and examine for *Oestrus ovis*.

Most worms are confined to certain habitats and busy practitioners will save themselves a lot of time if they divide the organs of the gastrointestinal tract with double ligatures of strong twine or string at the following points:
- The junction of the omasum and reticulum where there is a convenient notch.
- The duodenum caudal to the pyloric valve
- The ileum cranial to the ileocaecal valve
- In sheep and goats draw an imaginary line from the tip of the caecum to the last coil of the *ansa spiralis* and tie a double ligature where this line crosses the decending colon and
- In cattle, tie a single ligature round the rectum cranial to the anus. Subsequently strip the mesentry by hand in the sheep and goats or with bowel scissors in cattle and note the following:

S. mattheei may be released from the mesentric veins or the first 3 m of the mesentry of the small intestine may be oedematous due to immature *Paramphistomum microbothrium.*

Ecchymoses in the wall of the small intestine visible from the external surface caused by *Gaigeria pachyscelis* in sheep or *Bunostomum phlebotomum* in cattle may be noted. Yellow lymphatics and yellowish green mesenteric lymph nodes in sheep caused by L_3, L_4 and 5th stages of *Dictyocaulus filaria.*

Discard the decending colon and rectum in sheep and goats which may have nodules of *Oesophagostomum columbianum* but no adult worms. Then cut between the double ligatures separating the gastrointestinal tract into 4 specimens viz:

Tongue, pharnyx, oesophagus, rumen and reticulum. Omasum and abomasum. Small intestine. Caecum and colon (including the decending colon and rectum in cattle).

Place these organs in the tray. Examine the important organs, abomasum, small intestine and colon first. Before opening these organs label separate wide-mouthed jars (1 ℓ) and flat bottomed containers (45 ml) as follows:

abo = abomasum; si = small intestine and cc = caecum and colon.

Separate the omasum from the abomasum and place the former with the forestomachs for later examination. Open the abomasum along the greater curvature pouring the ingesta into a bucket and place any visible worms in the 45 ml container (labelled abo.) Wash the mucosa thoroughly into a bucket containing the rest of the ingesta and place the abomasal wall aside (for subsequent examination). Subsequently, empty and place the entire ingesta of the small intestine into another bucket, remove any tapeworms and wash them clean and place in a jar in formalin. Thereafter wash the mucosa thoroughly by threading the gut through the fingers of one hand and pulling it with the other. Repeat this process with the caecum and colon. Add 2-3 ml of concentrated iodine solution to the ingesta and mix thoroughly to kill the worms and then add 10-20 ml formalin to 'fix' them. Pour all the ingesta of the abomasum into the special sieving bucket (see equipment and Fig. 3) and wash the ingesta through the sieve with a strong spray provided by attaching the plastic pipe and shower rose to a tap. A few drops of detergent (Teepol*) facilitates the sieving process which must continue until all the foam has disappeared and the water running through the ingesta and sieve is clean.

Transfer the residue on the sieve's surface to a labelled 1 ℓ jar and add 5 ml of formalin. Repeat the whole process with the ingesta of the small intestine. Wash all the previously sieved abomasal ingesta into the graduated bucket and fill with water to the 5 ℓ mark. Stir vigorously with the ladle and transfer small quantities with the 25 ml pipette to the wide-mouthed 1 ℓ jar until a 500 ml aliquot has been collected. Transfer this aliquot to a jar labelled 1/10 abo. Repeat the process and collect another 1/10 aliquot. If many thousands of worms are present collect 2 x 50 ml aliquots in a similar fashion. Discard the balance of the specimen i.e. 78/100. Repeat the process for the ingesta of the small intestine. Sieve the ingesta of the caecum and colon, transfer the sievings to a wide-mouthed jar and formalize. Do not collect any aliquots but leave for subsequent examination.

Prepare 1 ℓ of a mixture of 30 ml concentrated HCl plus water at 50°C and pour 500 ml into the thermos flask. Place the abomasal wall in this solution and if necessary fill the flask with more warm acidified water before sealing the flask. Leave for 4 hours by which time the mucosa of the wall has been digested (J Schröder cited by P C Van Schalkwyk, Smith Kline, Isando, Personal communication 1984). Remove the undigested serosa, wash into a bucket, pour the digested mucosa and sediment into the thermos into the same bucket, add formalin and sieve using a fine meshed (38 μm) sieve. Collect the sievings in a labelled jar and preserve with formalin. Only if large numbers of worms are present, need 1/10 aliquot be made as described for the abomasal ingesta. Use hot water when sieving digested gut (see below).

Trematodes (Brief descriptions of the morphology are given in Table 3)

Liver: The presence of *Fasciola* spp. is usually shown by thickening of the bile ducts which is particularly noticeable in cattle. Take a butcher's knife and cut narrow slices 8-10mm across the liver tissue and express the parasites in the bile ducts with pressure on the cut edges. Place these worms in special jars (45 ml). The two species can be differentiated as follows:

F. hepatica – narrow shoulders, sloping to the sides, posterior end pointed, branches of the caeca sloping (Table 3).

F. gigantica – shoulders tend to be square, sides straight, posterior end rounded, branches of caeca more rectangular than those of *F. hepatica* (Table 3).

Always check the bone marrow where *Fasciola* spp. is present to determine whether erythoropoiesis has taken place. A colour illustration of the bone marrow and of the two *Faciola spp.* are given in Reinecke[5].

Paramphistomum spp. – the pathogenic stages are the immature worms in the small intestine or abomasum[5]. These minute worms 2-3 mm long attach themselves to the mucosa of the duodenum and the first 3 m of the small intestine by the posterior sucker and are pink in colour, resembling strawberry jam. Only if present in massive numbers 40 000 or more in sheep and goats and 80 000 to 100 000 in cattle are they pathogenic causing a projectile diarrhoea and death 10-14 days after infestation.

Schistosoma mattheei

At necropsy grey lungs in sheep, black livers in sheep and cattle, a hobnailed liver in goats and a red haemorrhagic ileocaecal valve projecting into the caecum of cattle, are pathognomonic[5]. The male is white and stubby, and has a gynaecophoric canal down the length of its body and envelopes the slender brown female in this canal. Both are found in the mesenteric blood vessels and easily seen in the mesentry before the fat coagulates[5] (Table 3)

Recovery of helminths

Microscopical examination

With the exception of the caecal and colonic ingesta which is examined macroscopically, all specimens are examined microscopically with the aid of a stereoscopic microscope with a light source in the base.*

The 500 ml of the abomasal ingesta is poured into an Ehrlenmeyer flask and a few drops of 45 % iodine solution (see above) added to stain it. The specimen is thoroughly mixed by swirling the suspension in the flask and pouring 2-3 ml onto the surface of the counting chamber. *Never pour more than a very thin layer of ingesta onto the petri dish.* The reddish brown worms will easily be seen if they are not covered with ingesta. Count the worms and with the needle on the 1 ml syringe transfer the worms to a specimen bottle (50 ml) containing formalin until 120 or all the worms present have been transferred. Continue counting the worms still present in the first aliquot if there are more than 120. If there are very few worms present (less than 100) count the 2nd aliquot. Repeat this process with the digested abomasal wall and ingesta of the small intestine.

Macroscopical examination

Few worms are present in the ingesta of the caecum and colon but they are white in colour and easily seen macroscopically against a black background. Pour all

*Shell Chemicals, P.O. Box 494, 2000 Johannesburg

*Footnote: Vickers Instuments Kyowa MG6, P.O. Box 6378, 1508 Dunswart: O'Connell Instrument Co., WMF4, P.O. Box 32029, 2017, Braamfontein.

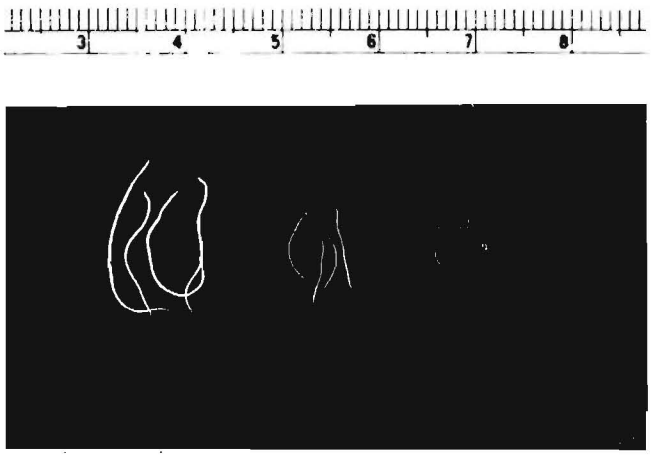

Fig. 4 Abomasal worms of cattle; left to right *Haemonchus placei, Ostertagia ostertagi, Trichostrongylus axei.* Scale in mm. (Helena Smit, University of Pretoria)

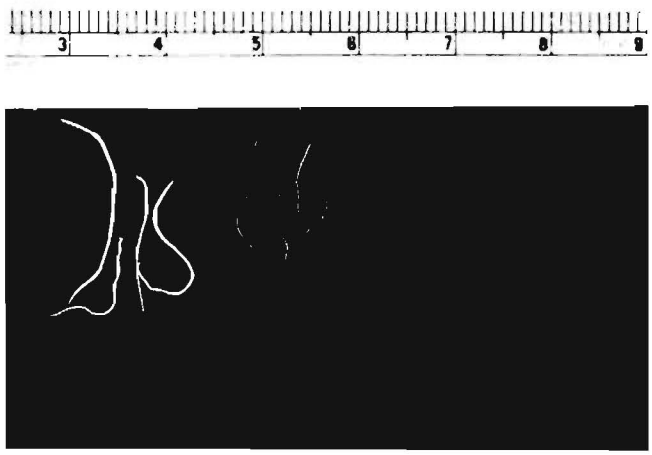

Fig. 5 Abomasal worms of sheep: left to right *Haemonchus contortus, Ostertagia circumcincta, Trichostrongylus axei.* Scale in mm. (Helena Smit, University of Pretoria)

the sieved ingesta into a bucket and fill with water to the 10 ℓ mark. Mark the upper surface of the black pan or the glass pie dish with white parallel lines (40-50 mm apart). Place the pan or dish on a perfectly level surface. The pie dish should be placed on black paper or the dull blue or green surface of carbon paper. Stir the ingesta thoroughly and pour the ingestal suspension onto the pan to form a thin layer of not more than 2-3 mm. If ingesta tends to accumulate add more water and tip the tray back and forth to form an even layer. Face the source of light and never cast a shadow over the tray either with the head or body. Examine the tray systematically between the parallel lines, count and pick up the worms and place in a small container.

Pepsin HCl digestion

This is only done in diagnostic laboratories where larval stages are diagnosed. Place the gut on a tray. With a butchers knife (cattle) and a glass microscopic slide (sheep and goats) apply firm pressure to the mucosal surface of the gut wall scraping the mucosa and underlying muscle layers of the serosa. Discard the serosa. Prepare a solution of 5 ℓ or 10 ℓ for sheep and cattle respectively of the digesting fluid. Add 50 g pepsin to 4 ℓ warm water and dissolve before adding 150 ml concentrated (commercial grade) HCl and fill the container to the 5 ℓ mark. Place the scraped mucosa and muscle layers of the abomasum in one 1 ℓ and divide the intestine into two 1 ℓ (sheep and goats) – or 3-5 1 ℓ (cattle) glass jars and fill each jar to within 2 cm of the upper rim with pepsin / HCl solution, place the cap on the jar and incubate the jars in a waterbath at 40 °C for 2-3 h.

Fig. 6 Small intestinal worms of cattle; left to right *Bunostomum phlebotomum, Cooperia* spp., *Paramphistomum* spp. (immature). Scale in mm. (Helena Smit, University of Pretoria)

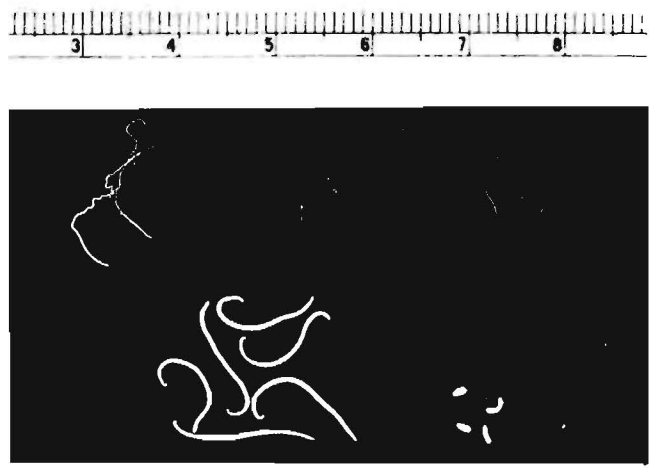

Fig. 7 Small intestinal worms of sheep: left to right above *Nematodirus spathiger, Strongyloides papillosus* and *Trichostrongylus colubriformis*; below left *Gaigeria pachyscelis* and right *Paramphistomum* spp. (immature). Scale in mm. (Helena Smit, University of Pretoria)

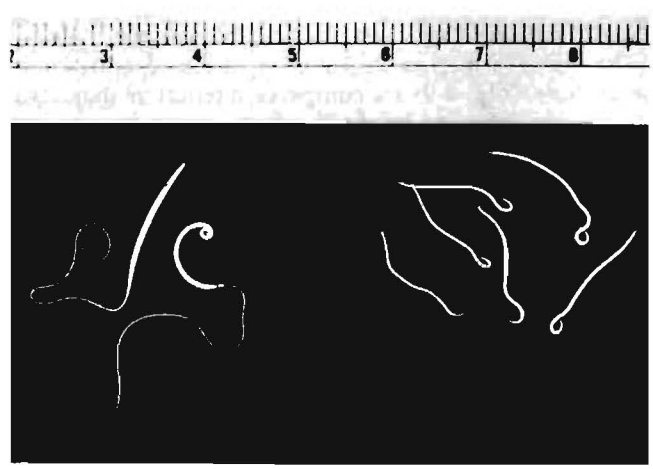

Fig. 8 Ceacal and colonic worms of cattle: left to right *Trichuris* spp. and *Oesophagostomum radiatum.* Scale in mm. (Helena Smit, University of Pretoria)

Fig. 9 Caecal and colonic worms of sheep: left to right *Trichuris* spp., *Chabertia ovina* and *Oesophagostomum columbianum*. Scale in mm. (Helena Smit, University of Pretoria)

Shake the jars vigorously every 15 minutes to mix the gut and digesting fluid. When the gut is digested, add 10 ml formalin to fix the worms, and sieve through a fine sieve (38 μm apertures) with hot water which dissolves the fat which is often present. Pour the residue on the sieve surface into a jar and add formalin as preservative.

Forestomachs

Open the omasum along the dorsal lateral aspect and examine the mucosa for immature *Paramphistomum* spp. 2-3 mm long and pink in colour. Open the rumen and reticulum with a butchers knife and examine the mucosa for adult *Paraphistomum* spp. 5-13 mm in length and pink in colour.

Occasionally there may be *Gongylonema* present in the submucosa. If putrefication has set in and the mucosa strips from the submucosa *Gongylonema* spp. may be released and found lying as a long white worm on top of the ingesta. Open the oesophagus with a scissors and examine the mucosa for *Gongylonema* spp. curled in a zig zag fashion submucosally.

Preliminary worm identification

The identification of larvae (L_3 and L_4) is the work of an expert and not within the scope of this paper. Adult worms previously collected are poured from the specimen bottles into a petri dish and a tentative diagnosis on a generic basis is made under low magnification using a stereoscopic microscope.

In the information in Tables 1, 2 and 3 and the illustrations (Fig. 4-9) are compared a tentative diagnosis on length alone can be made. Males are always shorter and more slender than females and the male's bursa gives the posterior end of the worm a racket-shaped appearance. In Fig. 4 the 3 abomasal parasites of cattle are illustrated – the longest on the left and shortest on the right. On the left is *Haemonchus placei* in which the male is 15 mm or longer and the female more than 20 mm in length; *Ostertagia ostertagi* in the middle in which the male is only 8 mm and female 8-12 mm long; the finest and shortest worm on the right is *Trichostrongylus axei* and males are 4 mm and females no more than 8 mm in length. In sheep *Haemonchus contortus, Ostertagia circumcincta* and *Taxei* have a similar length (Fig. 5). The common parasites of cattle in the small intestine illustrated in Fig. 6 are left to right, the large white *Bunostomum phlebotomum*, the small curled up *Cooperia* spp. (red when alive) and the pink immature *Paramphistomum* spp. with a sucker on one end. The five worms in the photograph (Fig. 7) occur in the small intestine of sheep. Above from left to right is the long thin *Nematodirus* spp. (long necked bankrupt worm), the very small *Trichostrongylus* spp. the most

Table 1: HOST PARASITE LIST OF HELMINTHS IN CATTLE IN THE REPUBLIC OF SOUTH AFRICA

Oesophagus
Gongylonema spp. – kartelwurm; zigzag worm

Rumen reticulum and omasum:
Gongylonema spp.
Paramphistomum microbothrium – peervormige slakwurm; conical fluke

Abomasum:
Haemonchus placei – haarwurm; barbers pole worm; wireworm
Ostertagia ostertagi – bruin maagwurm; brown stomach worm
Trichostrongylus axei – maag bankrotwurm; stomach bankrupt worm

Small intestine:
Bunostomum phlebotomum – haakwurm; hook worm
Cooperia spp. – bees bankrotwurm; cattle bankrupt worm
Moniezia spp. – lintwurm; tape worm
Nematodirus helvetianus – langnekbankrotwurm; long necked bankrupt worm
Paramphistomum spp. – (immature)
Strongyloides papillosus – wit bankrotwurm; white bankrupt worm
Thysaniezia giardi – lintwurm; tape worm
Toxocara vitulorum – spoelwurm; round worm
Trichostrongylus spp. – bankrotworm; bankrupt worm

Caecum:
Trichuris spp. – sambokwurm; whip worm

Colon:
Oesophagostomum radiatum – knoppieswurm; nodular worm

Abdominal cavity:
Setaria labiato-papillosa

Brisket:
Onchocerca gibsoni

Eyes:
Thelazia rhodesii – eye worm, oogwurm

Ligaments:
Onchocerca linealis

Liver:
Fasciola gigantica – reus lewerslakwurm; giant liver fluke
Fasciola hepatica – gewone lewerslakwurm; normal liver fluke
Larvae of *Echinococcus* spp. – hidatied; hydatid
Stilesia hepatica – lewerlintwurm; liver tape worm

Lungs:
Dictyocaulus viviparus – longwurm; lung worm
Fasciola gigantica
Larvae of *Enchinococcus* spp.

Mesenteric veins:
Schistosoma mattheei – bilharzia

Muscles:
Cordophilus sagitta – hartwurm; heart worm
Larvae of *Taenia saginata* – masel; measle

Subcutaneous tissue:
Parafilaria bovicola

Table 2: HOST PARASITE LIST OF HELMINTHS IN SHEEP AND GOATS IN THE REPUBLIC OF SOUTH AFRICA

Oesophagus:
Gonglyonema spp. – kartelwurm; zig zag worm

Rumen reticulum and omasum
Gongylonema spp.
Paramphistomum microbothrium – peervormigeslakwurm; concial fluke

Abomasum:
Haemonchus contortus – haarwurm; barbers pole worm, wire worm
Ostertagia circumcincta – bruinmaagwurm; brown stomach worm
Trichostrongylus axei – maagbankrotwurm; stomach bankrupt worm

Small intestine:
Avitellina spp. – lintwurm; tape worm
Bunostomum trigonocephalum – grasveld haakwurm; grassveld hook worm
Cooperia curticei
Gaigeria pachyscelis – sandveldhaakwurm; sand veld hook worm
Moniezia spp. – lintwurm; tape worm
Nematodirus spathiger – langnekbankrotwurm; long necked bankrupt worm
Paramphistomum spp. – (immature)
Stilesia globipunctata
Strongyloides papillosus – wit bankrotwurm; white bankrupt worm
Thysaniezia giardi – lintwurm; tape worm
Trichostrongylus spp. – bankrotwurm; bankrupt worm

Caecum:
Trichuris spp. – sambokwurm; whip worm

Colon:
Chabertia ovina – grootbekwurm; large mouth worm
Oesophagostomum columbianum – knoppieswurm; nodular worm
Oesophagostumum venulosum
Skrjabinema spp.

Abdominal cavity:
Larvae of *Taenia hydatigena* – (Cysticercus tenuicollis)

Brain:
Larvae of *Taenia multiceps* – (Coenurus cerebralis) malkopsiekte, gid sturdy

Liver:
Fasciola gigantica – reuse lewerslakwurm; giant liver fluke
Fasciola hepatica – gewone lewerslakwurm; common liver fluke
Larvae of *Echinococcus* spp. – hidatied; hydatid
Larvae of *Taenia hydatigena* – (Cysticercus tenuicollis)
Larvae of *Taenia ovis* – (Cysticercus ovis)
Stilesia hepatica – lewerlintwurm; liver tape worm

Lungs:
Dictyocaulus filaria – longwurm; lung worm
Fasciola gigantica
Larvae of *Echinococcus* spp.
Muellerius capillaris

Mesenteric veins:
Schistosoma mattheei – bilharzia

Muscles:
Larvae of *Taenia ovis* – masel; measle

common of all worms in the small intestine. Below on the left (Fig. 7) is *Gaigeria pachyscelis* a large worm and the minute (seedlike pink coloured when fresh) immature *Paramphistomum* spp. In the caecum and colon the worms of the cattle, *Trichuris* spp. (left) and *Oesophagostomum radiatum* right are shown in Fig. 8, and similar worms in sheep with the additional large mouthed worm *Chabertia ovina* in the middle are shown (Fig. 9).

Identification on a species basis

Almost all the namatodes of ruminants are identified to species level by examining the spicules and bursae of males with a compound microscope. The following will assist in arriving at a rapid accurate diagnosis.

Draw a line on paper and place a clean microscope slide across the line about 20 mm from the one end. Place a drop of lactophenol on the line and with the aid of the needle (on the end of a 1 ml syringe) transfer the males from the petrie dish onto the slide with the bursa on top of the line. Don't place more than 10 worms on one slide and never mix large worms with small worms. Place a cover slip (preferably circular) on top of the drop and examine microscopically with a low magnification at first (4x objective) only using the higher magnification when the spicule of structure to be identified is in the middle of the field. In Table 3 some morphological features are described. The details of the morphology are well described and illustrated in standard textboks[4-6].

Frequently the spicules, or any other structure on the worm or the bursa are not lying in the correct plane to be albe to make a diagnosis. In this case use the lowest magnification, and place a finger on the coverslip and roll the worm until the structure you are looking for is in the correct position. Thereafter change to a higher magnification and identify.

The importance of worm counts

Anamnesis

It is absolutely essential to have a complete record of the history of the herd of flock before carrying out a necropsy to confirm a diagnosis of helminthosis. Suckling lambs may die with low worm burdens or none at all because the ewe is suffering from agalactia due to verminosis.

Relatively low worm burdens combined with poor nutrition can cause mortalities and stress factors such as lambing, weaning, sudden changes in temperature, rain and drought must all be noted before arriving at a diagnosis.

Unless animals have recently been treated and the compound is ineffective against a certain parasite e.g. *H. contortus* or *O. circumcincta* no animal has a single species. Moreover the total worm burdens of one species may be insufficient to cause the death but combined with sublethal infestations of other species sufficient to cause clinical signs and death.

Counting worms at necropsy

The real reason a stereoscopic microscope is essential is to ensure that the worker does not miss the minute parasites viz. *S. papillosus,* immature *Paramphistomum* spp., *Trichostrongylus* spp., *Cooperia* spp., and *Ostertagia* spp. or the immature larval stages in the digested

Table 3: SOME MORPHOLOGICAL AND OTHER FACTORS OF ASSISTANCE IN MAKING A TENTATIVE DIAGNOSIS OF THE HELMINTHS OF DOMESTIC RUMINANTS

Avitellina spp.:	small intestine, thin tapeworm line down the centre of gravid proglottids due to paruterine organs large scolex, weaned and older sheep
Bunostomum phlebotomum:	duodenum thick white nematode with purple stripe down the length, ingesta blood stained – cattle (Fig. 6)
Bunostomum trigonocephalum:	small intestine thick white worm – common in kraaled sheep in Lesotho – elsewhere in old ewes on pasture, sheep and goats
Chabertia ovina:	attached to colonic mucosa; large mouth gives the anterior end a chopped-off appearance, sheep and goats (Fig. 9)
Cooperia spp:	small intestine, red curled up when fresh; inflated anterior end with cross striations (tranverse grooves) spicule short and stout, dorsal ray lyre shaped (Fig. 6), cattle
Dictyocaulus filaria:	bronchi of sheep and goats long thin, male boot shaped spicules
Dictyocaulus viviparus:	bronchi of cattle, similar to *D. filaria* for differences[5]
Fasciola gigantica:	bile ducts often thickened, long shoulders straight sides posterior end round, secondary caecal branches right angled, sheep, goats and cattle
Fasciola hepatica:	bile ducts often thickened, leaf like sloping shoulders posterior end pointed, secondary caecal branches angled, sheep, goats and cattle
Gaigeria pachyscelis:	small intestine, ecchymoses visible through gut wall, ingesta blood stained (tomato sauce), white with purple stripe down centre, male dorsal ray of bursa very prominent (parrot beak) (Fig. 7), sheep and goats
Gongylonema spp.:	oesophagus, rumen, recticulum and omasum, red zig zag under mucosa, morphology[4–6], sheep and goats
Haemonchus contortus:	abomasum, female red and white (barber's pole) tongue shaped vulval flap, male Y-shaped dorsal ray, morphology[4–6] (Fig. 5) sheep and goats
Haemonchus placei:	abomasum, female button shaped vulvar flap, morphology[4–6] (Fig. 4), cattle
Moniezia benedeni:	small intestine, broad tapeworm
Moniezia expansa:	small intestine, broad tapeworm
Both *Moniezia* spp.:	proglottid wider than deep double genitalia in each mature proglottid protrudes laterally forming a pimple like swelling on each side, gravid segment pressed between slides will release eggs with the following shapes:- *M. benedini* – diamond shaped *M. expansa* – semi-triangular lambs, kids and calves
Nematodirus filicollis:	small intestine winter rainfall and non-seasonal rainfall areas, rare elsewhere, morphology[4–6] (see *N. spathiger* below details)
Nematodirus helventianus:	small, intestine rare, detailed morphology[4–6], cattle
Nematodirus spathiger:	small intestine, long necked, male long thin spicules, posterior tips spoon shaped, female eggs large, rugby ball shaped (Fig. 7), Karoo, sheep and goats
Oesophagostomum columbiamum:	colon particularly *ansa spiralis*, intestinal tract full of nodules, anterior end hook shaped (Fig. 9), sheep and goats
Oesophagostomum radiatum:	entire colon, nodules rare in intestinal tract, anterior end hookshaped (Fig. 8), cattle
Oesophagostomum venulosum:	colon, not hook shaped, no nodules in intestinal tract, winter and non-seasonal rainfall areas, sheep and goats
Ostertagia circumcincta:	abomasum, brown, long thin embedded in gastric pits particularly pyloric region (Fig. 5), sheep and goats
Ostertagia ostertagi:	abomasum, brown thin imbedded in gastric pits particularly fundus region (Fig. 4), cattle
Ostertagia trifurcata:	abomasum similar macroscopically to other *Ostertagia* spp., morphology[4–6] sheep and goats
Parafilaria bovicola:	subcutis, rarely seen, long white worm (2-5cm), false bruising brown or yellowish-green lesion, impression smear full of eosinophils, cattle
Paramphistomum microbothrium:	adults rumen and reticulum, 5-13 mm long, often stunted in sheep, pink large posterior sucker immatures in duodenum and 1st 3m. of small intestine, 2-5mm, pink, cattle, goats and sheep
Schistosoma mattheei:	mesenteric blood vessels, male white, stubby enfolds slender brown female in gynaecophoric canal, colour illustrations[5], cattle, sheep and goats
Skrjabinema ovis:	colon 4-5 mm long minute morphology[4,5], sheep
Stilesia globipunctata:	tapeworm small intestine, macroscopically similar to *S. hepatica*, sheep
Stilesia hepatica:	bile ducts – rarely thickened, long thin, proglottids overlaps succeding proglottid like eves of roof; paruterine organs form 2 distinct lateral lines down gravid proglottids, sheep, goats, cattle
Strongyloides papillosus:	small intestine, *females only*, very small 3-6 mm, long oesophagus, blunt pointed egg thin shells with larvae (Fig. 7), kids, lambs and calves
Thelazia rhodesii:	conjunctiva, white curled up marked transverse striations (grooves)[4], cattle
Thysanezia giardi:	small intestine broad tapeworm, mature proglottids single genitalia projection one side (like a pimple) of proglottid on irregular alternating basis, paruterine organs onion shaped containing numerous eggs, sheep, cattle and goats
Trichostrongylus axei:	abomasum, minute white worm (4-6mm) (Fig. 4 and 5) sheep, goats and cattle
Trichostrongylus spp.:	small intestine very small (up to 10mm long) excretory pore in ventral notch near anterior end, female thinner anterior end widens out posteriorly to accommodate eggs (Fig. 7), males stout, spicules detailed morphology[4,5,6] sheep, goats less often cattle
Trichuris spp.:	caecum, whip worm long thin anterior end shorter thicker posterior end curled up (Fig. 8 and 9), sheep, goats and cattle

Table 4: WORM BURDENS THAT CAN CAUSE CLINICAL SIGNS AND EVEN DEATH. (DATA FROM FIELD TRAILS DONE BY THE AUTHOR AND PERSONAL COMMUNICATIONS BY I.G. HORAK, J. SCHRÖDER AND J.H. VILJOEN)

SPECIES	NUMBER OF WORMS
Cattle	
Bunostomum phlebotomum	100
Cooperia spp.	40 000 – 50 000
Fasciola gigantica	200 or more
Fasciola hepatica	700 – 1 000
Haemonchus placei	8 000 – 10 000
Oesophagostomum radiatum	800 – 1 000
Ostertagia ostertagi	20 000 – 30 000
Paramphistomum spp. (immature)	100 000 – 150 000
Sheep and Goats	
Bunostomum trigonocephalum	300 – 400
Fasciola gigantica	60 – 100
Fasciola hepatica	150 – 200
Gaigeria pachyscelis	100 or more
Haemonchus contortus	1 000 – 3 000
Nematodirus spathiger	1 000 – 10 000 (lambs) (adults)
Oesophagostomum columbianum	200 – 500
Ostertagia circumcincta	10 000
Paramphistomum spp. (immature)	40 000 or more
Strongyloides papillosus	11 000 lethal for kids
Trichostrongylus axei	25 000 – 40 000
Trichostrongylus colubriformis and other intestinal *Trichostrongylus* spp.	20 000 – 25 000

abomasal or intestinal gut. Obviously, if there are 10 or 20 parasites of these species present in the first petrie dishful counted, may hundreds or even thousands will be present in the 1/10 aliquot of 500 ml. Logically therefore, no useful object is served by merely counting worms. The smaller 1/100 aliquot of 50 ml will give as accurate an estimate of the total burden of 2 000 worms or more, than the larger 1/10 aliquot.

Significant worm burdens

The data summarised in Table 4 show that the most pathogenic worm in cattle is *B. phlebotomum* followed by *F. gigantica, F. hepatica* and *Oesophagostomum radiatum*. Massive numbers are necessary to cause clinical signs with the other parasites ranging from 8 000 *H. placei* to 100 000 *Taxei* or immature *Paramphistomum* spp.

Data for sheep show that *F. gigantica* (60) and *G. pachyscelis* are the most pathogenic worms followed by *Oesophagostomum columbianum, F. hepatica* and *B. trigonocephalum* (300-400). The other parasites must exceed 1 000 (*N. spathiger* in lambs) with up to 40 000 immature *Paramphistomum* spp. to cause mortalities (Table 4).

It is essential however, that a detailed necropsy be carried out and that the lesions themselves be taken into account. A few examples will illustrate this point.

It is known that young flukes migrating through the liver, literally chew a path through the parenchyma before arriving in the bile ducts. A few *F. gigantica* leave haemorrhagic paths in their wake which accumulate and may cause vast haemorrhages in the liver and death due to internal haemorrhage[5]. These and other blood sucking parasites cause anaemia and a more subtle approach to arrive at a diagnosis has to be followed.

As mentioned above a long bone is sawn down its length to expose the bone marrow. This will give an indication of the haemopoietic activity and Allonby[1] has used this as one of the criteria to classify 3 syndromes caused by *H. contortus* in sheep:

(i) Hyperacute: expansion of red marrow at the epiphysis with 10 000 or more worms.
(ii) Acute: entire medullary cavity is filled with red marrow into the epiphysis: burdens of 1 000-10 000.
(iii) Chronic: there is evidence of chronic expansion of red marrow combined with reversion to white marrow indicating a marked iron deficiency with a residual burden of 100 – 1 000 worms only.

This is a clear indication of the danger of relying on mere numbers to make a diagnosis. Any parasite that causes anaemia such as *F. hepatica, F. gigantica* or the hookworms *G. pachyscelis* or *B. phlebotomum* will also stimulate compensatory erythropoiesis but iron deficiency may occur with reversion to white marrow.

Some parasites may cause most of the pathogenic effects during the early stages of their life cycle. In sheep the L_4 of *O. columbianum* causes ulcerations with adhesions, nodule formation, diphtheritic typhlitis and colitis and many larvae and adult worms are expelled before death. Similarly 5th stage and young adult *O. radiatum* cause haemorrhagic colitis with projectile diarrhoea expelling most of the worm burden leaving only a few residual worms at necropsy. In these cases no faith can be placed in actual numbers of *Oesophagostcmum* spp. as the cause of death.

It must never be forgotton that other pathogens combined with worms can be fatal. In Table 4, 40 000 *Cooperia* spp. are stated as being the number which can cause clinical signs. If this, however, is combined with coccidiosis, calves can develope haemorrhagic enteritis and die with very moderate numbers of *Cooperia* spp. Scrapings of the gut wall for *Eimeria* spp. should always be made to confirm or reject either or both parasites as a cause of death.

Finally it is hoped that this article has emphasized the importance of a thorough necropsy examination before making a diagnosis. We all use compound microscopes every day and I hope you will include a stereoscopic microscope as standard equipment in your clinic, hospital or laboratory to make a simple, easy and accurate diagnosis of worms.

ACKNOWLEDGEMENTS

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TO THE EDITOR — AAN DIE REDAKSIE

SUPERFICIAL CORNEAL OEDEMA

I read with interest Dr S.W. Petrick's Article in the Journal Vol. 54 No. 4 on Superficial Corneal Oedema.

Some points in his summary I disagree with.

He states: "It is known that a hypertonic salt solution will dehydrate the cornea". To my knowledge this holds true for a hydrated cornea and not for a normal, intact, cornea.

1. Normal corneal tissue is dehydrated by comparison to other soft tissue (75 – 80 % water). This state of dehydration is essential in maintaining the parallelism of collagen fibres in the corneal matrix.
2. Should corneal oedema develop in the post operative period after canine cataract surgery, topical osmotic agents are used to clear the cornea temporarily. This allows observation of pupil position, size, etc., and enables identification of problems that may be reversible medically if detected early. Amongst the frequently used agents is 2 – 8 % sodium chloride solution.

   I conducted a small experiment with two rabbits with normal corneas. For 5 consecutive days, drops of a saturated sodium chloride solution were instilled into the eyes, three times a day.

   Apart from an immediate burning sensation, which caused vigorous rubbing and washing, no adverse effects were noted. At the end of the experiment, all four rabbit corneas were 100 % normal.

   The author further stated: "It seems that in this patient a very concentrated salt solution caused rapid dehydration and accumulation of fluid in the superficial layers of the corneas".

   I fail to follow his reasoning altogether.

   Allow me to suggest the following:
3. The epithelium in the eyes operated on, were most unlikely to be damaged by a surgeon as experienced as Dr. Petrick.
4. The endothelium is far more important in maintaining corneal hydration. In rabbits removal of the epithelium produces a 200 % increase in corneal thickness in 24 h, compared to a 500 % increase when the endothelium is damaged.
5. The servere nature of the corneal oedema observed in the case discussed, plus the fact that the aqueous integrity had been disturbed during surgery, would point to endothelial pump failure in this case.
6. Dr. Petrick does not mention if fluorecein was used before performing keratectomies.
7. Performing a keratectomy on such massively oedematous corneal tissue is, in my opinion, contra-indicated, as it deprives the tissue of the protection afforded by the tearfilm and epithelium. This then exposes the corneal matrix to bacteria and collagenase.

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TO THE EDITOR — AAN DIE REDAKSIE

DR M. THOMSON'S LETTER ON "SUPERFICIAL CORNEAL OEDEMA"

A hypertonic salt solution will dehydrate a hydrated cornea (oedematous) as well as a normal intact cornea in a state of deturgescence (slightly dehydrated) by the simple process of osmosis.

The "small experiment with two rabbits with normal corneas" proved nothing because the patient's corneas were not normal due to the lentectomy operations, yet in both circumstances osmosis did take place.

Although after two days there was no sign of endothelial oedema in the patient it is quite possible that there was a degree of endothelial damage due to the intraocular surgery.

Thus the fluid imbided by the stroma and the rapid dehydration was faster than it could escape from the epithelium and the accumulation in the superficial layers of the cornea took place.

The use of fluorescein was not indicated because it would only have stained the oedematous fluid and after disrupting the epithelium.

Removing loose, traumatised or diseased superficial cornea is not harmful to the rest of the cornea as the outcome of the karatectomy in this patient proved.

I believe my statements are still valid but that for the sake of my colleague I should have mentioned the possibility of endothelial damage.

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THE S.A. VETERINARY ASSOCIATION'S PREMIER AWARD: THE FIRST GOLD MEDAL

WILHELM OTTO DANIEL MARTIN NEITZ

Recently the Association awarded its 13th Gold Medal for exceptional scientific achievement and significant contribution to the advancement of science. As befits the event, the name, photograph and *curriculum vitae* of the recipients have all been prominently placed in the Association's Journal – with one notable exception: the first award!

Reasons for this omission are unclear at this stage but they certainly have no bearing on the importance and dignity of the occasion or the recipient. With the recent unearthing of a photograph taken on the auspicious occasion it now becomes possible to correct the matter.

At the Association's Biennial Scientific Congress and 66th Annual General Meeting of its members held in September in 1971 in East London, the first Gold Medal was awarded to Prof Wilhelm Otto Daniel Martin Neitz, then head of the Department of Infectious Diseases of the University of Pretoria's Faculty of Veterinary Science at Onderstepoort, where he had completed a brilliant research career at the Veterinary Research Institute. Prof Neitz died in August 1979 and his *curriculum vitae* appears in the Journal of the SAVA Vol 51 No 2 p 125.

It was also co-incidental that the members of the Association decided at the AGM to dispense with the word "medical" in the name which the Association had held since its inception i.e. the "South African Veterinary Medical Association."

Prof. Neitz receiving the citation from the then President of the Association, Dr L.W. van der Heever.

REVIEW OORSIG

FLEECE-ROT : THE EPIDEMIOLOGY AND SIGNIFICANCE OF THE DISEASE IN SHEEP

R.H. SALISBURY and P.R. BARROWMAN*

ABSTRACT: Salisbury R.H.; Barrowman P.R. **Fleece-rot : The epidemiology and significance of the disease in sheep.** *Journal of the South African Veterinary Association* (1984) **55** No. 3, 147-151 (En). Department of Animal Science, Faculty of Agriculture, University of Natal, P.O. Box 375, 3200 Pietermaritzburg, Republic of South Africa.

Fleece-rot is a disease of sheep manifested by a superficial dermatitis with seropurulent exudation and matting and, in some cases, pigmentation of wool which occurs following periods of excessive wetness. In Australia it is recognised as the most important predisposing factor to body blowfly strike, and as such represents a major loss to the Australian wool growing industry. This review of the literature suggests that there is no reason for the condition not to be intermittently a significant problem under South African conditions. A long term solution to the problem is achieved by the breeding of resistant sheep. Measurement of fleece wettability is possibly the best method of distinguishing resistant from susceptible sheep.

Key words: Fleece-rot, blowfly strike, fleece properties, fleece wettability, resistance.

INTRODUCTION

Fleece-rot as a pathological condition has received scant attention in South Africa whereas research in Australia indicates that fleece-rot is an important source of economic loss. Together with the associated body blowfly strike it represents one of the major losses of production to the Australian sheep industry[1 3 18 19]; body and breech strike together were estimated for 1977/78 to have cost that industry 55 million dollars (Brideoake 1979, cited by Hart et al.[10]).

Abnormalties of the fleece of sheep following periods of wetness were described as early as 1523[2]. Initially attention was largely focussed on the presence of areas of discolouration (shades of green, brown, orange, pink or blue) that developed in a band across the staple of chronically wet fleeces. Stuart (1894) followed by Seddon & McGarth (1929) cited by Hayman[13] suggested that these discolourations were caused by bacterial activity. In South Africa green bacterial staining of Merino wool was reported by van Tonder et al.[25] to have been frequently encountered in the Eastern Cape and Karoo regions following excessive rains experienced there during January 1974. As many as 50 % of the fleeces in some flocks were affected. The role played by the chromogenic bacterium *Pseudomonas aeruginosa* was demonstrated. The economic implication of stained wool is that the pigment involved (pyocyanin in green discolourations) is not scourable and so the end value of the fibre in processing is considerably reduced. There are no other deleterious effects associated with the physical properties of the fibre[25].

Closer studies of the changes occurring following fleece wetting conducted by Seddon (1931), cited by Hayman[13], revealed a less apparent abnormality known variously as fleece-rot, wool-rot, yolk-rot or water-rot. A definition of the condition incorporating the findings of several workers[5 13 22 26] would be : a superficial dermatitis resulting from bacterial proliferation induced by wetness at skin level and manifested by seropurulent exudation and matting of the wool fibres so that a band of matted fleece adjacent to the skin is formed. Fleece discolouration commonly accompanies the disease but bacterial colouration without skin exudation is not classified as fleece rot (Belschner 1937, cited by Hayman[13]). Despite the superficially similar appearance to other dermal conditions (e.g. lumpy wool and sheep scab) fleece-rot is readily diagnosed. The odours produced by bacterial decomposition in the lesion encourage oviposition by the primary strike blowflies, principally *Lucillia cuprina*[19], thereby giving rise to the most economically significant feature of the disease.

EPIDEMIOLOGY

Under natural and experimental conditions, continued wetting of sheep, such that saturation of the skin surface persists, will give rise to fleece-rot[13 18]. It is more likely to occur in animals with well grown fleeces. Under normal circumstances the skin will only become wet after sustained heavy rainfall[13]. Rural Research (1960)[2] cites the work of Hayman who found that approximately a week of continual skin wetting was sufficient to induce the disease and this situation would most likely arise in months having 8 or more wet days and a total of 100 mm or more of rain. Temperature in itself is not an important factor; fleece-rot lesions may be expected to develop at any time of the year[14 25]. The seasonal incidence of fleece-rot will therefore coincide with the months of maximum rainfall and so varies from place to place and year to year. The role of high rainfall of high intensity in initiating the disease is exemplified by the comparison of its incidence in Trangie, New South Wales, Australia, in a relatively dry year (14,5 % of sheep affected) to that in a consecutive wet year (50,5 %)[18]. Cole[6] records a fleece-rot incidence as high as 92 % in wet years. According to Hayman[13], an occurrence of 82 % is "severe" and a 37 % incidence of "medium severity".

Belschner (1937) cited by Hayman[13] was the first to recognize that certain sheep showed a predisposition to fleece-rot. This may be attributed to the variations in physical characteristics of the fleece and skin that exist amongst sheep of different breeds and strains, and even amongst individual sheep in the same flock[2 6 7 13 17 18 23 24]. Increasing staple length reduces

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penetration of rain to skin level, though once wet a fully grown fleece will dry out far more slowly. Conversely, fleeces of short staple length are rapidly penetrated but dry out more quickly[13]. Character (staple conformation) and fleece architecture, both readily appraised subjectively, are important in that in compact fleeces showing little tendency to open, water tends to penetrate by diffusion only[17] while in less dense fleeces that open easily direct wetting of the skin is permitted. Fraser[8] found that close compact fleeces rather flat at the tip had a lower moisture content at the skin surface than did open fleeces having staples with a pointed tip.

The products of the sebaceous glands are important in 2 respects relating to resistance. Firstly, the hydrophobic properties of wool wax may be expected to reduce water penetration of the fleece. Secondly, the wax layer of the skin itself provides an important protective barrier to moisture and other substances. James & Warren[15] postulated that "integrity of the sebaceous layer on the skin is important in maintaining resistance to fleece-rot". In an experiment they compared the incidence and severity of fleece-rot in sheep which had been treated with a petroleum solvent to disrupt the sebaceous layer with that of untreated control sheep with intact sebaceous layers. Significantly ($P<0,005$) more 'treated' sheep developed fleece-rot than did untreated control sheep and the severity of the lesions was far greater. No studies examining the relative thickness of the sebaceous layer have been conducted but the percentage wool wax (condition) may be expected to give an indication of the extent of this barrier.

Fraser[8] suggests that a yellowish fleece colour (high suint content) is indicative of susceptibility and Thornberry et al.[24] found a correlation of 0,56 between fleece-rot and the basal suint content of the fleece. White, bright wool (low suint) has been shown to be associated with resistance to fleece rot[18]. It may thus be concluded that a high wax : suint ratio is desirable. Increased fibre density is naturally associated with a higher sebaceous gland density. Increased fibre density is also related to reduce fibre diameter. This partially explains the higher degree of resistance of fine-wooled sheep as opposed to strong-wooled sheep[18] (Table 1). Similarly, the fleece characteristics of handle and secondary : primary follicle ratio (S:P ratio) can be correlated with fleece-rot resistance. Handle is largely a function of differences in fibre fineness and plasticity. Burley & Speakman, cited Lipson[17], found the plasticity of primary fibres to be much greater than that of secondary fibres. This means that a wool of softer handle is likely to have a greater S:P ratio. Where 2 samples have the same measured fibre diameter, the softer handling wool will generally represent the more resistant fleece[17]. That any one fleece charactaristic will dictate relative susceptibility or resistance to fleece-rot is evidently unlikely. It appears that more complex interrelationships exist.

Secondary factors also influence these physical characteristics of fleece and skin. With increasing age there is a decreasing susceptibility to fleece-rot[13]. However, repeatability of susceptibility at different ages is reasonably high[18]. The various fleece properties characteristic of a particular breed or strain will largely dictate the relative resistance or susceptibility of that breed or strain[17 18 22]. Severe nutritional stress may affect the production of fleece components other than wool[17]. Increased susceptibility may therefore be associated with a high stocking rate or poor grazing,

Table 1: INCIDENCE OF FLEECE-ROT AND BODY STRIKE AMONGST DIFFERENT STRAINS OF MERINO SHEEP OF THE TRANGIE (N.S.W. AUSTRALIA) BREEDING FLOCK (after McGuirk et al.[16])

Flock Strain	Average fibre diameter (μm)	% lambs with fleece-rot	% lambs with body-strike
Fine wool, ave, of 2 flocks	18,3	6	3
Medium wool, non Peppin strain, 2 flocks	20,8	40	17
Medium wool, Peppin strains, 10 flocks	20,5	39	19
Strong wool, 1 flock	21,4	67	33

ewes in late gestation and prior to weaning and following a period of drought. Conversely and abundance of rich pasturage may give a lower scoured yield but confers a higher degree of resistance to fleece rot.

PATHOGENESIS

Belschner (1937) cited by Hayman[13] observed that excessive moisture on the skin surface promoted multiplication of fleece bacteria and postulated that this initiated a superficial dermatitis and hence the fleece-rot lesion. However, Hayman[13] declared that constantly wet skin alone was sufficient to induce a mild dermatitis. He maintained that bacterial activity was a secondary feature, important only in producing the sometimes associated condition of fleece discolouration. Subsequently there was general support for this view[6 11 18 22]. To determine the precise significance of the fleece micro-organisms associated with fleece-rot, Merrit & Watts[20] conducted a study investigating the changes in microbial flora and protein content of the fleece and on the skin surface. The found that marked proliferation of bacteria, almost exclusively *Pseudomonas* spp., occured in the fleece and on the skin of all experimental sheep within 24 hours of wetting. For the first 5 days or so no appreciable changes in the mean concentration of suluble protein from skin washings were recorded for resistant sheep whereas those sheep that developed fleece-rot showed a marked increase of soluble protein on the skin within the same period.

The accumulation of "viscid, brownish material" on the skin surface found at fleece-rot sites was shown electrophoretically to contain plasma proteins that had leaked into the skin surface[26]. Nay & Watts[22] studied histopathological changes associated with the development of fleece-rot. On the basis of their findings it was suggested subsequently[19 20] that damaged or abnormal follicles provided a route for the leakage of plasma protein onto the skin and adjacent fleece. Hay et al.[12] found that exposure to rainfall induced changes in white cell numbers in the blood and concluded that some immune response to bacterial products had been triggered. Burrel et al.[5] went further and experimentally induced characteristic fleece-rot lesions on areas of sheep's skin to which wool pads saturated with *Ps. aeruginosa* culture had been attached. The dermatitis produces was of far greater severity than that induced by control pads saturated with sterile culture medium containing a

bacterostat. In addition, this study produced evidence that the loss of seropurulent fluid through the epidermis could be due to microabscesses forming in the stratum corneum as a result of the inflammatory response. Burrel et al.[5], citing Heckley (1970), describe various toxins or enzymes viz. exotoxin A, haemolysin, lecithinase, protease and elastase, produced by *Ps. aeruginose*, that are capable of causing dermal pathology. It was concluded that pathogenesis may conceivably be attributed to the proliferation of *Ps. aeruginosa* and the subsequent action of its metablites and that leakage of fluid through the epidermis occurred probably via both damaged follicles and ruptured micro-absceses.

CLINICAL FINDINGS

Hayman[13] examined 876 fleece-rot affected Merino sheep of all ages and sexes and found 83 % had lesions on the withers or withers and other regions, 14 % has lesions on the backline or backline and other regions excluding the withers, whilst less than 3 % had lesions on regions other than the wither or backline. Similarly Fraser[8] found the sites of most abundant skin exudate to be on the shoulder and mid back. Initially, in these areas where moisture is retained, the skin is seen to assume a deep purple hue[9]. This is followed by the exudation and accumulation of seropurulent material that causes the characteristic band of matted fleece. The wool in the affected area is always saturated and is characteristically "leached and dingy"[13]. These changes in the appearance and feel of the wool can be attributed to changes in the wool wax[9 11]. In severe cases the wool is easily plucked from the lesion[13].

The area of fleece-rot emanates putrid odours which attract gravid blowflies that then lay clusters of eggs close to the skin in the wet fleece[19]. The first instar larvae are only capable of feeding on the bacterial 'soup' found in the lesion. This rich source of protein enable development to the second instar stage and with more elaborate mouthparts the maggots are capable of abrading skin and extending the area of strike[20].

PATHOLOGY

Various histopathological changes are revealed on examination of skin sections from affected sites. Sections examined by Hayman[13] showed hyperkeratosis, with thickening of the stratum corneum granulosum and rete mucosum, shedding of cells from the corneum and some subcutaneous oedema. In addition, Thornberry et al.[24] decribe unpublished data of Kowal where increased capillary diameter and reduced skin surface wax thickness were observed. More recently, Burrell et al.[5] conducted a more comprehensive study of histopathological changes. Wetting (without fleece-rot) induced thickening of the epidermis with hyperkeratosis especially noticeable in the follicular openings, mild acanthosis and oedematous swelling of the basal layers with focal spongiosis of the stratum spinosum and stratum germinativum. There was mild hyperaemia, oedema and polymorphonuclear cell infiltration of the dermis. Associated with natural fleece-rot these changes were more pronounced and microabscesses were present in the stratum corneum. There was haemorrhagic extravasation in the dermis. Experimental fleece-rot also caused extensive parakeratosis and acanthosis, microabscess formation within the stratum corneum and discharge of frank pus into the surface of the skin. Occasional purulent invasion of the outer root, sheath of follicles and the follicles themselves, was observed but never to the same extent as observed in the epidermis.

In the studies of Nay & Watts[22] on wool follicle abnormalities in sheep exposed to prolonged wetting, weakened stretches of fibres resulting from impaired keratinization occurred frequently. This leads to fibre "tenderness". Hypertrophic thickening and duplication of the inner root sheath keratin was also found. This material tended to encase the proximal ends of broken fibres to form plugs. The plugs were observed to grow rapidly up the follicle and evidence of disrupted cellular membranes and free flowing serous exudate suggested that damage to the skin occured during their emergence form the collapsed follicular canals.

Goodrich & Lipson[9] and later Hay & Mills[11] found that ester-splitting in wax took place in conjuntion with the development of fleece-rot and proposed that the high concentration of free cholesterol and lanosterol resulted from bacterial action. The latter study revealed an increase in free sterols in wet wool but the expected simultaneous rise in free fatty acids did not occur. They postulated that free fatty acids may combine with potassium salts of suint to form a soap which may then be washed from the fleece, thereby simultaneously emulsifying remaining wax and further reducing its content. The implications of this would be decreased waterproofing of the fleece and a potential mechanism for disruption of the sebaceous layer of the skin, shown to be central to the development of fleece-rot[15]. Alternatively free fatty acids may be consumed by the multiplying bacteria.

DIFFERENTIAL DIAGNOSIS

Excessive rainfall also favours outbreaks of lumpy wool. This fungal infection caused by *Dermatophilus congolensis* is distinguished from the fleece-rot lesion by the development of scabs and skin ulceration leading to raw wounds[21]. The scab remains firmly attached to the skin and the wool in affected areas has a characteristic hard horny handle[4]. The lesion first appears as a small red spot on the skin that gradually enlarges to produce a watery exudate and a scab after 10 – 14 days[21]. The first signs of fleece-rot, however, are seen when the usually dry, supple and pinkish skin of a healthy sheep becomes wet, begins to take on a blue hue and is sensitive to touch. The affected areas deepen in colour, the skin may be easily torn when parting the fleece and a characteristic exudation from the purplish skin becomes pronounced. Bands of fleece-rot are then clearly visible in the fleece overlying the areas of macerated skin[13 22]. In addition, fleece-rot is restricted to the wooled body regions, whereas lumpy wool lesions are also found on the face, lips and ears[21].

REDUCING SUSCEPTIBILITY

Tail docking, mulesing and crutching effectively reduce susceptibility to breech myiasis but body-strike initiated by fleece-rot can only be influenced by insecticides, time of shearing and breeding[24]. The costs associated with insecticides, in addition to the greatly increased management requirement and continual need to overcome fly pesticide resistance, can be expected to reduce reliance

on this form of control in future. Shearing may be timed to precede expected seasonal rainfall[13], though this may not be applicable in all situations for obvious reasons. Breeding, therefore, offers perhaps the best long-term strategy for increasing resistance to fleece-rot and body-strike[18]. The first approach is a programme of direct selection for resistance, with all animals showing body-strike or perhaps even only fleece-rot being culled. However, as Copland & Pattie[7] observe, the effectiveness of discrmination between sheep is greatest when the incidence is near 50 %. This level of challenge does not always exist due to differences in seasonal conditions. This is illustrated by the situation described by McGuirk et al.[18] where in one year a flock showed an incidence of only 7,8 % of sheep infected, resulting in fleece-rot free rams being selected from the top 92 % of the population; a negligible selection differential in effect. A further disadvantage of direct selection is that studs may be located in areas of low rainfall but sell rams to high rainfall regions. To increase the effectiveness of selection, an artificial wetting regime could be used to identify susceptible animals. McGuirk et al.[18] report reasonable success with this technique, though the feasibility under practical conditions may be questionable.

A programme of indirect selection for resistance would appear to be a preferable alternative. The failure to discover a single fleece characteristic showing a high correlation with fleece-rot has been mentioned. However, Lipson[16] has adopted a more direct approach to the problem. He considers that since the ability of a sheep's fleece to absorb and retain water is central to the development of fleece-rot, fleece wettability as such may be a useful criterion for selection. A technique for measuring fleece wettability was developed[16]. It invloves placing a sample staple in a plastic cylinder with one end in water and measuring the mass of water taken up in a given time. The technique was shown to be consistent and capable of distinguishing resistant from susceptible sheep. Susceptible sheep are seen to have a high fleece wettability.

The result of Lipson's work prompted Pascoe[23] to conduct a study to evaluate the usefulness of fleece wettability as a means of indirect selection. Once again the technique was found to be repeatable and capable of distinguishing between sheep. Measurement is independent of seasonal conditions and the character is expressed on a continuous scale so that potentially intense selection pressure can be applied. The heritability of fleece wettability in 2 flocks selected for resistance was estimated as 0,26, though it is suggested that by minimizing the error variance associated with the technique, the heritability will be increased.

Following the same reasoning as Lipson[16], Copland & Pattie[7] propose that the rate of drying of sheep wet to skin level is inversely proportional to their susceptibility to fleece-rot. It was found using a relative humidity probe inserted into the fleece to skin level, that one hour after wetting, all sheep had fleece humidity measurements of 100 %. Thereafter it was found that "susceptible" sheep dried out at a significantly slower rate than "resistant" sheep. Ambient humidity and temperature were seen to affect drying out rates, so the technique at present appears capable only of distinguishing susceptible sheep form resistant sheep when measurements are taken from sheep treated as a single group. However, further work directed at standardizing the technique may result in a useful means of predicting susceptibility to fleece-rot and fly-strike.

DISCUSSION

The Australian experiences suggest that fleece-rot may represent a significant loss to the sheep industry in South Africa but a loss that to date has not been fully realised. It may be asked why this condition has hitherto received such minor consideration in South Africa. A likely answer is that more obvious diseases such as blue tongue, foot and mouth, jaagsiekte and others, present in South Africa but absent from Australia, receive prime consideration (Dr G. Bath, Allerton Laboratory, Pietermaritzburg, 1984, personal communication). In addition, the main sheep farming areas, namely the Karoo and Eastern Cape, are not renowned for high or seasonal rainfall. Despite this, body strike is seen to occur and may well be associated with primary fleece-rot.

Recognition of fleece-rot and the associated body strike problem is the first step. In the past, few prophylactic measures could be taken. Prevention of strike by regular dipping or treatment of struck sheep provided the only means of control. Now with a clearer understanding of the aetiology of the disease, long term preventative measures bay be taken. On the one hand prevention is achieved by developing a sheep with a fleece showing a minimal penetration by water and which, in addition, rapidly dries out once wet. The fleece wettability indirect selection criteria of Lipson[16] at the present time represent the most suitable means of breeding resistant sheep. It is a test easily applied to individual sheep and one well within the scope of a progressive stud breeder selecting rams. It is envisaged, however, that the main application of this technique would lie with the fleece testing centres.

If skin wetting cannot be prevented, a second short term means of fleece-rot prevention may exist. The resent evidence suggesting that proliferation of *Ps. aeruginosa* is responsible for pathogenesis, gives rise to the possibility that pathological changes may be prevented by modification of the host's immune response. Burrell et al.[5] citing unpublished data of Burrell, briefly mention that the subacute dermatitis reaction characterising the early stages of fleece-rot was prevented in sheep by immunization of sheep with *Ps aeruginosa* preparations. However, further investigations are necessary to evaluate the significance of the whole fleece-rot question in South Africa.

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CORRIGENDUM

Jl. S. Afr. Vet. Ass. 55 (1), 1984
The following corrections should be effected:

1. S van Amstel: "Primary renal cell carcinoma in a horse"
   p 35, 2nd column: 5 m should read 5 ml
   p 38, under "Conclusion" "cronic development" should read "chronic development"
   p 38, under "Discussion": "Tubular renel cell" should read "tubular renal cell".
2. S van Amstel: "Observations on the symptomatology and diagnosis of clinical cases of Johne's disease" p 46, Table 1. "BODY TEMPERATURE" under column "O" 39.0 should read 39,8.

INFORMATION — INLIGTING

MERGER CREATES NEW FORCE IN ANIMAL HEALTH RESEARCH

JOHN F. WEBB, LPS Science Correspondent

London (LPS): Two of Britain's major pharmaceutical companies are to merge their worldwide animal health interests. The link-up will create what is described as "a significant force" in this particular market.

The giant Imperial Chemical Industries (ICI) and the Wellcome Foundation have agreed in principle to form a group of companies, the largest of which will be a joint UK-based company controlling operations worldwide. In addition two other joint companies will operate in Australia and New Zealand.

The UK company, to be known as Coopers Animal Health, will together with the other two newcomers take over ICI/Wellcome in the UK, US, South America, Federal Germany, Australia and New Zealand. Wellcome will contribute major research assets in the UK and US, and ICI major research facilities in New Zealand.

In a statement issued in London on 2 March, the two groups said the joint organisation will have the financial strenght to fund a significant research and development programme. A spokesman commented: "The new organisation will undertake significant development of the international animal health businesses of both parents by combining their research, production and marketing interests in this area. This concentration of scientific, veterinary and marketing skills will ensure that the service which the two companies have independently provided to animal health throughout the world will be extended and improved."

Total Sales

Animal health sales account for about 10 % of ICI's total worldwide pharmaceutical sales with manufacturing facilities in the UK, Australia, New Zealand, India and Malaysia. Development work over the last two decades has resulted in such products as a round worm remedy and one of for controlling liver fluke.

Wellcome's strengths include the development of vaccines for both human and animal health protection. The company is now the world leader in control of organisms such as ticks which attack the hides of animals.

Among Wellcome's recent developments are a new leptospiral vaccine for dogs and the first effective treatment for East Coast fever. The latter disease is estimated to kill half a million cattle a year in Africa (LPS).

BOOK REVIEW — BOEKRESENSIE

VETERINARY REPRODUCTION AND OBSTETRICS

GEOFFREY H. ARTHUR, DAVID E. NOAKES AND HAROLD PEARSON

5th Edn Baillière Tindall, London SW1P 1 SB. 1982 pp 501, 330 illustrations and 30 tables, Price R51,95
(ISBN 0-7020-0923-7)

This is an excellent book for the veterinary student (undergraduate as well as post-graduate) and the practioner with a special interst in theriogenology. A systems approach is followed in the book. At the outset of each section the reader is provided with a sound physiological basis before proceeding to the clinical aspects.

The references are current and provide one, for instance, with the latest information on the endocrine control of parturition. The illustrations are excellent, particularly those in Parts II and III, which deal with "Pregnancy and Parturition" and "Dystocia and Other Problems Associated with Parturition" respectively. The step by step illustrations make the text easy to follow, even for the uninformed under-graduate student. There is a good section on defects of the penis and prepuce and correction thereof (Part VI) for the prospective penis surgeon. The final chapter of the book provides some useful physiological data on reproduction in the camel.

H.J. Bertschinger