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TYDSKRIF VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING

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CONTENTS/INHOUD

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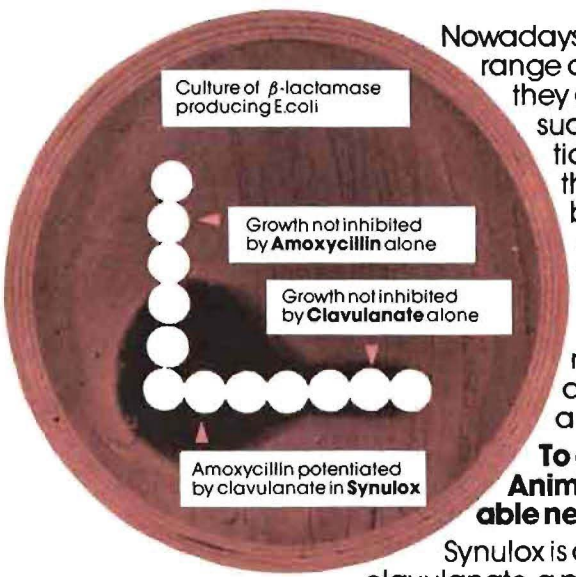

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TYDSKRIF VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING

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BERIA

ARTICLE ARTIKEL

SOME ASPECTS OF SUPEROVULATION AND FERTILISATION OF DORPER SHEEP

J G E VAN ZYL*, D F STEYN**, W A VAN NIEKERK*, R J COERTZE* AND H T GROENEVELD

ABSTRACT: Van Zyl J G E; Steyn D F; Van Niekerk W A; Coertze R J and Groenewald H T. **Some aspects of superovulation and fertilization of Dorper sheep.** *Journal of the South African Veterinary Association* (1978) **58** No 1, 3-4 (En). Dept. Animal Science, University of Pretoria, 0083 Hillcrest, Republic of South Africa. Response of Dorper sheep to treatment with FSH and PMSG and fertilisation after natural service, natural service following application of $PGF_{2\alpha}$ -containing gel on the posterior os cervix and surgical insemination with fresh semen were evaluated. FSH gave a significantly ($P > 0,05$) better response than PMSG. No significant differences between the different methods of fertilisation were observed.

Key words: Dorper sheep, superovulation, fertilisation.

INTRODUCTION

Little information regarding the superovulatory response of indigenous sheep in general, and Dorper sheep in particular, is currently available. As a result of the increasing importance of embryo transfer in the livestock industry, different methods regarding the superovulation and fertilisation of superovulated ewes were investigated.

MATERIAL AND METHODS

During October 1985, 25 adult, non-lactating Dorper ewes in good condition were synchronised using intravaginal sponges, each impregnated with 60 mg medroxy-progesterone acetate (Repromap, Upjohn (Pty) Ltd). These ewes (5 animals per group) were randomly assigned to the following treatments:

1. Superovulation with a single dose containing 1250 IU pregnant mare serum gonadotrophin (PMSG) (Seragon, Ferring GmbH), administered via intramuscular injection 48 hours prior to sponge withdrawel on day 12. Oestrus was determined by vasectomised rams, whereupon surgical insemination[9] with 0,02 ml freshly collected semen per uterine horn ($\pm$ 48 x 10^6 live spermatozoa) was carried out.
2. Superovulation as in (1). Ewes in oestrus were served twice by rams of proven fertility and mating ability with a 12 hour interval.
3. Superovulation with follicle stimulating hormone (FSH) (FSH-P, Burns Biotec). A total dosage of 24 mg FSH, spread over six intramuscular injections starting on day 10 after sponge insertion, were given. Sponges were withdrawn on day 12 and oestrus determined by vasectomised rams. Insemination as in (1).
4. Superovulation as in (3). Prostaglandin F_{2a} containing gel (experimentally prepared by mixing 50 mg dinoprost (Lutalyse, Upjohn Pty. Ltd) with 0,88 g hydroxy ethyl cellulose, made up to 27 ml with distilled water) was applied on the posterior os cervix when oestrus, as indicated by vasectomised rams, was observed. Ewes were served one hour later by fertile rams.
5. Superovulation as in (3) and fertilisation as in (2).

Ewes were flushed three to four days after oestrus according to recognised methods[9]. Statistical analyses were done by means of the General Linear Models programme of the Statistical Analysis Systems (SAS) and levels of significance determined by Bonferoni's test.

Superovulatory response was determined by counting the number of corpora lutea (CL) on both ovaries. The number of unovulated follicles (UF) was noted. The fertilisation rate was calculated as the percentage fertilised ova (as determined by means of a stereo microscope, 45 x magnification) of the total number of ova recovered.

RESULTS AND DISCUSSION

Results obtained are presented in Table 1.

Number of CL and UF: The superovulatory response with FSH was significantly better than the response with PMSG ($P = 0,004$, Table 1). Similar results have been obtained in Angora goats [3 4] and Suffolk sheep[1]. Although the average number of UF did not differ significantly ($P = 0,550$, Table 1), some PMSG treated ewes had as many as 14 UF. Occasional "overstimulation" as a result of PMSG treatment has been observed[1]. Increasing dosages of PMSG have been reported to result in increased numbers of UF, and ewes with more UF were found to take longer to return to oestrus than ewes with fewer UF[7]. The higher number of UF in PMSG-treated ewes has been contributed to the longer half-life of PMSG, which results in a continued recruitment of follicles[3].

In general, it is recommended that FSH be used for commercial purposes, in spite of the increased workload[1] (extra injections, increased labour requirements), especially since the use of PMSG may result in premature luteolysis[3].

Fertilisation rates: No significant difference in fertilisation rate was observed between surgical insemination,

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natural service and natural service following the application of $PGF_{2\alpha}$ containing gel to the cervix in FSH-treated ewes. The nonsignificance arises from the relative small number of animals available for each method of fertilisation, and more animals per treatment would probably have shown significant differences. Fertilisation rates from ewes treated with PMSG were excluded, due to the small number of ova and embryos recovered from these animals.

Reports indicating both enhanced fertilisation with surgical insemination when compared to natural service as well as the contrary, exist,[2 8] but it should be borne in mind that different breeds of sheep were used, and a possible breed effect cannot be excluded.

Application of $PGF_{2\alpha}$-containing gel one hour prior to mating resulted in a 66,8% fertilisation vs. 47,50% for ewes mated naturally without the application of gel (Table 1). Although these differences were non-significant, the coefficient of variation was less for the gel treated group, indicating a more uniform fertilisation rate. This result supports the observation[5] that significantly more spermatozoa reach the uterus and oviducts of $PGF_{2\alpha}$ gel-treated ewes than in controls within a specified time after insemination. The technique could be valuable when frozen semen is used, and the labour and time required for either surgery or laparoscopy[6] is to be avoided, since low fertilisation rates with cervical insemination of frozen semen has been reported[2].

No clear picture regarding the best way of fertilisation emerged. Since the use of frozen semen will probably gain importance in future with the increasing awareness of performance testing, and thus the better utilisation of semen from superior rams[6], refinement of existing techniques demands further investigation. Furthermore, methods to stimulate a more complete ovulation in superovulated ewes could result in a higher rate of embryo production, thus reducing overhead costs.

Attention should be given to the level of feeding and management of donor ewes, since overfat and too lean animals will in all probability give poor results, either because of difficulty in surgery in overfat animals or failure to respond in lean ones.

Table 1: Results obtained with different superovulatory regimes and methods of fertilisation

Parameter	Treatment	LSM*	SE**	P***
No. corpora lutcea (CL)	FSH	18,229	2,419	P = 0,004
	PMSG	6,700	2,712	
No. unovulated follicles (UF)	FSH	3,000	1,191	P = 0,550
	PMSG	4,083	1,335	
Fertilisation rate (%) (all animals superovulated with FSH)	Natural service	47,50	15,35	
	Natural service + gel	66,80	19,42	
	Surgical insemination	93,3	23,21	

* Least square means
** Standard error
*** Probability of differences being non-significant

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The authors acknowledge the assistance of Upjohn (Pty) Ltd., Lion Bridge Feeds and Centaur Laboratories.

REFERENCES

1. Armstrong D T, Evans G 1983 Factors influencing success of embryo transfer in sheep and goats. Theriogenology 19:31-42
2. Armstrong D T, Evans G 1984 Intrauterine insemination enhances fertility of frozen semen in superovulated ewes. Journal of Reproduction and Fertility 71:89-94
3. Armstrong D T, Pfitzner A P, Warnes G M, Ralph M M, Seamark R F 1983 Endocrine responses of goats after induction of superovulation with PMSG and FSH. Journal of Reproduction and Fertility 67:395-401.
4. Armstrong D T, Pfitzner A P, Warnes G M, Seamark R F 1983. Superovulation treatments and embryo transfer in Angora goats. Journal of Reproduction and Fertility 67:403-410
5. Bruckner G, Kampfer I 1983. Wirkung einer zervikalen Prostaglandin $F_{2\alpha}$-gel applikation auf der spermienmigration im Genitaltrakt des Schafes post inseminatiomen mit 131 I-markierten sperma. Monatschifte für Veterinarmedizin 38:221-226
6. Lawrenz, R 1985 Preliminary results of non-surgical intra-uterine insemination of sheep with thawed frozen semen. Journal of the South African Veterinary Association 56:61-63
7. Mutiga E R, Baker A A 1982 Superovulatory response in Merino ewes to three PMSG dose levels. Theriogenology, 17:1
8. Trounson A O, Moore N W 1974 Fertilisation in the ewe following multiple ovulation and uterine insemination. Australian Journal of Biological Science 27:301-304.
9. Willadsen S M 1980 Embryo transplantation in sheep. In: The Management and Diseases of Sheep. Commonwealth Agricultural Bureau, Farnham.

ARTICLE ARTIKEL

PROFESSIONELE GEHEIMHOUDING EN BEKENDMAKING VAN INLIGTING IN DIE VETERINÊRE BEROEP

H.N. VAN DER MADE*

ABSTRACT: Van der Made H.N. **Professional secrecy and the divulgence of information within the veterinary profession.** *Journal of the South African Veterinary Association* (1987) No. **58** No 1, 5-7 (Afrik) Department of Infectious Diseases, Faculty of Veterinary Science, University of Pretoria, P/Bag X04, 0110 Onderstepoort, Republic of South Africa.

Aspects of professional secrecy as applied to the veterinary profession are examined. The origin and the modern basis of this precept are referred to. Consideration is given to its application in the Veterinary and Paraveterinary Professions Act, the common law liability of the veterinarian employed in industry, the veterinarian negotiating the purchase of a practice, and its application to moral, ethical and contractual obligations. The need to dispense with professional secrecy in emergency situations is examined, as well as an approach for the discussion of clinical cases amongst colleagues, publication of such cases in scientific journals and the application in a situation where a veterinarian is being sued by a client. Professional secrecy is also examined where the veterinarian is employed in the Public Service and where an obligation arises to give evidence in civil and criminal court proceedings.

Key words: Veterinary professional secrecy, veterinary ethics.

INLEIDING

Die vereiste van geheimhouding van inligting wat uit die praktyk verkry word in die mediese beroepe, kan teruggevoer word na Hippokrates van Cos (460 VC). Een van die bepalings wat in die eed van Hippokrates[14] neergelê word, lees as volg: *"Whatever in connection with my professional practice, or not in connection with it, I see or hear in the life of men, which ought not to be spoken abroad, I will not divulge, as reckoning that all such should be kept secret".*

Hierdie vereiste vir geheimhouding vind ook toepassing op die veterinêre beroep en maak deel uit van die veterinêre etiese kodes van baie lande en ook van die RSA. Hier te lande is dit 'n wetlike- en professioneel etiese opgelegde verpligting vir die veearts. Daarenteen bestaan daar ook wetlike en morele verpligtings tot openbaarmaking van sekere inligting en 'n noodtoestand kan dit ook noodsaaklik maak.

Die verpligting tot geheimhouding van inligting word daarin gesien dat elke persoon 'n persoonlikheidsreg tot privaatheid het. Die motivering vir die bestaan van hierdie reg word as volg saamgevat deur Joubert[13]: 'Daar kan geen reg wees meer inherent aan die erkenning van die eiewaarde van die mens as sy aanspraak om in sy private lewe in 'n mate afgesonderd van die openbaarheid te lewe nie...' en verder tot die gevolgtrekking kom: 'Dit is 'n ernstige krenking van die eer van die mens om in sy private lewe in te dring of om dit bloot te lê vir die oë en ore van die publiek, of selfs vir die enkeling wat geen reg op die kennisname het nie'.

'n Persoon kan sy reg op privaatheid laat geld en beskerm deurdat hy skadevergoeding kan verhaal vir die deliktuele oortreding van inbreukmaking op hierdie reg.

GEHEIMHOUDING VAN INLIGTING

Kennisname van die bestaan van so 'n reg op privaatheid is van groot belang vir die veearts wat in sy professionele hoedanigheid 'n persoonlike diens aan kliënte lewer en sodoende kennis opdoen van hulle persoonlike sake, besigheidsbedrywe, en moontlik ook besigheidsgeheime.

Vir die veearts self, word hierdie reg op privaatheid beskerm deur Wet 19 van 1982[1]. Persone wat onder hierdie wet werksaam is, soos diegene in diens van die professionele beheerliggaam, is by die uitvoering van hulle pligte verplig tot geheimhouding van inligting wat hulle verkry oor die besigheid of sake van geregistreerde persone, behalwe waar dit te doen het met die uitvoer van pligte onder die wet of waar 'n hof hulle verplig tot bekendmaking. Geen strafbepalings is in die wet gestel vir die oortreding van hierdie beperking nie, maar 'n benadeelde sou privaatregtelik teen so 'n persoon kon optree, afgesien van die feit dat 'n goeie saak vir onetiese optrede uitgemaak sal kan word.

Die veearts wat doenig is in die bedryf van medisynevervaardiging, prosessering van voedsel vir menslike gebruik, die bio-industrie of die dierevoerbedryf en so meer, neem onvermydelik kennis van prosesse wat gebruik word. Dit mag prosesse wees wat eie is aan die besondere bedryf en nie tot die kennis van soortgelyke ondernemings mag kom nie. Dit is belangrik om te besef dat die vrylating van sulke inligting tot deliktuele aanspreeklikheid kan lei. Waar dit aan ongemagtigde persone vrygestel word, sou die besondere onderneming skadevergoeding kon verhaal indien as gevolg daarvan skade gely is.

Daar rus 'n verpligting op 'n persoon vir die bewaring van inligting wat uit 'n kontraktuele ooreenkoms verkry word[16]. Dit vind toepassing by die veearts wat sy professionele funksies in die normale kontraktuele verhouding tot sy kliënt uitvoer en vind ook weerklank in die kontraktuele verhouding van 'n veearts wat met 'n kollega onderhandel aangaande die koop van 'n praktyk. Inligting wat uit sulke ooreenkomste verkry word mag nie aan derdes vrygestel word nie.

Op elke persoon rus 'n morele verpligting om nie inligting wat aan hom in vertroue meegedeel is, aan die wêreld uit te basuin nie. Hierdie verpligting geld in die vertrouensverhouding tussen die veearts en sy kliënt.

Hierdie inligting stel hom dikwels in staat om deur te dring na die grond oorsaak van siektetoestande soos bestuursaspekte en so meer wat remediëring nodig het. Die voortsetting van 'n effektiewe diens aan die kliënt is

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afhanklik van die ongeskondenheid van hierdie vertrouensverhouding.

Die veearts word gebind deur 'n veterinêre etiese verpligting tot geheimhouding. Hierdie reël vereis dat 'n persoon wat 'n veterinêre beroep beoefen, alle inligting wat hy in die loop van sy beroepsbeoefening bekom en wat op 'n dier betrekking het, as streng vertroulik behandel, ongeag of sulke inligting bekom word as gevolg van die ondersoek, diagnose of behandeling van daardie dier, of deur iemand aan hom meegedeel is[2]. Enige oortreding van hierdie reël sal deur die beroepsbeheerliggaam as onprofessionele gedrag beskou word.

Die veearts wat 'n diagnose aan 'n kliënt meedeel, doen dit in die *bona fide* uitoefening van sy beroep, dat dit 'n ware uitdrukking is van wat hy glo om die probleem van sy besondere pasiënt te wees. Die mededeling van sy diagnose aan ander of aanverwante persone tot sy kliënt[3] sal nie geregverdig wees nie, tensy die siekte objektief gesien, 'n ernstige bedreiging vir sulke persone of hulle diere inhou. Die siekte moet 'n werklike noodtoestand skep om bekendmaking te regverdig, waar belange van bedreigde persone die privaatheidsbelange van die kliënt oorheers. Waar 'n aanmeldbare siekte betrokke is, sal dit meestal 'n Staatsveearts se taak wees om sulke kennis te gee.

Waar 'n statutêre verpligting tot aanmelding afwesig is, bly dit 'n morele verpligting. Die beoordeling na watter geval aanmelding vereis, is subjektief en moet versigtig mee te werk gegaan word. Die aard van die geval sal bepaal aan wie dit bekend gemaak moet word. Die misdaad van bestialiteit wat onder die veearts se aandag kom, sou byvoorbeeld by die geregsowerhede aangemeld word met geen wyer bekendmaking nie.

Daarteenoor sou die bekendmaking van 'n aansteeklike siekte van diere aan die bure van 'n besmette plaas geregverdig wees, sodat voorsorgmaatreëls getref kan word, maar sou geen plek hê om verder bekendgemaak te word nie.

Bekendmaking mag ook dringend word waar 'n veearts gevaar loop om as mededader aan 'n misdaad beskou te word. Enkele uitsonderings kom voor waar inligting aan derde onbetrokke persone bekendgemaak kan word: Dit word aanvaar dat veeartse onderling kliniese gevalle kan bespreek. Dit sou nie oneties wees nie, alhoewel die gewoonte bestaan om die naam van die betrokke eienaar te verswyg[15].

Geen wetlike of etiese besware kan gehef word teen publikasie van besonderhede van veterinêre gevalle in artikels in veterinêre of mediese joernale nie. Waar die identiteit van die eienaar of sy diere in die besonder, nie blootgelê word nie, is toestemming oorbodig. Maar andersins mag dit '...slegs met die toestemming van die eienaar van die betrokke dier aan iemand anders openbaar word'[4].

Die verpligting tot geheimhouding van inligting val weg waar 'n praktisyn deur 'n kliënt gedagvaar word in 'n siviele of kriminele geding. Inligting wat normaalweg vertroulik sou wees en wat 'n essensiële deel van die praktisyn se verdediging sou uitmaak, mag deur hom aangewend word[12].

Die verpligting tot geheimhouding van inligting teenoor kliënte, verskil nie in amptelike diens van enige ander diens wat gelewer word nie. Die professionele persoon is steeds verplig tot geheimhouding, behalwe waar hy 'n statutêre verpligting tot bekendmaking opgelê word. Gevalleverslae van diere van kliënte is vertroulike inligting wat nie gesien of inspekteer mag word deur persone wat nie direk by die gevalle betrokke is nie. Ook nie die direkte bevel van 'n hoof in die Staatsdiens kan geheimhouding negeer nie en sou *ultra vires* wees tensy dit statutêr onderskraag word.

Nie-professionele of ongeregistreerde personeel wat in 'n veearts se diens is en wat nie deur etiese reëls gebind word nie, moet deur hom aangesê word om die geheimhouding van inligting te bewaar, waar daarmee in aanraking gekom word[11].

VERPLIGTE BEKENDMAKING VAN INLIGTING

'n Geldige beginsel in ons reg bepaal dat enige persoon wat in 'n hofsaak betrokke is, hom op enige ander persoon kan beroep om inligting bekend te maak waaroor hy omtrent die aangeleentheid beskik[12]. 'n Hofbevel kan dienooreenkomstig uitgereik word om 'n persoon te dwing om sulke inligting in 'n hof bekend te maak. Op dieselfde wyse kan professionele persone gedwing word om inligting wat hulle in die loop van hulle professionele aktiwiteite bekom het te verstrek. Geen wetlike voorsiening bestaan vir uitsluiting van sulke inligting nie[10]. Voor 'n hofverhoor aan die gang is, moet 'n professionele persoon weier om enige inligting bekend te maak. Word hy gedagvaar, is hy verplig om in die hof te verskyn, omdat nie-verskyning 'n vonnis van minagting van die hof tot gevolg mag hê. In die loop van die saak mag hy vervolgens versoek word om 'n mededeling te maak, waartoe hy die regterlike amptenaar se beslissing moet vra. Sou laasgenoemde hom beveel om die mededeling te maak, word hy daartoe verplig[5]. Sulke bekendmaking van inligting gaan gepaard met die voorbehoud dat indien dit 'n inkriminasie van die professionele persoon self sou meebring, hy nie daartoe verplig kan word nie.

'n Persoon kan ook onder 'n bepaling van die kriminele kode deur 'n magistraat verplig word, om inligting wat normaalweg as vertroulik geld, bekend te maak[6]. In die siviele hofprosedure kom 'n soortgelyke geval voor, waardeur 'n persoon as kliënt van 'n professionele persoon, inligting aangaande homself kan opeis. Hierdie inligting kan vrygestel word onder Hofreël 36[7].

Die Wet op Dieresiektes[8*] verplig veeartse om siektes wat beheerde dieresiektes is, en wat by 'n dier, voortbrengsel van 'n dier, of produk aangetref word, by die Direkteur van Veeartsenydiens aan te meld. Hierdie siektes word gelys in die staande regulasies onder dieselfde wet. Geheimhouding moet bewaar word van alle inligting wat betrekking op die besigheid of sake van 'n persoon het, en wat deur werksaamhede onder die wet verkry word. Sulke inligting kan alleenlik vir geregtelike verrigtinge onder die besondere wet vrygestel word, of vir ander hofverrigtinge (uitgesonderd die van 'n siviele hof), of kragtens 'n ander wet, of met die skriftelike toestemming van die Minister van Landbou Tegniese Dienste[9]. Dieselfde beperkings geld vir 'n insae in die aantekeninge en registers wat onder die besondere wet gehou word[10].

GEVOLGTREKKINGS

Na die beste van my wete het daar tot op datum nog geen sake voor die howe in Suid-Afrika gedien, waar 'n veearts betrokke was by vertrouebreuk teenoor sy kliënt nie. Dit bly egter belangrik om van die implikasies van

*Hierdie wet sal waarskynlik eersdaags in werking gestel word.

statute bewus te wees en om aan die morele en etiese verpligtinge herinner te word.

In vergelyking met die medikus, waar integriteit van die kliënt se liggaam sowel as sy sake in die gedrang kom by geheimhouding, is in die geval van die veearts slegs saaklike regte betrokke. Skadevergoedingseise vir saakbeskadiging en gevolgsverliese kan egter ook aansienlike bedrae beloop.

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BRONNELYS

1. Anoniem Wet op Veterinêre en Paraveterinêre Beroepe (Wet 19 van 1982) Art 37
2. Anoniem GK R2086 van 1.10.1982, Reël 15.1
3. Anoniem Ex Parte Rautenbach 1938 SA 150: 152-153
4. Anoniem GK R2086 van 1.10.1982, Reël 15.2.1
5. Anoniem GK R2086 van 1.10.1982, Reël 15.2.2
6. Anoniem Strafproseswet (1955) (Wet 56 van 1955) Art 212 soos gewysig met art 27 van Wet 80 van 1964
7. Anoniem Hooggeregshofreglement GK R48 van 12.1.1965
8. Anoniem Wet op Dieresiektes 1984 (Wet 35 van 1984), Art 11
9. Anoniem Wet op Dieresiektes (Wet 35 van 1984) Art 25(1)
10. Anoniem Wet op Dieresiektes (Wet 35 van 1984) Art 25(2)
11. Gordon I, Turner R, Price TW 1953 Medical Jurisprudence 3rd edn E & S Livingstone : 53
12. Hiemstra VG 1967 Strafprosesreg. Butterworths, Durban
13. Joubert WA 1953 Grondslae van die Persoonlikheidsreg. AA Balkema, Kaapstad : 135
14. Mason JK, McCall Smith RA 1983 Law and Medical Ethics. Butterworths, London. : 251
15. Masters NC & Shapiro HA (1966) Medical Secrecy and the Doctor-Patient Relationship AA Balkema, Kaapstad.
16. SALJ Vol XXXIV p 53, Analoog met die verhouding van *fiducius* tussen 'n prokureur en sy kliënt, Wessels Law of Contract in SA 1e uitgawe Vol 1 para 1235

ABSTRACT

SAMEVATTING

TICK TRANSMISSION OF *ANAPLASMA CENTRALE*

Anaplasma centrale was isolated from a field collection of *Rhipicephalus simus*. Transstadial transmission of *A. centrale* with adult ticks was demonstrated, but the infection was not carried transovarially. Ticks from this collection were subsequently reared as a non-infected, laboratory strain. It was proved that the Onderstepoort live blood vaccine strain of *A. centrale*, isolated by Theiler in 1911, is still tick transmissible after more than 75 years of needle passage through cattle in the laboratory. Attempts to demonstrate transstadial transmission of the vaccine strain with *Boophilus decoloratus* and *Boophilus microplus* failed. (Potgieter, F.T. & Van Rensburg, L., 1987. Tick transmission of *Anaplasma centrale*. *Onderstepoort Journal of Veterinary Research*, 54, 5-7 (1987).)

ABSTRACT

SAMEVATTING

CONTROL OF INDUCED INFESTATIONS OF ADULT *AMBLYOMMA HEBRAEUM*

The efficacy of ivermectin, administered in a sustained release formulation by intraruminal pumps at approximate daily dose rates of 20, 40 and 60 μg/kg, was evaluated in 16 cattle against induced infestations of 3 strains of adult *Amblyomma hebraeum*. Engorged female ticks were mass-measured and incubated, and reproductive data recorded. There was an increase in mortality of male and female ticks compared to that of controls with increasing daily dose of ivermectin, and a decrease in the number of ticks engorging. Ticks fed on ivermectin-treated cattle had a smaller mass when engorged and laid smaller egg masses, both absolutely and as a proportion of engorged mass. Index of reproduction was reduced 100% at 60 μg/kg/day, >99% at 40 μg/kg/day and 96% at 20 μg/kg/day. Differences occurred between the 3 strains of *A. hebraeum* used in the study, especially with regard to engorged mass and reproductive variables.

Practical implications of the application of sustained release ivermectin for the control of *A. hebraeum*, specifically with reference to heartwater *(Cowdria ruminantium)*, are discussed. (Soll, M.D., Carmichael, I.H. & Gross, Sheila J., 1987. Control of induced infestations of adult *Amblyomma hebraeum* with sustained release ivermectin. *Onderstepoort Journal of Veterinary Research* 54, 17-20 (1987).)

ABSTRACT

SAMEVATTING

SALMONELLA AND *ESCHERICHIA COLI* ISOLATED FROM REGISTRABLE FARM FEEDS

Resistance to 20 antibiotics of 128 *Salmonella* and 97 *Escherichia coli* isolates from various registrable farm feeds was determined. A high frequency of comparatively low levels of resistance was found in both the *Salmonella* and *E. coli* isolates. This, together with an elevated frequency of multiple resistance, indicates that problems related to an effective transfer in bacterial populations of resistance to certain antibiotics are a distinct possibility. The addition of antibiotics, such as penicillin and tetracyclines, to animal feeds can create conditions for rapid selection amongst bacteria resistant to antibiotics. The numbers of resistant bacteria in the animal environment may be increased and may lead to the development of veterinary and human health problems from the possible transfer of antimicrobial resistance from animal pathogens to human pathogens or spreading in the human population of animal pathogens resistant to antibiotics.

There is a need for caution in the use of antibiotics, particularly in animal feeds. Extended survey of, and epidemiological research on, farm feeds, manufacturing mills and animal production units are emphasized. (Durand, Anette M., Barnard, Marie-Luise, Swanepoel, Martha L. & Engelbrecht, Marie M., 1987. Resistance to various antibiotics of *Salmonella* and *Escherichia coli* isolated from registrable farm feeds. *Onderstepoort Journal of Veterinary Research*, 54, 21-26 (1987).)

ARTICLE ARTIKEL

COMPARISON OF VARIOUS CRITERIA FOR DETERMINING THE HEALTH STATUS OF THE BOVINE UDDER

J.H. DU PREEZ*

ABSTRACT: Du Preez J.H. **Comparison of the various criteria for determining the health status of the bovine udder.** *Journal of the South African Veterinary Association* (1987) **58** No. 1, 9-14 (En). Department of Veterinary Public Health, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, 0110 Onderstepoort, Republic of South Africa.

The International Dairy Federation's (IDF) criteria for classification of subclinical bovine health conditions does not provide a true picture of the health status of the udder. When combining these criteria with the serum albumin levels of milk samples, the IDF's classification of sub-clinical mastitis could be extended to include 8 possible conditions such as normal quarters, septic mastitis, aseptic mastitis, relevant and irrelevant teat canal infection, specific and unspecific hyperalbumingalactia and unspecific cellular reaction. When the bacteriological results of teat canal swab samples were combined with the IDF's standards, these conditions could be scaled down to 4 diagnoses which included normal quarters, aseptic mastitis, teat canal infections, subclinical mastitis and a combination of the latter 2 conditions. When applying the teat canal swabbing technique, up to 16% of quarters classified as normal and 10% as cases of aseptic mastitis according to the IDF's criteria in fact, have TCI.

Since classification of the health status of quarters according to the IDF's criteria and with due regard to the bovine serum albumin values of milk, did not include the bacteriological results of the teat canal swab samples, further research must be done to clarify the existence of conditions in the IDF classification.

Teat canal infections may result in damage to the udder parenchyma as manifested by the elevated bovine serum albumin content of milk.

Key words: Bovine serum albumin, International Dairy Federation's criteria, teat canal swab samples.

INTRODUCTION

The International Dairy Federation (IDF) defined mastitis according to recommendations of Kästli[17] and Tolle[28], who based their diagnoses on bacteriocytological findings on foremilk. When applying these criteria, a quarter may be classified as normal, as having a latent infection (LUI), subclinical (SCM) or clinical mastitis or it may suffer from a specific or an aseptic mastitis (ASM). From the literature on clinical and subclinical mastitis [9 22 30], it is apparent that the disease is a multifactorial condition and SCM should be considered as a problem giving cause for special concern.

Inflammation results in increased capillary permeability[20 21]. In the case of mastitis an increased permeability would be noticed by an increase in the serum albumin content of the milk[11 13 29]. The concentration of bovine serum albumin (BSA) in milk is believed to be a useful criterion for determining the degree of inflammation in clinical mastitis[21 23]. Giesecke & Viljoen[13] also report that the BSA concentration in milk is a suitable criterion for estimating the defect of tissue damage in SCM. In addition, they stated that it is possible to differentiate between infection of the teat canal per se and the udder by bacteriological examination and the estimation of the BSA level and somatic cell content of the milk.

According to Giesecke et al.[14] the conventional and the new polyvalent immunodiffusion tests recommended for the diagnosis of mastitis in dairy cows leave much to be desired with regard to their repeatability of diagnostic accuracy.

By applying both the IDF's standards and BSA concentrations on the same milk samples, Giesecke & Barnard[10] were able to extend the above classification to one which also included disease conditions such as relevant (RTI) and irrelevant teat canal infection (ITI), specific (SHAG) and unspecific hyperalbumingalactia (UHAG) and unspecific cellular reaction (UCR). In addition to this new classification, the BSA content of milk was also shown to be a useful criterion for estimating the degree of inflammation in cases of clinical mastitis as well as the degree of tissue damage in the case of SCM. By determining the presence of bacteria in milk samples, the BSA levels and the somatic cell counts (SCC) thereof, Giesecke & Viljoen[10] also found it possible to differentiate between teat canal infections and actual mastitis.

The Kruskal-Wallis distribution-free test[29] showed that the BSA concentration in milk is less suitable as a diagnostic criterion than the SCC for differentiating between udder and teat canal infections (TCI) involving primary pathogens. This is at variance with the findings by Giesecke & Viljoen[13]. Verhoeff & Smit[29] state that the cell count provides more reliable information than does the BSA level of milk. Milk antitrypsin is a sensitive indicator of mastitis[27] as well as in milk BSA concentrations [16 18 27]. A high correlation between milk BSA content and SCC/ml (Coulter Counter method) was seen (r = 0,742) when quarter milk samples were analysed[15].

Du Preez[4-7] gave a review on the diagnosis, incidence, nature, therapy, extent and significance of TCI or colonization in dairy cows. He stated that TCI can only be diagnosed accurately with teat canal swab samples (TCSS). In the routine examination of foremilk samples (FMS) many cases of TCI are not recognised and TCI serves as a potential source of bacteria for infection of the udder parenchyma.

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The aim of this study was to compare the status of various udder conditions diagnosed according to the IDF's criteria alone, the IDF criteria in combination with the diameter of monovalent radial immunodiffusion reactions (BSA precipitation zones) in mm, and lastly the cytology of the IDF's criteria in combination with the bacteriological results of the TCSS to determine whether TCI can lead to damage of the udder parenchyma, as indicated by the BSA content of milk determined by the average diameter of the BSA precipitation zone of milk from quarters with TCI.

MATERIALS AND METHODS

Experimental animals

The investigation was performed on 250 Friesian dairy cows in 3 different herds maintained on the Transvaal Highveld on a semi-intensive system and milked twice a day. They were kept under similar conditions of feeding and relatively good management, animal husbandry and hygiene. The cows varied in age, number of lactations, daily milk yield and stage of lactation (2-8 months). All the cows were clinically healthy and in good condition. Disinfectant teat dipping was routinely applied after milking by immersion in chlorhexidine gluconate 0,75% m/v ("Hibitane" G1000, ICI, Johannesburg) teat dip.

Collection of parallel FMS and TCSS

FMS and TCSS were obtained immediately before milking and after thorough udder washing with clean running water, drying with a disposable paper towel and disinfecting the teat end with 70% alcohol on cotton wool. After discarding the initial 3 jets of foremilk, a quarter milk sample was aseptically collected into a sterile 5 ml 'Monoplast' (Labotec, Johannesburg) tube following the standard procedure[19]. Teat canal swabs were prepared and TCSS taken as described by Du Preez[4-7]. Before implementing our sampling routine we had established the effectivity of the disinfection process by swab sampling and culturing 20 teat tips subsequent to disinfection; all were bacteriologically negative. All samples were transported on ice and analysis proceeded within 6 hours of sampling.

SCC

A defatted Prescott-Breed smear[1] of each FMS was stained according to the Broadhurst-Paley method[25]. The SCC per ml milk was determined microscopically after counting the cells in 100 microscopic fields[1].

Isolation and identification of bacteria

Facultative and micro-aerophilic bacteria were isolated as described by Du Preez[4] and Du Preez & Greeff[5]. Identification and speciation was done according to the methods and criteria described by Cowan & Steel[2]. Specific attempts to culture anaerobic bacteria were excluded for practical reasons.

BSA (= Monovalent radial immunodiffusion test)

The BSA test was performed according to the methods described by Giesecke & Viljoen[13] and Giesecke et al.[14].

Health status of quarters and classification

The health status of quarters, determined according to data obtained by examination of FMS, was classified according to the IDF's criteria[17,28]. The health status of the same quarters was classified according to IDF's criteria based on the cytological examination of the FMS and the bacteriological examination of the TCSS. The health status of the quarters was also classified ac-

Table 1: The prevalence of normal quarters, teat canal infection (TCI), aseptic mastitis (ASM) and subclinical mastitis (SCM) diagnosed according to the foremilk sample (FMS) (International Dairy Federation's (IDF)) criteria and teat canal swab sample (TCSS) (IDF criteria combined with TCSS)* in 99 quarters, with details of the average somatic cell count (SCC) and bovine serum albumin (BSA) precipitation zones

Status of quarter	Bacteria isolated	Method of sampling of frequency of status		Average BSA precipitation zone, mm		SCC X 10^3/ml of milk	
		FMS (%)	TCSS (%)	FMS	TCSS**	FMS	TCSS+
Normal		69 (69,7)	53 (53,5)	4,7	4,6	172	166
LUI/TCI***		10 (10,1)	26 (26,3)	5,2	5,1	232	225
	Staphylococcus aereus	1	8	4,0	5,5	175	175
	Coagulase negative staphylococci (CNS)	5	13	4,8	5,0	260	266
	Streptococcus spp.	1	2	6,9	5,5	250	58
	Other mastitogenic bacteria (OMB)		3	4,8	3,9	364	210
ASM		13 (13,1)	3 (3,3)	7,3	6,1	1,367	1,596
SCM		7 (7,1)	17 (17,2)	5,5	6,3	2,883	1,979
	S. aureus	1	1	6,1	6,1	628	628
	CNS	3	10	6,2	7,9	7,221	2,692
	Streptococcus spp.	0	0				
	OMB	3	6	5,0	5,9	1,026	1,098
Total		99 (100)	99 (100)				

* See Materials and Methods

** The BSA precipitation zone and the SSC+ was done/determined on the FMS after the health status of quarters was classified according to the combined IDF/TCSS criteria — see Materials and Methods

*** Classified here according to Du Preez[5]

cording to the combined IDF's and BSA criteria[10] (Table 5). For the purpose of this report it was assumed that the presence of bacteria in TCSS indicates a TCI.

Coagulase test

The tube coagulase test on cultures of staphylococci was done according to the method described by Cruikshank et al.[3].

RESULTS

From Table 1 it is apparent that the prevalence of the various udder health states differed according to the type of sample investigated. Such differences become still more obvious from a comparison of the percentage values of udder quarters classified according to their health condition.

Table 2: The status of 10 of the 99 quarters (Table 1) according to the International Dairy Federation's (IDF) criteria alone, IDF criteria with due regard to the diameter of the bovine serum albumin (BSA) precipitation zones and lastly the IDF criteria (only the somatic cell count (SCC)) with due regard to the bacteriological results of the teat canal swab sample (TCSS)

Quarter No	Bacteria isolated		SSC x10³ per ml milk	BSA mm	Status of quarter according to		
	Foremilk sample	TCSS			IDF criteria	IDF criteria combined with BSA values	IDF criteria combined with TCSS results
1	None	None	1,237	6,9	Aseptic mastitis (ASM)	Unspecific cellular reaction (UCR)	ASM
2	None	CNS and *Corynebacterium bovis*	1,525	8,1	ASM	ASM	Teat canal infection (TCI)
3	None	CNS	2,009	11,8	ASM	ASM	TCI
4	None	CNS	461	8	Normal	unspecific hyperalbumingalactia (UHAG)	TCI
5	*Staphylococcus aureus** and Coagulase negative staphylococci (CNS)	*S. aureus* and CNS	162	3,5	Latent udder infection (LUI)	Irrelevant teat canal infection (ITI)	TCI
6	None	CNS	761	6	ASM	UCR	TCI
7	*S. aureus**	*S. aureus* CNS	2,042	5,5	Subclinical (SCM)	Relevant teat canal infection (RTI)	TCI and SCM
8	*S. aureus**	*S. aureus*	12,339	8	SCM	Septic mastitis (SM)	TCI and SCM
9	*S. aureus*	CNS	125	8	LUI	Specific hyperalbumingalactia (SHAG)	TCI
10	None	None	65	5	Normal	Normal	Normal

* Coagulase positive *S. aureus*

Table 3: The health status of the same 255* quarters diagnosed as normal and mastitic according to the International Dairy Federation's (IDF), combined IDF/bovine serum albumin (BSA) and IDF/teat canal swab sample (TCSS) criteria

Quarters examined	Criteria	Status of quarter										
		N	LUI	ASM	SCM	UHAG	UCR	ITI	SHAG	RTI	TCI	TCI & SCM
255	IDF	168	40	18	29	–	–	–	–	–	–	–
255	IDF/BSA	160	–	2	16	8	17	37	1	14	–	–
255	IDF/TCSS	145	–	12	17	–	–	–	–	–	67	14

IDF criteria:
N = Normal
LUI = Latent udder infection
ASM = Aseptic mastitis
SCM = Subclinical mastitis

IDF/TCSS criteria:
N, teat canal infection (TCI)**,
IDF/BSA criteria:
ASM, SCM and SCM combined with TCI

N = Normal
UHAG = Unspecific hyperalbumingalactia
UCR = Unspecific cellular reaction
ASM = Aseptic mastitis
ITI = Irrelevant teat canal infection
SHAG = Specific hyperalbumingalactia
RTI = Relevant teat canal infection
SCM = Subclinical septic mastitis/Septic mastitis

– = Not applicable
* These are not the same quarters as discussed in Tables 1 or 2
** Classified according to Du Preez[8]

BSA: There is a slight and progressive increase in the undermentioned order (except SCM, diagnosed according to the FMS) of the size of the BSA precipitation zones for normal quarters, TCI, ASM and SCM cases diagnosed according to the FMS and TCSS method.

The health status of 10 of the 99 (Table 1) quarters examined according to the IDF's criteria, the IDF's criteria with due regard to the BSA values, and thirdly according to the TCSS, is represented. According to the combined IDF's and TCSS's criteria one finds quarters that are normal, quarters with TCI, with ASM, with SCM, and quarters that had TCI combined with SCM.

Quarters are classified for their health status according to the IDF's criteria, the IDF's and BSA's and the IDF's and TCSS's criteria combined.

The prevalence of the health status of quarters as classified according to the IDF's, IDF's and BSA's and the IDF's and TCSS's criteria combined, is represented.

Table 4: The prevalence of the health status of the same 255 quarters (Table 3) diagnosed as normal and mastitic according to the International Dairy Federation's (IDF), combined IDF/bovine serum albumin (BSA) and IDF/teat canal swab sample (TCSS) criteria

Quarters examined	Criteria	Status of quarters:	Prevalence of quarters, %											
			N	LUI	ASM	SCM	UHAG	UCR	ITC	SHAG	RTI	TCI & SCM	SCM	Total
255	IDF		65,9	15,7	7,0	11,4	–	–	–	–	–	–	–	100
255	IDF/BSA		62,7	–	0,8	6,3	3,1	6,7	14,5	0,4	5,5	–	–	100
255	IDF/TCSS		56,8	–	4,7	6,7	–	–	–	–	–	26,3	5,5	100

N = Normal
LUI = Latent udder infection
ASM = Aseptic mastitis
SCM = Subclinical mastitis
UHAG = Unspecific hyperalbumingalactia
UCR = Unspecific cellular reaction
ITI = Irrelevant teat canal infection
SHAG = Specific hyperalbumingalactia
RTI = Relevant teat canal infection
TCI = Teat canal infection

IDF/BSA criteria: ITI + RTI = 20%
IDF/TCSS criteria: SCM + (TCI & SCM) = 12,2%
IDF/BSA criteria, N quarter minus IDF/TCSS criteria, N quarters = 5,9%
IDF/TCSS criteria, TCI minus IDF/BSA criteria, ITI plus RTI = 6,3%

– = Not applicable

Table 5: Key for the differentiation between subclinical mastitis and other udder health states in normally lactating cows by means of somatic cell count (SCC), bacteriological and bovine serum albumin (BSA) determinations

SCC values x 10^3 per ml of milk	Culture of potentially pathogenic bacteria	Diagnosis depending on International Dairy Federation's (IDF) criteria 28	BSA values (diameter of precipitation zone in mm)	Diagnosis depending on IDF/BSA criteria[28]*
≦500	Negative	Normal secretion	<8,0 ≧8,0	Completely normal Unspecific hyperalbumingalactia
>500	Negative	Non-specific mastitis (Aseptic mastitis)	<8,0 ≧8,0	Unspecific cellular reaction Aseptic mastitis
≦500	Positive	Latent infection	<8,0 ≧8,0	Irrelevant teat canal infection Specific hyperalbumingalactia
>500	Positive	Mastitis	<8,0 ≧8,0	Relevant teat canal infection Septic mastitis

* Amended depending on additional data [11 12]; compiled from Giesecke & Barnard[10]

DISCUSSION

Classification of subclinical bovine mastitis according to the IDF's criteria[17 28] does not provide for a condition such as TCI (Tables 3 & 4). All the corresponding TCSS (Tables 1, 3 & 4) of quarters, diagnosed and classified according to the bacterio-cytological results of the IDF's criteria as LUI, were found to be bacteriologically positive. It is assumed (Materials and Methods) that the presence of bacteria in TCSS indicates TCI, therefore TCI must be considered identical to LUI (IDF's criteria, Table 1). According to the findings of this study, how-

ever, TCI and LUI are two different entities. Newbould[22] as well as Verhoeff & Smit[29] have pointed out that the existence of a LUI is questionable. For the classification of the various forms of SCM the IDF's criteria fall short as a procedure for making an accurate evaluation since such diagnoses are based only on the bacterio-cytological results of the FMS (Table 2).

In no instance was the FMS of a quarter bacteriologically positive, the TCSS bacteriologically negative and the SCC > 500 x 10^3 per ml. Although cases occurred where the FMS was bacteriologically positive while the SCC was less than 500 x 10^3 per ml, the condition in the quarters concerned would be classified as a LUI according to the IDF's criteria, that is to say without taking the TCSS into account. Since the bacteriological results of the examination of TCSS corresponded in all cases with those of the FMS where the SCC was less than 500 x 10^3 per ml, a TCI does in fact exist in such quarters while the existence of LUI is questioned, as other workers have pointed out[24 27]. The existence of a LUI is either a condition of short duration or very rare. However, it could possibly represent a stage in the pathogenesis of udder infections.

According to the IDF's criteria and BSA precipitation zones the condition in quarters 2 and 3 (Table 2) appears to be an ASM, but since the TCSS was bacteriologically positive, it is in fact regarded as a TCI with a high SCC (> 500 x 10^3 per ml), quite probably as a result of toxins liberated by the bacteria in the teat canal. Of the 99 quarters investigated there were only three of the same status as quarters 2 and 3 (Table 3). A total of 3% could therefore have been regarded as cases of ASM according to the FMS (IDF's criteria) but they are in fact cases of TCI.

Quarter No. 1 is the only one of the 99 (i.e. 1%) quarters examined which could be regarded as an ASM on the basis of the TCSS or as a unspecific cellular reaction (UCR) on the basis of the BSA. There are several reasons for an increased (> 500 x 10^3 per ml) SCC, which could have given rise to incorrect diagnosis of the status as an ASM, e.g. due to involution of the secretory epithelium, stress conditions, late lactation and immediately after calving[8]. Thermic and chemical conditions may cause ASM where micro-organisms play no role[26]. Necrobiosis may also cause non-inflammatory changes of the mammary epithelium[9] with an increase in SCC. According to the IDF's criteria Quarter No. 6 is regarded as an ASM and according to the BSA content of the milk as a UCR. Since the TCSS was bacteriologically positive, it is regarded as a TCI with a high SCC, possibly as a result of toxins liberated by the bacteria that colonized the teat canal.

According to the IDF's criteria, Quarter No. 4 is normal, i.e. a case of unspecific hyperalbumingalactia (UHAG) on the basis of the BSA. According to the results of the TCSS it is, however, a TCI caused by *Staphylococcus aureus*. The milk in this quarter had an increased BSA content, quite probably as a result of toxin action on the udder parenchyma, which increased the permeability of the blood vessels[20 21]; the toxin could have originated from the teat canal.

According to the IDF's criteria the condition in Quarter No. 5 would be a LUI, an ITI. (BSA precipitation zone is less than 8 mm in diameter, while SSC exceeds 500 x 10^3 per ml and the FMS is bacteriologically positive); on the basis of the TCSS it would be a TCI combined with a SCM (see also Table 4 where in 5,5% of quarters a combination of TCI and SCM occurred). This indicates that udder inflammations are not necessarily differentiated sharply into types. If the milk in the teat canal had been infected from the existing TCI, the high (> 500 x 10^3 per ml) SCC could possibly be caused by toxins liberated by the bacteria infecting the teat canal and since the BSA precipitation zone is smaller than 8 mm, (5,5 mm) this is a definite indication that the condition is purely a TCI accompanied by slight udder parenchyma damage.

According to the IDF's criteria the condition in Quarter No. 8 is a SCM, a septic SCM according to the quantity of BSA in the milk and a combined TCI and SCM according to the TCSS. All three the abovementioned diagnostic methods point to the same condition, namely SCM.

According to the IDF's criteria the condition in Quarter No. 9 is a LUI, a specific hyperalbumingalactia (SHAG) according to the quantity of BSA in the milk and a TCI according to the TCSS. Toxins (e.g. *S. aureus*) isolated from the teat canal are probably responsible for the BSA precipitation zone of more than 8 mm which points to udder parenchyma damage. The reason for the low SCC of 125 x 10^3 per ml is not very clear but it could be due to leucocytes being in the initial stage of mobilisation. Criteria available for the classification of the different forms of SCM/TCI/udder infections according to the IDF's standards, with due regard to the extent of the BSA precipitation zones and the results of bacterial examination of TCSS, cannot explain the complexity of udder inflammation.

Quarter No. 10 is normal according to the IDF's criteria, the size of the BSA precipitation zone and the bacteriological examination of the TCSS.

Tables 3 & 4 show that examination of the TCSS for diagnosing udder infections indicates fewer quarters being normal than examination of the FMS (IDF's and IDF/BSA's criteria). In other words, the teat canal swab sampling method combined with the cytology of the FMS is diagnostically more sensitive and accurate in determining the health status of quarters. With teat canal swab sampling the bacteria in the teat canal are removed together with keratin and epithelial cells for purposes of bacterial culturing. The greater degree of mechanical friction caused by the swab allows greater diagnostic accuracy than the flushing action during FMS. With the use of the FMS (IDF's and IDF/BSA's criteria) to establish the status of the quarters, false negative results were obtained in 9,1% (IDF's criteria) and 5,9% (IDF/BSA's criteria) of the cases, that is to say when compared with the results of the TCSS (Table 4). The milk obtained from the quarter by FMS is therefore not necessarily a true reflection of the bacteriological status of the quarter as has also been proved by Du Preez[4]. The prevalence of RTI plus ITI, diagnosed according to the IDF/BSA's criteria, is 20% while that of TCI, diagnosed according to the IDF/TCSS's criteria is 26,3%. In 6,3% (Table 4) of the cases the IDF/BSA's criteria were not able to diagnose TCI for the same reason as mentioned above. The prevalence of SCM plus TCI combined with SCM, as diagnosed according to the IDF/TCSS's criteria, is 12,2% in comparison with the 6,3% of SCM cases diagnosed according to the IDF/BSA's criteria and 11,4% according to the IDF's criteria alone (Table 4). It

is clear that cases of quarters classified as normal or ASM according to the IDF's or IDF/BSA's criteria, are in fact cases of TCI which could not be diagnosed without resorting to the TCSS. Some of the TCI have a SCC of more than 500 000 per ml and a combination of the IDF/TCSS's criteria provides a more accurate diagnosis of the udder health status. According to the IDF's and IDF/BSA's criteria respectively, 9,1% and 5,9% more quarters are diagnosed as normal compared with the IDF/TCSS's criteria (Table 4).

According to the FMS (IDF's criteria) up to 20% or more cases of TCI cannot be diagnosed[4]. In the classification of the health status of quarters, neither the IDF's nor IDF/BSA's criteria[10 12 13] take the bacteriological results of the TCSS into consideration. Further research is therefore required to clarify the existence of conditions such as RTI, ITI, UCR, UHAG and SHAG.

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REFERENCES

1. Carter G R 1973 Diagnostic Procedures in Veterinary Microbiology, 2nd edn. Charles C Thomas, Springfield, Illinois
2. Cowan S T, Steel K J 1974 Manual for the Identification of Medical Bacteria. Cambridge University Press
3. Cruikshank R, Duguid J P, Marmion B P, Swain R H A 1975 Medical Microbiology. 12th edn, Volume 2, Churchill Livingston, New York
4. Du Preez J H 1985 Teat canal infections. progress in the control of bovine mastitis. Kieler Milchwirtschaftliche Forschungsberichte 37: 267-273
5. Du Preez J H 1986 The prevalence of teat canal infections in lactating dairy cows as determined from foremilk and teat canal swab samples. Journal of the South African Veterinary Association 57:
6. Du Preez J H, Greeff A S 1985 Comparison of the effect of antibiotic dry cow teat canal and intramammary dry cow therapy of dairy cows on the prevalence of teat canal and intramammary infections at calving. Journal of the South African Veterinary Association 56: 191-194
7. Du Preez J H, van den Heever L W 1980 Teat canal infections in dairy cattle: Therapy, diagnosis and relation to subclinical mastitis. Proceedings of the XI International Congress of Diseases of Cattle, Tel-Aviv: 107-110
8. Giesecke W H 1985 The effect of stress on udder health of dairy cows. Onderstepoort Journal of Veterinary Research 52: 172-193
9. Giesecke W H 1985 The diagnosis and control of bovine mastitis as a means of improving the quality of milk. South African Journal of Dairy Technology 17: 3-21
10. Giesecke W H, Barnard M L 1985 Persistence, deterioration and improvement of various subclinical conditions as major variables of the dynamic balance of udder health status monitored by means of the International Dairy Federation and bovine serum albumin combined criteria. Proceedings of the International Dairy Federation Seminar on Progress in the Control of Bovine Mastitis, Kiel, West Germany (in press)
11. Giesecke W H, Van den Heever L W 1981 Levels of glucose, serum albumin and somatic cells before and during stages of acute clinical mastitis artificially induced in cows by means of human strains of Group B streptococci (GBS) administered intracisternally. Onderstepoort Journal of Veterinary Research 48: 69-75
12. Giesecke W H, Van den Heever L W 1975 Milk hygiene considered in terms of recent observations on definition and diagnosis of bovine mastitis. South African Journal of Dairy Technology 7: 51-61
13. Giesecke W H, Viljoen M W 1974 The diagnosis of subclinical mastitis in lactating cows: A comparison of cytological methods and a monovalent radial immunodiffusion test. The Onderstepoort Journal of Veterinary Research 41: 51-74
14. Giesecke W H, Van den Heever L W, Du Toit I J, Beyer M C E 1973 The diagnosis of bovine mastitis: A critical evaluation of a polyvalent radial immuno-diffusion test and other methods. Onderstepoort Journal of Veterinary Research 40: 59-68
15. Honkanen-Buzalski T 1982 Protein transfer between blood and milk as a marker of bovine mastitis — with special reference to serum albumin, antitrypsin and secretory immunoglobulins. Academic Dissertation. College of Veterinary Medicine, Helsinki, Finland
16. Honkanen-Buzalski T, Sandholm M 1981 Trypsin-inhibitors in mastitic milk and colostrum: correlation between trypsin-inhibitor capacity, bovine serum albumin and somatic cell contents. Journal of Dairy Research 48: 213-223
17. Kästli P 1967 Definition of mastitis. Annual Bulletin of the International Dairy Federation, Part III: 1-5
18. Kitchen B J 1981 Review of the progress of dairy science: Bovine mastitis: milk compositional changes and related diagnostic tests. Journal of Dairy Research 48: 167-188
19. Laboratory methods for use in mastitis work 1981 Document 132. International Dairy Federation Bulletin. Square Vergote 41, 1040 Brussels, Belgium
20. Larson B L, Gillespie D C 1957 Origin of the major specific proteins in milk. Journal of Biological Chemistry 272: 565
21. Lesse J G, Legates J E 1959 Changes in the paper electrophoretic whey protein pattern of cows with acute mastitis. Journal of Dairy Science 42: 698-704
22. Mayer E 1980 Reports and summaries. XI International Congress on Diseases of Cattle. Tel Aviv, Haifa: Israel Association for Buiatrics
23. Menkin A 1956 Biology of inflammation - chemical mediators and cellular injury. Science 124: 527
24. Newbould F H S 1974 Microbial diseases of the mammary gland. In: Larson B L, Smith V R (ed) 2: 269-316
25. Schalm O W 1964 A syllabus on the bovine mammary glands in health and disease. Department of Clinical Pathology, School of Veterinary Medicine, University of California, Davis
26. Schalm O W, Carrol E J, Jain N C 1971 Bovine Mastitis. Lea & Febiger, Philadelphia
27. Sandholm M 1983 Milk antitrypsin assay; A novel method of screening for mastitis. Proceedings of the Third International Symposium. World Association of Veterinary Laboratory Diagnosticians. June 13-15, 1983 Ames, Iowa, USA 2: 571-576
28. Tolle A 1971 A monograph on bovine mastitis. International Dairy Federation Bulletin. Square Vergote 41, 1040 Brussels, Belgium, Part I: 1-23
29. Verhoeff J, Smit J A H 1981 Bovine serum albumin and cell counts in the diagnosis of subclinical udder infection. The Veterinary Quarterly 3: 38-45
30. Wisniowski J (ed) 1981 Proceedings 4th International Symposium on Mastitis Control. Bydgoszoz, Veterinary Institute, Pulawy, Poland

ARTICLE ARTIKEL

THE SCRUB HARE, A RELIABLE INDICATOR OF THE PRESENCE OF *HYALOMMA* TICKS IN THE CAPE PROVINCE

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ABSTRACT: Horak I.G.; MacIvor K.M. De F. **The scrub hare, a reliable indicator of the presence of *Hyalomma* ticks in the Cape Province.** *Journal of the South African Veterinary Association* (1987) **58** No 1, 15-19 (En). Tick Research Unit, Rhodes University, 6140 Grahamstown. Republic of South Africa.

The host preferences of immature and adult *Hyalomma marginatum rufipes, Hyalomma marginatum turanicum* and *Hyalomma truncatum* were studied at various localities in the Cape Province. The immature stages of all 3 ticks preferred scrub hares *(Lepus saxatilis)* as hosts and were recovered from these animals even when few adult ticks were present on large herbivores in the locality. Ground-frequenting birds were also infested, but only with immature *H. marginatum rufipes* and *H. marginatum turanicum.* Some small rodents were also infested but only with the immature stages of *H. truncatum.* The adult ticks preferred Cape mountain zebra *(Equus zebra zebra),* eland *(Taurotragus oryx)* and cattle. The immature stages of *H. marginatum rufipes* were most abundant on scrub hares from February to August. Too few adult ticks of this species were recoved to determine their seasonal abundance. The immature stages of *H. marginatum turanicum* were most abundant on scrub hares during the winter months and the adults were most abundant on scrub hares during the winter months and the adults were most abundant on Cape mountain zebra and eland during summer. Immature *H. truncatum* were most abundant on scrub hares during February and May, while peak adult burdens were found on zebra and eland during February, August, November and December.

INTRODUCTION

Three species of *Hyalomma* ticks, commonly known as bont-legged ticks, are found in South Africa[11]. These are *Hyalomma marginatum rufipes, Hyalomma marginatum turanicum* and *Hyalomma truncatum. H. marginatum rufipes* and *H. truncatum* are indigenous to South Africa, while *H. marginatum turanicum* was presumably introduced into this country[11]. The geographical distributions of these ticks within South Africa are illustrated in Fig. 1-3.

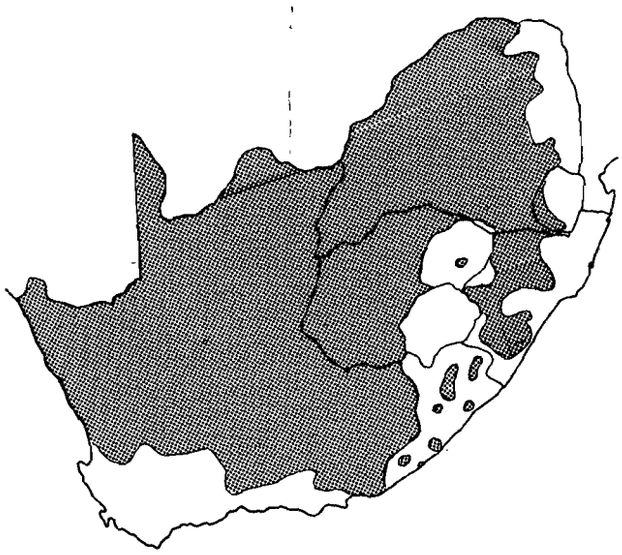

Fig. 1: The distribution of *Hyalomma marginatum rufipes* in the Republic of South Africa[11]

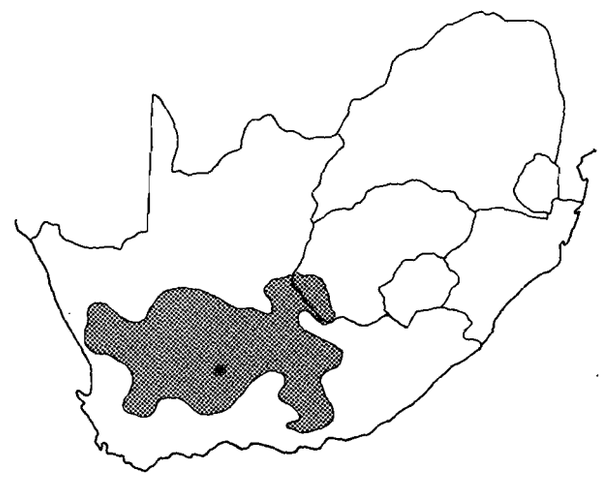

Fig. 2: The distribution of *Hyalomma marginatum turanicum* in the Republic of South Africa[11]

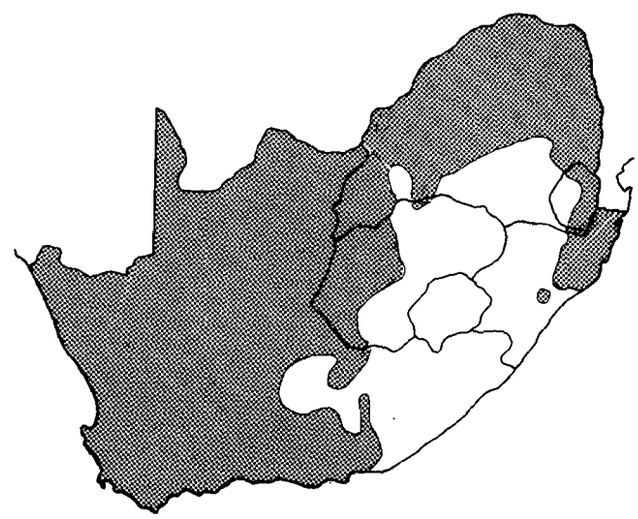

Fig. 3: The distribution of *Hyalomma truncatum* in the Republic of South Africa[11]

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With the exception of the north-eastern Orange Free State, Lesotho, portions of the eastern Cape Province, Ciskei and Transkei and part of coastal Natal, the distribution of one or more of the 3 *Hyalomma* ticks covers the entire country.

These are 2-host ticks and the larvae and nymphs may be found on ground-frequenting birds, certain rat and mice species and on scrub hares *(Lepus saxatilis)* and other lagomorphs[2 4 13 15]. The adults prefer large herbivores such as cattle, eland *(Taurotragus oryx)*, buffalo *(Syncerus caffer)*, horses and zebras as hosts[4 7 9 13].

Hyalomma spp. have been implicated in the transmission of Crimean-Congo haemorrhagic fever virus to man and to animals[3 5], and virus has been collected from both *H. marginatum rufipes* and *H. truncatum*[14]. Certain strains of *H. truncatum* also transmit a toxin causing "sweating sickness" in cattle[12].

Staff of the Tick Research Unit, Rhodes University, have for several years been involved in determining the abundance of ticks on numerous host species in a variety of habitats. The present paper extracts the data for *Hyalomma* spp. from those surveys that were conducted in the Cape Province, with particular attention being paid to scrub hares, which are the preferred hosts of the immature stages of these ticks[2 13]. A similar survey has recently been conducted near Bloemhof in the south-western Transvaal[13] and our results complement those of that survey.

MATERIALS AND METHODS

Scrub hares and other small and large mammals as well as ground-frequenting birds were shot and domestic animals were slaughtered at regular intervals, for periods ranging from 12 to 36 months. These animals were all processed for tick recovery[6 8 10] and the numbers of *Hyalomma* ticks they harboured were determined.

We are unable to differentiate between the immature stages of *H. marginatum rufipes* and *H. marginatum turanicum*. However, in our surveys the adults of these ticks seemed to be geographically separated and we consequently allocated the immature *H. marginatum* we recovered to whichever sub-species of adults that was present.

The surveys were conducted at 4 localities in the Cape Province, namely:

(i) The Bontebok National Park (34°02'S; 20°25'E) 5km south of Swellendam in the south-western Cape Province, situated in a vegetational zone classified as False Macchia and Coastal Rhenosterbosveld[1].
(ii) The farm "Brakhill" (33°33'S; 25°25'E) 25 km north of Uitenhage in the eastern Cape Province. The vegetation in this region is classified as Valley Bushveld[1].
(iii) The farm "Bucklands" (33°05'S; 26°41'E) and the adjoining Andries Vosloo Kudu Reserve (33°07'S; 26°40'E) 30 km north-east of Grahamstown in the eastern Cape Province. The vegetation of this region is also classified as Valley Bushveld[1].
(iv) The Mountain Zebra National Park (32°15'S; 25°41'E) situated approximately 24 km south-west of Cradock in the eastern Karoo. The vegetation of this region is classified as Karroid *Merxmeullera* Mountain Veld replaced by Karoo[1].

RESULTS

The locality, the number and species of animals examined and the total numbers of *Hyalomma* ticks collected are summarized in Tables 1-5.

Table 1: Total numbers of *Hyalomma truncatum* collected from animals in the Bontebok National Park

Host species	No examined	No infested	Total number of ticks recovered				
			Larvae	Nymphs	Males	Females	Total
Striped mouse *Rhabdomys pumilio*	19	0	0	0	0	0	0
Vlei rat *Otomys irroratus*	9	0	0	0	0	0	0
Scrub hare *Lepus saxatilis*	11	3	10	5	0	0	15
Vaal ribbok *Pelea capreolus*	30	0	0	0	0	0	0
Bontebok *Damaliscus dorcas dorcas*	16	1	0	0	1	0	1

Table 2: Total numbers of *Hyalomma truncatum* collected from wild animals and domestic goats on the farm "Brakhill"

Host species	No examined	No infested	Total number of ticks recovered				
			Larvae	Nymphs	Males	Females	Total
Scrub hare *Lepus saxatilis*	48	11	60	248	0	0	308
Grysbok *Raphicerus melanotis*	12	0	0	0	0	0	0
Duiker *Sylvicapra grimmia*	12	1	0	0	1	0	1
Kudu *Tragelaphus strepsiceros*	12	0	0	0	0	0	0
Angora goat *Capra hircus*	24	10	0	0	46	20	66
Boer goat *Capra hircus*	24	5	0	0	6	9	15

Table 3: Total numbers of *Hyalomma marginatum rufipes* collected from wild and domestic animals on the farm "Bucklands"

Host species	No examined	No infested	Total number of ticks recovered				
			Larvae	Nymphs	Males	Females	Total
Helmeted guinea fowl *Numida meleagris*	14	1	1	0	0	0	1
Scrub hare *Lepus saxatilis*	23	4	48	12	0	0	60
Kudu *Tragelaphus strepsiceros*	5	1	0	0	2	0	2
Angora goat *Capra hircus*	24	0	0	0	0	0	0
Dorper sheep *Ovis aries*	24	0	0	0	0	0	0
Cattle *Bos indicus* X *Bos taurus*	22	5	0	0	15	13	28

Table 4: Total numbers of *Hyalomma marginatum rufipes* collected from helmeted guinea fowl, scrub hare and kudu in the Andries Vosloo Kudu Reserve

Host species	No examined	No infested	Total number of ticks recovered				
			Larvae	Nymphs	Males	Females	Total
Helmeted guinea fowl	35	17	38	3	0	0	41
Scrub hare	20	13	146	145	0	0	291
Kudu	12	1	0	0	3	0	3

Table 5: Total numbers of *Hyalomma marginatum turanicum* and *Hyalomma truncatum* recovered from birds and mammals in the Mountain Zebra National Park

Host species	No. examined	*H. marginatum turanicum*						*H. truncatum*					
		No. of hosts infested	Total number of ticks recovered					No. of hosts infested	Total number of ticks recovered				
			Larvae	Nymphs	Males	Females	Total		Larvae	Nymphs	Males	Females	Total
Nicholson's pipit *Anthus similis*	5	3	6	4	0	0	10	0	0	0	0	0	0
Familiar chat *Cercomela familiaris*	5	3	7	12	0	0	19	0	0	0	0	0	0
Spike-heeled lark *Chersomanes albifasciata*	3	0	0	0	0	0	0	0	0	0	0	0	0
Long-billed lark *Certhilauda curvirostris*	9	7	67	17	0	0	84	0	0	0	0	0	0
Thick-billed lark *Galerida magnirostris*	4	3	20	6	0	0	26	0	0	0	0	0	0
Helmeted guinea fowl	16	5	19	3	0	0	22	0	0	0	0	0	0
Pouched mouse *Saccostomus campestris*	3	0	0	0	0	0	0	0	0	0	0	0	0
Striped mouse	30	0	0	0	0	0	0	2	20	4	0	0	24
Namaqua rock mouse *Aethomys namaquensis*	6	0	0	0	0	0	0	2	6	0	0	0	6
Bush Karoo rat *Otomys unisulcatus*	39	0	0	0	0	0	0	2	4	0	0	0	4
Springhaas *Pedetes capensis*	17	4	13	1	0	0	14	0	0	0	0	0	0
Red rock rabbit *Pronolagus rupestris*	28	6	325	0	0	0	325	5	35	8	0	0	43
Scrub hare	26	16	996	1672	0	0	2668	15	273	604	0	0	877
Rock dassie *Procavia capensis*	25	1	1	0	0	0	1	0	0	0	0	0	0
Mountain reedbuck *Redunca fulvorufula*	18	0	0	0	0	0	0	1	0	0	2	0	2
Springbok *Antidorcas marsupialis*	18	1	0	0	2	0	2	1	0	0	2	0	2
Black wildebeest *Connochaetes gnou*	10	1	0	0	1	0	1	1	0	0	5	0	5
Cape mountain zebra *Equus zebra zebra*	14	14	0	0	201	44	245	13	0	0	174	31	205
Eland *Taurotragus oryx*	11	11	0	0	955	191	1146	11	0	0	350	51	401

Bontebok National Park (Table 1)

Although only one of the 46 antelope examined was infested, and that with a single male *H. truncatum*, 3 of the 11 scrub hares harboured immature ticks. As the hares were able to pass freely through the fence of the park it is likely that they acquired their infestations outside the park where cattle frequently grazed.

The farm "Brakhill" (Table 2)

Angora and Boer goats and one grey duiker *(Sylvi-*

capra grimmia) were invested with adult *H. truncatum.* Eleven of the 48 scrub hares were infested with the immature stages of this tick.

The farm "Bucklands" (Table 3)

Few adult *H. marginatum rufipes* were recovered and all of these from the cattle and the kudu *(Tragelaphus strepsiceros).* This is not surprising because the domestic stock on the farm were treated with an acaricide at regular intervals. Nevertheless a number of scrub hares and one of the helmeted guinea fowl *(Numida meleagris)* were infested with the immature stages of this tick. One scrub hare also harboured one larva and 2 nymphs of *H. truncatum.*

The Andries Vosloo Kudu Reserve (Table 4)

The majority of scrub hares and nearly half the helmeted guinea fowl were infested with immature *H. marginatum rufipes,* which was the major species in this reserve. Only 2 kudu were infested with adult *Hyalomma* species. The one harboured 3 male *H. marginatum rufipes* (Table 4) and the other 2 male *H. truncatum.* However, this reserve also harboured eland and buffalo, both of which are good hosts of adult *Hyalomma* species[4 13].

The seasonal abundance of immature *H. marginatum rufipes* (larvae and nymphs combined) on the scrub hares in the Andries Vosloo Kudu Reserve is graphically illustrated in Fig. 4.

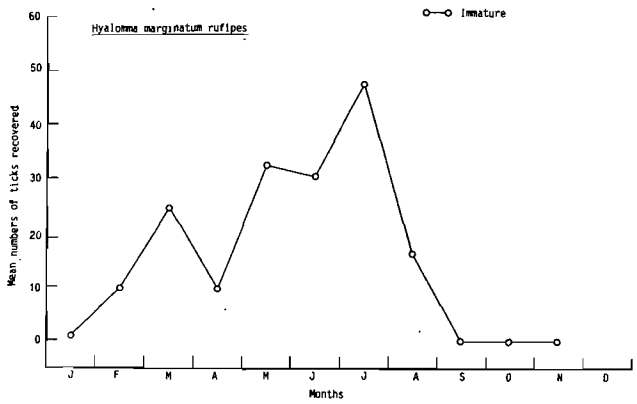

Fig. 4: The seasonal abundance of immature *Hyalomma marginatum rufipes* on scrub hares in the Andries Vosloo Kudu Reserve

The largest numbers of immature ticks were present from February to August.

The Mountain Zebra National Park (Table 5)

Both *H. marginatum turanicum* and *H. truncatum* were present in this park. With the exception of the spike-heeled larks *(Chersomanes albifasciata),* of which only 3 were examined, each of the other ground-frequenting bird species was infested with immature *H. marginatum turanicum.* None of the birds were infested with immature *H. truncatum.* With the exception of the springhaas *(Pedetes capensis),* none of the rodents were infested with the immature stages of *H. marginatum turanicum,* while the striped mice *(Rhabdomys pumilio),* Namaqua rock mice *(Aethomys namaquensis)* and bush Karoo rats *(Otomys unisulcatus)* were infested with immature *H. truncatum.*

The red rock rabbits and the scrub hares were infested with the larvae of both *H. marginatum turanicum* and *H. truncatum,* but whereas the scrub hares also carried large numbers of nymphs, no nymphs of *H. marginatum turanicum* and only 8 nymphs of *H. truncatum* were recovered from the red rock rabbits. Of the larger animals only the Cape mountain zebra *(Equus zebra zebra)* and eland were efficient hosts of adults of both species.

The seasonal abundance of the immature stages (larvae and nymphs combined) of both ticks on scrub hares and that of the adults on mountain zebra and eland is graphically illustrated in Fig. 5 & 6.

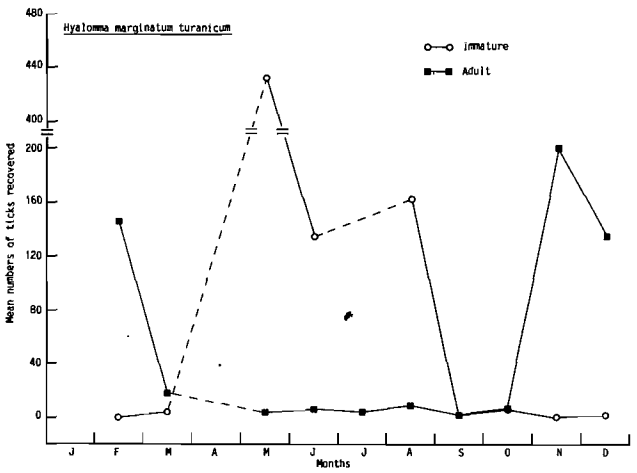

Fig. 5: The seasonal abundance of immature *Hyalomma marginatum turanicum* on scrub hares and adults on eland and Cape mountain zebra in the Mountain Zebra National Park

The largest numbers of immature *H. marginatum turanicum* were present from May to August and the largest numbers of adults from November to February (Fig. 5). The largest numbers of immature *H. trunctum* were present during February and may. Peak adult burdens were recorded during February, August, November and December (Fig. 6).

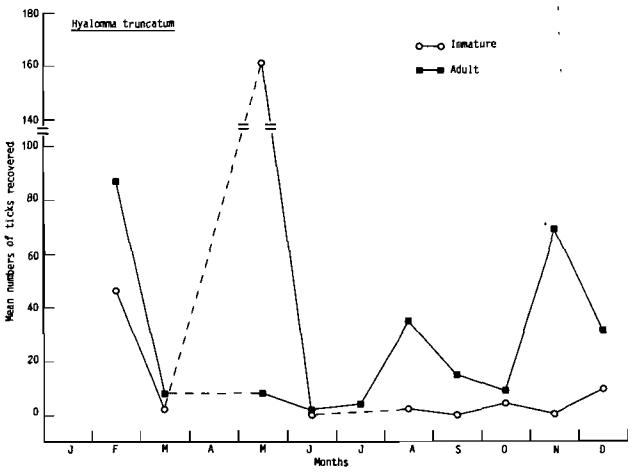

Fig. 6: The seasonal abundance of immature *Hyalomma truncatum* on scrub hares and adults on eland and Cape mountain zebra in the Mountain Zebra National Park

DISCUSSION

As in the survey conducted by Rechav[13] at Bloemhof more nymphs than larvae were recovered from the scrub hares while the converse was true for the guinea fowl. Contrary to Rechav's findings that black wildebeest *(Connochaetes gnou)* are preferred hosts of *H. truncatum*, we found only one of 10 animals to be infested compared with all or nearly all the eland and zebra respectively.

The ground-frequenting birds we examined seem capable of carrying the immature stages of only the 2 sub-species of *H. marginatum* and not those of *H. truncatum*. Conversely certain species of mice and rats seem capable of harbouring only immature *H. truncatum*. The scrub hare, however, is an efficient host of the immature stages of *H. truncatum* and of both sub-species of *H. marginatum*. Scrub hares also appear to be reliable indicators of the presence of *Hyalomma* species within a region, for even when only a few adult ticks were recovered from the larger hosts, a fairly large proportion of scrub hares was often infested with the immature stages.

Although the ground-frequenting birds and smaller rodents harboured comparatively few immature *Hyalomma* compared with the scrub hares, the birds' and the rodents' role in the maintenance of these ticks could be important in view of their own large numbers.

We have examined many sheep, goats, cattle, large and small antelope, zebras and domestic and wild carnivores and with only 2 exceptions, namely one larva on a sheep and one larva on a caracal *(Felis caracal)*, we have not recovered immature *Hyalomma* from any of these hosts. We have, however, recovered the adults in fairly large numbers from the larger mammal species such as cattle, horses, mountain zebra and eland. However, as seen in the Mountain Zebra National Park and as reported by Rechav[13], smaller antelope such as mountain reedbuck *(Redunca fulvorufula)*, springbok *(Antidorcas marsupialis)*, blesbok *(Damaliscus dorcas phillipsi)* and black wildebeest may also be infested. Domestic goats can also serve as hosts, as determined on the farm "Brakhill".

It would seem to us that these rather specific host-preferences dictate the low incidence of Crimean-Congo haemorrhagic fever due to tick-bite in humans in this country, despite the widespread distribution of the ticks and virus within the Republic of South Africa[14].

In addition to the precautions to be taken to prevent infection with the virus suggested by Rechav[13] we would like to advise against the practice of many stockmen of removing engorging or engorged female ticks from stock and bursting them between the fingers or nails. Like Rechav[13] we do not advocate the destruction of wildlife in order to reduce the tick population as the presence of a large variety of alternative hosts makes this a futile operation.

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REFERENCES

1. Acocks J P H 1975 Veld types of South Africa with accompanying veld type map. Memoirs of the Botanical Survey of South Africa No 40, 128 pp
2. Clifford C M, Flux J E C, Hoogstraal H 1976 Seasonal and regional abundance of ticks (Ixodidae) on hares (Leporidae) in Kenya. Journal of Medical Entomology 13:40-47
3. Darwish M, Hoogstraal H 1981 Arboviruses infecting humans and lower animals in Egypt: a review of thirty years of research. Journal of the Egyptian Public Health Association 56: 1-112
4. Hoogstraal H 1956 African Ixodoidea. I. Ticks of the Sudan (with special reference to Equatoria Province and with preliminary reviews of the genera *Boophilus, Margaropus,* and *Hyalomma).* Department of the Navy Bureau of Medicine and Surgery, Washington DC 1101pp
5. Hoogstraal H 1979 The epidemiology of tick-borne Crimean-Congo haemorrhagic fever in Asia, Europe and Africa. Journal of Medical Entomology 15: 307-417
6. Horak I G, Meltzer D G A, De Vos V 1982 Helminth and arthropod parasites of springbok, *Antidorcas marsupialis,* in the Transvaal and western Cape Province. Onderstepoort Journal of Veterinary Research 49: 7-10
7. Horak I G 1982 Parasites of domestic and wild animals in South Africa. XV. The seasonal prevalence of ectoparasites on impala and cattle in the northern Transvaal. Onderstepoort Journal of Veterinary Research 49:85-93
8. Horak I G, Williams E J 1986 Parasites of domestic and wild animals in South Africa. XVIII. The crowned guinea fowl *(Numida meleagris)*, an important host of immature ixodid ticks. Onderstepoort Journal of Veterinary Research 53: 119-122
9. Horak I G, Knight M M, De Vos V 1986 Parasites of domestic and wild animals in South Africa. XX. Arthropod parasites of the Cape mountain zebra *Equus zebra zebra.* Onderstepoort Journal of Veterinary Research 53: 127-132
10. Horak I G, Sheppey K, Knight M M, Beuthin C L 1986 Parasites of domestic and wild animals in South Africa. XXI. Arthropod parasites of vaal ribbok, bontebok and scrub hares in the western Cape Province. Onderstepoort Journal of Veterinary Research 53: 187-197
11. Howell C J, Walker Jane B, Neville E M 1978 Ticks, mites and insects infesting domestic animals in South Africa. Part 1. Descriptions and biology. Department of Agricultural Technical Services, Republic of South Africa, Science bulletin No 393, v + 69pp
12. Neitz W O 1959 Sweating sickness: the present state of our knowledge. Onderstepoort Journal of Veterinary Research 28: 3-38
13. Rechav Y 1986 Seasonal activity and hosts of the vectors of Crimean-Congo haemorrhagic fever in South Africa. South African Medical Journal 69: 364-368
14. Swanepoel R, Struthers J K, Shepherd A J, McGillivray G M, Nel M J, Jupp P G 1983 Crimean-Congo haemorrhagic fever in South Africa. American Journal of Tropical Medicine and Hygiene 32: 1407-1415
15. Theiler G 1962 The Ixodoidea parasites of vertebrates in Africa south of the Sahara (Ethiopian Region). Report to the Director of Veterinary Services, Onderstepoort 260pp

ABSTRACT **SAMEVATTING**

THE CLINICAL PATHOLOGY OF SWEATING SICKNESS IN CATTLE

Experimentally-induced cases of sweating sickness in calves were used in an effort to correlate the blood chemistry with some of the known pathological changes. Results showed that the "sweating" associated with necrotic dermatitis did not alter blood electrolyte levels. Laboratory evidence of a disseminated intravascular coagulopathy was found which correlated with the microthrombi described in cases of sweating sickness. A high blood cortisol level was found in one of the animals that died from the disease and could possibly be used as a prognostic indicator in clinical cases. Recommendations are made with regard to the supportive treatment based on the clinical pathological findings. (Van Amstel, S.R., Reyers, F., Oberem, P.T. & Matthee, O., 1987. Further studies on the clinical pathology of sweating sickness in cattle. *Onderstepoort Journal of Veterinary Research,* 54, 45-58 (1987).)

ABSTRACT **SAMEVATTING**

SOME WILD HOSTS OF THE KAROO PARALYSIS TICK

A variety of mammal and bird species in the Mountain Zebra National Park, eastern Cape Province, were examined for *Ixodes rubicundus*. In addition, caracal *(Felis caracal)* from the park and its vicinity as well as from the Graaff-Reinet region of the Karoo were examined. Amongst the animals collected, the red rock rabbit *(Pronolagus rupestris)* was a preferred host of the immature stages of the tick. Caracal were moderately good hosts of these stages and, with mountain reedbuck *(Redunca fulvorufula)* and eland *(Taurotragus oryx)*, are the preferred hosts of adult ticks.

The seasonal abundance of the various parasitic life stages was also determined. (Horak, I.G., Moolman, L.C. & Fourie, L.J., 1987. Some wild hosts of the Karoo paralysis tick, *Ixodes rubicundus* Neumann, 1904 (Acari: Ixodae). *Onderstepoort Journal of Veterinary Research,* 54, 49-51 (1987).)

ABSTRACT **SAMEVATTING**

SUSPECTED LIPOFUSCIN STORAGE DISEASE OF SHEEP ASSOCIATED WITH INGESTION OF THE PLANT, *TRACHYANDRA DIVARICATA*

Paresis afflicted 85 our of a flock of 770 young Merino ewes kept on old wheat lands in the western Cape during a period of drought. Many of the paretic ewes died. The vegetation was sparse and was dominated by *Trachyandra divaricata*. At necropsy, yellowish-brown discoloration of the grey matter throughout the brain and spinal cord and mild brown discoloration of the liver, renal cortex and lymph nodes were consistently seen. Light microscopical examination revealed abundant, yellowish-brown pigment granules in the cytoplasm of most of the larger neurons. Similar pigment also occurred in some non-nervous tissues. Shrinkage and loss of a few randomly scattered axons were observed in the white matter of the spinal cord in 2 sheep. Histochemical and ultrastructural features of the pigment were consistent with those of lipofuscin.

T. divaricata failed to reproduce the condition when dosed to a sheep, but the paresis and pigmentation shown to be caused by the closely related plant, *T. laxa,* are strikingly similar. *Trachyandra* poisoning appears to be the first documented example in farm animals of an acquired lipofuscin storage disease involving nervous and non-nervous tissues for which a specific plant has been causally implicated. (Newsholme, S.J., Schneider, D.J. & Reid, Clare, 1985. A suspected lipofuscin storage disease associated with ingestion of the plant, *Trachyandra divaricata* (Jacq.) Kunth. *Onderstepoort Journal of Veterinary Research,* 52, 87-92 (1985).)

ARTICLE ARTIKEL

THE EFFECT OF LATERAL AND DORSAL RECUMBENCY ON CARDIOPULMONARY FUNCTION IN THE ANAESTHETISED HORSE

G.F. STEGMANN* and A. LITTLEJOHN**

ABSTRACT: Stegmann G.F.; Littlejohn A. **The effect of lateral and dorsal recumbency on cardiopulmonary function in the anaesthetised horse.** *Journal of the South African Veterinary Association* (1987) **58** No 1, 21-27 (En) Department of Surgery, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, 0110 Onderstepoort, Republic of South Africa.

The effect of lateral and dorsal recumbency on cardiopulmonary function in six anaesthetised horses were compared. Cardiac output/kg, stroke volume/kg, alveolar ventilation, venous admixture, pulmonary shunt and blood-gas values were determined. From lateral to dorsal recumbency cardiac output/kg decreased non-significantly. A significant increase in pulmonary shunt occurred. A positive correlation between body mass and pulmonary shunt in dorsal recumbency was found. Alveolar ventilation increased significantly in dorsal recumbency when breathing air compared to oxygen.

Key words: Lateral recumbency, dorsal recumbency, pulmonary shunt, alveolar ventilation, equine anaesthesia.

INTRODUCTION

Induction of anaesthesia leads to a change in body position from standing to either lateral or dorsal recumbency. Negative changes in cardiopulmonary function were ascribed either to the abnormal body position, the effect of the anaesthetic drugs or a combination of both factors. Conscious (awake) laterally recumbent horses were able to maintain normal arterial blood gas values[7 14]. A small increase in pulmonary shunt occurred in the conscious (awake) laterally recumbent horse[7]. During anaesthesia the partial pressure of oxygen in arterial blood (Pa_{O_2}) decreased, the partial pressure of carbon dioxide in arterial blood (Pa_{CO_2}) and the alveolar-arterial oxygen partialpressure difference ($P(A\text{-}a)_{O_2}$) increased after induc-tion of anaesthesia and lateral recumbency[6]. Pulmonary shunt is greatly increased from the standing (awake) horse to the anaesthetised, laterally recumbent horse[5 9]. Administering halothane with high concentrations of oxygen increased Pa_{O_2} and $P(A\text{-}a)_{O_2}$ difference. Stolk[19]showed a decrease pulmonary blood flow to the dependent lung in the conscious and anaesthetised laterally recumbent horse. Changing from lateral to dorsal recumbency during anaesthesia further decreased Pa_{O_2},and increased Pa_{CO_2} and $P(A\text{-}a)_{O_2}$ differences. McDonellet al.[10] show-ed with X-rays that compression of the dependent lung occurs in lateral recumbency and that the functional residual capacity (FRC) is greatly reduced from the standing to the laterally recumbent horse. FRC is however less reduced in horses starved for 18 hours (reducing abdominal contents) and also in horses ventilating on room air during anaesthesia in comparison to horses breathing halothane-oxygen mixtures. Changing from lateral to dorsal recumbency improved FRC but pulmonary shunt was increased[9].

In this investigation the effects of body mass, and the effects of lateral and dorsal recumbency on cardiopulmonary function in the anaesthetised horse were investigated.

MATERIALS AND METHODS

Experimental horses

The horses were identified by their names. Five of the six horses were Thoroughbreds and in the age group 4 to 6 years, Polisieperd was a Thoroughbred X, seven years old.

On clinical examination no abnormalities of the pulmonary and cardiovascular systems could be detected. The horses were removed from training as result of chronic lamenesses.

Experimental preparation

To facilitate the collection of arterial blood samples from the arteria carotis communis, the arteries were sub-cutaneously relocated in the jugular groove[1].

Anaesthetic technique

The horses were starved from food for 24 hours before commencing the experimentation. Water was given ad lib up. and till induction of anaesthesia.

The horses were premedicated with propionyl promazine (Combelen, Bayer) at a dose of 0,02 mg/kg bodymass, 60 minutes before induction or the start of sample collection in the conscious horses.

Anaesthesia was induced with the intra-venous infusion of a 10% solution of glyceryl guaiacolate ether (GGE) at a dose of 55-110 mg/kg. When the animals became unstable the GGE infusion was stopped and thiopentone sodium (Intraval, Maybaker) injected intra-venously at a dose of 3g/500 kg body mass.

Anaesthesia was maintained on a closed circle system with halothane. Anaesthetic plane was maintained at a level where the palpebral reflex was just absent. The carrier gas was either oxygen or medical air obtained from compressed gas cylinders. When the carrier gas was changed from air to oxygen, a high flow rate (15 ℓ/min) was maintained for 10 minutes to allow for nitrogen washout from the animal and the anaesthetic circuit.

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Blood and gas sampling

The following blood and gas samples were collected:
1 arterial blood (a)
2 mixed venous blood ($\bar{V}$)
3 mixed expired gas ($\bar{E}$)
4 end-tidal gas (ET)

For the collection of arterial blood, a 18G teflon catheter was introduced per cutaneously in the relocated carotid artery. Mixed venous blood (v) was collected after the placing of a polyethylene tube in the pulmonary artery via per cutaneous puncture of the vena jugularis. Blood samples were collected anaerobically in 5 ml heparinised plastic syringes, stored in ice water. Analysis was done within 30 min. after collection, with a ABL3 (Radiometer).

Mixed expired gas was collected from the horses by placing a soft plastic facemask constructed from a 5ℓ household detergent bottle over the mouth and nostrils. The edges were sealed off by placing wet cotton wool in the intermandibular space and wrapping latex sheeting around the junction between mask and head. One-way-valves in a special constructed perspex casing were used to direct inspired gas to the lungs and expired gas to a 200ℓ latex metereological balloon. The volume of expired gas was determined with a Collins 600ℓ spirometer.

End-tidal gas was collected by placing a 18G hypodermic needle per cutaneously into the trachea at the base of the neck. This was attached to a 50 ml glass syringe.

The collection of blood and gas samples took place simultaneously and continuously over a period of three minutes.

Experimental protocol

The experiments were done in three stages. During the first stage samples were taken in the standing conscious horse. First breathing room air, thereafter breathing oxygen for a period of 10 minutes. The oxygen was administered with a Hudson demand valve.

In the second stage anaesthesia was induced and maintained for 10 minutes on a halothane-medical air mixture. The horses were kept in lateral recumbency. After the samples on air had been collected, the carrier gas was changed to oxygen.

The horses were allowed to breath the oxygen mixture for 15 min. before samples were collected. Thereafter the horses were recovered from anaesthesia.

To allow for full recovery from the anaesthetic and possible pathology in the dependent lung, the third stage was executed only after at least four weeks of rest from the previous anaesthetic. In the third stage the same sequence of events were followed as in the second stage, except that the horses were kept in dorsal recumbency.

Calculations

All ventilation volumes were corrected to BTPS and gaseous exchange volumes to STPD.

Cardiovascular parameters

1. Cardiac output was calculated using the Fick principle.

$$\text{Cardiac output} = \frac{\dot{V}O_2}{Ca_{O_2} - C\bar{v}_{O_2}}$$

Ca_{O_2} = Oxygen content of arterial blood

CV_{O_2} = Oxygen content of mixed venous blood

V_{O_2} = Minute volume (STPD) x O_2 fraction

2. The minute volume (STPD) was calculated with the aid of the 'Blood Gas Calculator'[16], deriving the values from the spirometer values at ATPS

ATPS = Ambient temperature, pressure, saturated
BTPS = Body temperature, pressure, saturated
STPD = Standard temperature, pressure, dry

3. O_2 fraction was calculated with the 'Blood Gas Calculator'[16] from the following values:
3.1 V_T (BTPS)
3.2 Oxygen concentration in expired gas

$$= \frac{PE_{O_2}}{P_B - 50} \times \frac{100}{1}$$

3.3 Carbon dioxide concentration in expired gas

$$= \frac{PE_{CO_2}}{P_B - 50} \times \frac{100}{1}$$

P_B = barometric pressure
V_T = tidal volume
$P\bar{E}_{O_2}$ = partial pressure of oxygen in mixed expired gas

4. Cardiac output/kg $= \frac{\text{cardiac output}}{\text{bodymass}}$ (ml/min/kg)

5. Stroke volume $= \frac{\text{cardiac output}}{\text{cardiac rate}}$ (ml/stroke)

6. Stroke volume/kg $= \frac{\text{stroke volume}}{\text{bodymass}}$ (ml/stroke/kg)

7. Pulmonary shunt was calculated by subtracting the calculated percentage venous admixture when breathing oxygen from the percentage venous admixture when breathing air.
8. Venous admixture was calculated as a percentage of the cardiac output according to the formula used by Nunn[11]

$$8.1 \text{ Venous admixture} = \frac{C\acute{c}_{O_2} - Ca_{O_2}}{C\acute{c}_{O_2} - Cv_{O_2}}$$

$C\acute{c}_{O_2}$ = oxygen content of pulmonary end-capillary blood

The value of $C\acute{c}_{O_2}$ was replaced with the calculated value from the formula by Nunn[12].

$$8.2 \; Pa_{O_2} = PI_{O_2} - PA_{CO_2} \frac{PI_{O_2} - P\bar{E}_{O_2}}{P\bar{E}_{CO_2}}$$

PA_{O_2} = partial pressure of oxygen in the alveolus

PI_{O_2} = partial pressure of oxygen in the inspired gas

PA_{CO_2} = partial pressure of carbon dioxide in the alveolus

$P\bar{E}_{CO_2}$ = partial pressure of carbon dioxide in mixed expired gas

8.3 Oxygen saturation was calculated with the 'Blood Gas Calculator'[16]

8.4 Oxygen content was calculated with the formula: oxygen content =

$$\frac{\text{hemoglobin (g/dl) x 1 33 x oxygen saturation}}{100}$$

Pulmonary parameters

9. Alveolar deadspace was calculated according to the formula of Severinghaus[15] as a percentage of the tidal volume:

9.1 $$V_D\,(alv) = \frac{Pa_{CO_2} - PET_{CO_2}}{Pa_{CO_2}}$$

9.2 $$\%\,V_D\,(alv) = \frac{V_D\,(alv)}{V_T - V_D\,(anat)} \times \frac{100}{1}$$

PET_{CO_2} = partial pressure of carbon dioxide in end-tidal gas

Pa_{CO_2} = partial pressure of carbon dioxide in arterial blood

V_D (anat) = anatomical deadspace

10.1 Alveolar ventilation (V_A) was calculated according to the formula of Nunn[12].

Alveolar ventilation = ($V_T - V_D$ (phys)) x respiration rate

10.2 Physological deadspace was calculated according to the Bohr formula[12]:

$$V_D\,(phys) = \frac{Pa_{CO_2} - P\bar{E}_{CO_2}}{Pa_{CO_2}}$$

Student's t test was used to compare results statistically

RESULTS

The mean values for the blood gases and calculated cardiopulmonary parameters are tabulated in Table 1 for the standing sedated horse, anaesthetised horse during lateral and dorsal recumbency, breathing either oxygen or air.

The mean difference and statistical significance for blood gases and cardiopulmonary parameters between the standing conscious and anaesthetised laterally recumbent; standing conscious and anaesthetised dorsally recumbent; anaesthetised laterally and dorsally recumbent horse, is tabulated in Table 2.

The correlation coefficient between the cardiopulmonary parameters for the standing, laterally and dorsally recumbent horse and its statistical meaning is given in Table 3.

The mean difference and statistical significance in alveolar ventilation between oxygen and air breathing is tabulated in Table 4.

The difference between obtained results were regarded as statistical significant if $P < 0,05$.

Table 1: Mean of cardiopulmonary parameters in the standing, laterally and dorsally recumbent horse

Parameter	Gas	Standing		Lateral		Dorsal	
		Mean (n)	SD	Mean (n)	SD	Mean (n)	SD
CO/kg	AIR	74,210 (6)	25,40	31,23 (6)	8,14	25,80 (6)	2,80
	OXYG	59,39 (6)	11,70	25,22 (6)	12,39	24,20 (6)	7.40
SV/kg	AIR	2.13 (6)	0,75	0,91 (6)	0,26	0,66 (6)	0,18
	OXYG	2,40 (6)	0,42	0,92 (6)	0,60	0,58 (6)	0,18
VA	AIR	6,50 (5)	2,90	42,60 (6)	16,70	43,53 (6)	15,76
	OXYG	8,80 (5)	2,10	22,50 (6)	18,80	15,92 (6)	3,00
SHUNT		−2,30 (5)	3,60	20,10 (6)	8,00	27,60 (6)	15,50
ALV VENT	AIR	21,66 (3)	1,81	9,98 (6)	3,34	19,26 (6)	9,06
	OXYG	17,00 (3)	0,94	8,23 (6)	3,65	9,83 (6)	8,98
VD (alv)	AIR	8,13 (5)	6,80	19,37 (6)	10,50	12,31 (6)	4,30
	OXYG	11,39 (5)	9,60	14,68 (6)	5,80	23,03 (6)	5,90
$P\,(A\text{-}a)_{O_2}$	AIR	1,33 (6)	1,30	4,50 (6)	0,40	4,90 (6)	1,90
	OXYG	16,20 (6)	4,30	35,60 (6)	17,10	42,10 (6)	6,80
Pa_{CO_2}	AIR	5,60 (6)	0,30	7,80 (6)	1,20	8,20 (6)	1,10
	OXYG	6,10 (6)	0,40	8,60 (6)	1,30	10,20 (6)	1,60
Pa_{O_2}	AIR	9,70 (6)	0,60	6,80 (6)	0,60	6,30 (6)	1,40
	OXYG	57,60 (6)	4,40	39,20 (6)	4,00	27,40 (6)	6,10

* Explanation of abbreviations used in Tables 1-4

CO/kg = cardiac output/kg (ml/min/kg)
SV/kg = stroke volume/kg (ml/stroke/kg)
VA = venous admixture (% of cardiac output)
SHUNT = pulmonary shunt (% of cardiac output)
ALV VENT = alveolar ventilation (l/minute)
VD (alv) = alveolar dead space (% of tidal volume)
Pa_{CO_2} = partial pressure of carbon dioxide in arteral blood (kPa)
Pa_{O_2} = partial pressure of oxygen in arterial blood (kPa)
NS = nog significant
(n) = number of animals used in the calculation of mean
OXYG = Oxygen
SD = Standard deviation

Cardiac output/kg and stroke volume/kg

Statistical significant decreases occurred from the standing conscious horse to the anaesthetised laterally and dorsally recumbent horses. See Table 2. A significant negative correlation (r = −0,86 Table 3) was found between the cardiac output parameters (cardiac output/kg and stroke volume/kg,) and pulmonary shunt in lateral recumbency (Table 3).

Table 2: Mean difference and statistical significance for the cardiopulmonary parameters in the horse

		Standing to lateral			Standing to dorsal			Lateral to dorsal		
Parameter	Gas	Mean (n)	T	P	Mean (n)	T	P	Mean (n)	T	P
CO/kg	AIR	−43,00 (6)	5,75	0,01	−48,40 (6)	3,11	NS	− 5,40 (6)	1,03	NS
	OXYG	−34,20 (6)	5,21	0,02	−35,20 (6)	5,65	0,01	− 1,00 (6)	0,17	NS
SV/kg	AIR	− 2,13 (6)	6,99	0,01	− 0,78 (6)	3,12	NS	− 0,66 (6)	1,25	NS
	OXYG	− 2,42 (6)	4,97	0,01	− 0,83 (6)	4,97	0,02	− 0,58 (6)	0,89	NS
VA	AIR	+36,15 (5)	4,02	0,02	36,99 (5)	8,34	0,01	0,84 (6)	0,10	NS
	OXYG	–	–	–	–	–	–	6,61 (6)	0,80	NS
SHUNT		–	–	–	–	–	–	+13,17 (6)	3,44	0,05
ALV VENT	AIR	−11,68 (3)	4,93	NS	− 2,40 (3)	0,40	NS	+ 9,28 (6)	1,90	NS
	OXYG	− 8,77 (3)	3,60	NS	− 7,17 (3)	1,22	NS	+ 1,60 (6)	0,40	NS
VD (ALV)	AIR	+11,24 (5)	2,71	NS	+ 4,18 (5)	2,40	NS	− 7,06 (6)	1,50	NS
	OXYG	− 3,29 (5)	4,01	0,05	+11,64 (5)	1,56	NS	+ 8,35 (6)	2,23	NS
P (A-A) 02	AIR	+ 1,20 (6)	5,38	0,01	+ 3,60 (6)	3,00	0,05	+ 0,40 (6)	0,50	NS
	OXYG	+19,40 (6)	1,97	NS	+25,90 (6)	9,66	0,001	+ 6,50 (6)	0,70	NS
Pa_{CO_2}	AIR	+ 2,20 (6)	2,97	0,05	+ 2,60 (6)	4,40	0,02	+ 0,40 (6)	0,30	NS
	OXYG	+ 2,50 (6)	3,65	0,05	4,00 (6)	6,60	0,01	+ 1,60 (6)	2,72	NS
Pa_{O_2}	AIR	− 2,80 (6)	2,10	NS	− 3,30 (6)	9,33	0,001	− 0,50 (6)	0,06	NS
	OXYG	+18,40 (6)	2,25	NS	−30,20 (6)	14,66	0,001	−11,80 (6)	2,07	NS

CO/kg = cardiac output/kg (ml/min/kg)
SV/kg = stroke volume/kg (ml/stroke/kg)
VA = venous admixture (% of cardiac output)
SHUNT = pulmonary shunt (% of cardiac output)
ALV VENT = alveolar ventilation (l/minute)
VD (alv) = alveolar dead space (% of tidal volume)
Pa_{CO_2} = partial pressure of carbon dioxide in arteral blood (kPa)
Pa_{O_2} = partial pressure of oxygen in arterial blood (kPa)
NS = nog significant
(n) = number of animals used in the calculation of mean
OXYG = Oxygen
SD = Standard deviation

Table 3: Correlation between cardiopulmonary parameters for the standing, laterally and dorsally recumbent horse

		Standing		Lateral Recumbency		Dorsal Recumbency	
		Correlation Coefficient	T P<	Correlation Coefficient	T P<	Correlation Coefficient	T P<
CO/kg to Shunt		NS	NS	−0,86	3,37 0,02	−0,01	NS
SV/kg to Shunt		NS	NS	−0,86	3,37 0,02	−0,22	NS
VD (Alv) to Pa_{O_2}	AIR	−0,50	NS	−0,01	NS	−0,77	NS
	OXYG	−0,89	3,38 0,02	−0,52	NS	0;74	NS
Body Mass to Ven Admix	AIR	−0,53	1,08	−0,08	NS	0,56	NS
	OXYG	−0,83	2,58 0,05	−0,21	NS	0,08	2,67 0,05
Body Mass to Shunt		NS	NS	−0,66	NS	0,08	2,67 0,05

* Explanation of abbreviations used in Tables 1-4

CO/kg = cardiac output/kg (ml/min/kg)
SV/kg = stroke volume/kg (ml/stroke/kg)
VA = venous admixture (% of cardiac output)
SHUNT = pulmonary shunt (% of cardiac output)
ALV VENT = alveolar ventilation (l/minute)
VD (alv) = alveolar dead space (% of tidal volume)
Pa_{CO_2} = partial pressure of carbon dioxide in arteral blood (kPa)
Pa_{O_2} = partial pressure of oxygen in arterial blood (kPa)
NS = nog significant
(n) = number of animals used in the calculation of mean
OXYG = Oxygen
SD = Standard deviation

Venous admixture and pulmonary shunt

In the standing sedated horse venous admixture increased when the horses breathed oxygen compared to when breathing air. From standing to lateral recumbency and to dorsal recumbency a statistical significant increase (respectively P < 0,02 and P < 0,01) occurred (Table 2).

For pulmonary shunt, negative values were calculated in the standing position as result of the increase in

Table 4: Mean difference and statistical significance in alveolar ventilation between oxygen and air breathing horses

Body position	Standing	Lateral	Dorsal
Mean difference	4,67	1,75	9,43
T value	3,45	0,78	4,17
P value	NS	NS	0,01

* Explanation of abbreviations used in Tables 1-4

CO/kg = cardiac output/kg (ml/min/kg)
SV/kg = stroke volume/kg (ml/stroke/kg)
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venous admixture when breathing oxygen. Shunt increased significantly from lateral to dorsal recumbency. (Table 2).

A positive correlation (r = 0,8) that is statistical significant (P < 0,05) was found between bodymass and pulmonary shunt in the dorsally recumbent horse. In the standing horse a negative correlation of 0,83 was found between bodymass and venous admixture (Table 3).

P $(A\text{-}a)_{O_2}$ differences

From standing to dorsal recumbency a significant increase occurred when breathing air (P < 0,05; Table 2) and oxygen (P < 0,001; Table 2).

Pa_{O_2} and Pa_{CO_2}

Pa_{CO_2} increased significantly from standing to lateral recumbency when breathing either air (P < 0,05, Table 2) or oxygen (P < 0,05; Table 2).

From standing to dorsal recumbency a highly significant decrease in Pa_{O_2} occurred when breathing either air or oxygen (P < 0,001; Table 2). Pa_{CO_2} increased significantly when breathing either air or oxygen (P < 0,02 and P < 0,01, respectively) (Table 2).

Alveolar deadspace

From standing to lateral recumbency a significant increase occurred when breathing oxygen (P < 0,05, Table 2). When breathing air only a small increase occurred (Table 2). A significant negative correlation between V_D (alv) and Pa_{O_2} (r = −0,89; Table 3) occurred in the standing position, but could not be found for the laterally and dorsally recumbent horse.

Alveolar ventilation

In dorsal recumbency a significant decrease was noted when breathing oxygen compared to air (Table 4).

DISCUSSION

Calculated values for stroke volume/kg in the standing conscious horse correlate with values obtained by other authors using the dye dilution technique[4 8]. Values obtained with the dye dilution technique for the anaesthetised laterally recumbent horse[6 8] were higher than values obtained in this investigation. No previous values for dorsal recumbency were available for comparison.

The negative inotropic effects of halothane on cardiac muscle were reflected in the decreases that occurred in cardiac output/kg and stroke volume/kg after induction of anaesthesia. Cardiac output decreased, although not statistically significant from the laterally recumbent to the dorsally recumbent animal during anaesthesia. The anaesthetic plane was the same in the two positions. The decrease observed in the dorsal position could be attributed to decreased venous return leading to a decrease in stroke volume and cardiac output. The ascending flow of blood in the venous system towards the heart during dorsal recumbency, and a decreased peripheral resistance as result of a higher Pa_{Co_2} may contribute towards a decreased venous return.

The partial pressure of oxygen in arterial blood (Pa_{O_2}) is influenced by:
— inspired oxygen concentration (Fl_{O_2})
— alveolar ventilation
— ventilation-perfusion mismatch in the lungs ($\dot{V}/\dot{Q}$)
— right to left shunting of blood
— diffusion between alveolus and alveolar capillaries
— decreased cardiac output ($\downarrow P\bar{v}_{O_2}$)

It is known that halothane depress respiratory function. In lateral recumbency alveolar ventilation and Pa_{O_2} values are decreased and Pa_{CO_2} values increased during anaesthesia. P $(A\text{-}a)_{O_2}$ differences are also increased, although only significantly in the horses breathing air during this investigation.

The effect of dorsal recumbency compared to the standing (awake) horse resulted in a significant increase in Pa_{CO_2} and a highly significant decrease in Pa_{O_2}. For the halothane-oxygen anaesthetised laterally recumbent horse, values for Pa_{O_2} and Pa_{CO_2} were recorded ranging from 12,5 to 42,6 kPa for O_2 and 6,8 to 8,8 kPa for CO_2 by Hall et al.[6] and Steffey et al.[17]. These values have a wider range than the recorded values during this investigation for Pa_{O_2}. Pa_{CO_2} values correlate with values recorded by Steffey et al.[17]. In dorsal recumbency Steffey et al.[17] recorded Pa_{Co_2} values lower than values recorded in this investigation. During this investigation

the highest value were recorded with the horses breathing a halothane-oxygen mixture in dorsal recumbency.

After induction of anaesthesia alveolar ventilation decreased non-significantly in the laterally recumbent horse. Associated with the decreased ventilation the expected increase in Pa_{CO_2} and the decrease in Pa_{O_2} occurred. There was no significant difference in ventilation between the oxygen and air breathing animal. In dorsal recumbency when the horses breathed air, a significant increase in ventilation occurred in comparison to breathing oxygen.

Associated with the increase in ventilation, Pa_{O_2} and Pa_{CO_2} decreased in comparison to the dorsally recumbent horse breathing oxygen. The anaesthetic plane was identical for dorsal and lateral recumbency and could therefore not be responsible for the increased ventilation. Pa_{O_2} did decrease but the difference is so small that the difference in respiratory stimulation could not be responsible for this significant increase in ventilation.

Alveolar deadspace increased significantly when breathing oxygen in the laterally recumbent horse as a result of the decreased cardiac output in this position. The changes seen in dorsal recumbency were small and insignificant possibly due to the change in the relative position of the heart. In the standing animal the heart lies below the lungs and in the dorsally recumbent horse it lies above the lungs, compensating for the decrease in cardiac output during anaesthesia[18].

Calculated values for pulmonary shunt by other authors vary from 21 to 51% in halothane — oxygen anaesthetised laterally recumbent horses[5,9]. The lower values correlate with calculations in this investigation. For the standing horse values for venous admixture increased from air to oxygen breathing and this could be the result of the Hudson demand valve causing an increase in venous admixture due to insufficient flow rates delivered by the valve during peak inspiratory requirements in the conscious horse. The oxygen inlet pressure to the demand valve was increased to 620 kPa for maximal flow during inspiration. The inadequate flow rate may only be a significant factor in spontaneously breathing conscious horses and should not affect its use in post operative oxygen administration to a hypoventilating horse. The significant correlation between body mass and pulmonary shunt in dorsal recumbency and for venous admixture in dorsal recumbency when breathing oxygen may be related to the effect of the abdominal organs causing decreased ventilation and closure of distal airways in the caudal lung lobes. During expiration lung volume decreases and functional residual capacity decreases below closing capacity (closing volume + residual volume). Closure of terminal bronchi as result of the mass of abdominal organs pressing on the caudal lung lobes leads to absorption atelectasis and venous admixture. When the horse breathes oxygen the incidence of atelectasis is higher as compared to when it breathes air. If the horse breathes air, which contains nitrogen, the sum of the partial pressure of gases in mixed venous blood is higher than the sum when the animal breathes oxygen and halothane[20]. The nitrogen has a 'splinting' effect on the alveoli, decreasing the incidence and rate of absorption atelectasis, decreasing venous admixture and increasing alveolar ventilation[3]. The improved ventilation is reflected in the decreased Pa_{CO_2} when breathing air in dorsal recumbency. In lateral recumbency the effect of the abdominal mass is directly on the dependent lung and the 'splinting effect' from nitrogen is insufficient to prevent hypoventilation, the closing down of distal airways and absorption atelectasis.

In conclusion it may be said that in the anaesthetised laterally recumbent horse hypoventilation, as a result of the direct effect of the abdominal mass immobilizing the dependent lung, results in arterial hypoxaemia. The hypoxia is caused by a combination of factors which include ventilation-perfusion mismatch in the dependent lung and a decreased cardiac output that results in a decreased in PV_{O_2}. In the dorsally recumbent horse a further decrease in cardiac output occurs; venous admixture from the V/Q mismatch decreases and from pulmonary shunt increases. The extent of pulmonary shunt is directly related to body mass in dorsal recumbency when breathing oxygen, but not in lateral recumbency. Alveolar ventilation in dorsal recumbency during halothane anaesthesia may be improved during spontaneous ventilation by the use of oxygen-enriched air instead of an oxygen-halothane mixture.

REFERENCES

1. Butler H C 1962 Subcutaneous relocation of the carotid artery for experimental purposes. American Journal of Veterinary Research 23: 165
2. Comroe J H, Foster R E, Dubois A B, Briscoe W A, Carlsen E 1962 Alveolar ventilation. In: The Lung. Year Book Medical Publishers Inc, Chicago
3. Déry R, Pelletier J, André J, Clavet M, Houde J 1965 Alveolar collapse induced by denitrogenation. Canadian Anaesthetic Society Journal 12: 531-544
4. Eberley V E, Gillespie J R, Tyler W S Fowler M E 1968 Cardiovascular values in the horse during halothane anesthesia. American Journal of Veterinary Research 29: 305-313
5. Gillespie J R, Tyler W S, Hall L W 1969 Cardiopulmonary dysfunction in anaesthetised laterally recumbent horses. American Journal of Veterinary Research 30: 61-72
6. Hall L W, Gillespie J R Tyler W S 1968 Alveolar-arterial oxygen tension differences in anaesthetised horses. British Journal of Anaesthesia 40: 560-568
7. Hall L W 1984 Cardiovascular and pulmonary effects of recumbency in two conscious ponies. Equine Veterinary Journal 16: 89-92
8. Hillidge C J, Lees P 1975 Cardiac output in conscious and anaesthetised horses. Equine Veterinary Journal 7:16-21
9. McDonell W N 1974 The effect of anaesthesia on gas exchange and arterial oxygeneration in the horse. PhD Thesis University of Cambridge, England
10. McDonald W N, Hall L W, Jefcott L B 1979 Radiographic evidence of impaired pulmonary function in laterally recumbent anaesthetised horses. Equine Veterinary Journal 11:24-32
11. Nunn F J 1963 Indirect determination of the ideal alveolar oxygen tension during and after nitrous oxide anaesthesia. British Journal of Anaesthesia 35: 8-10
12. Nunn F J 1969 Distribution of the pulmonary blood flow. In: Applied Respiratory Physiology with Special Reference to Anaesthesia. Butterworths, London
13. Riley R L, Cournand A 1949 'Ideal' alveolar air and the analysis of ventilation/perfusion relationships in the lungs. Journal of Applied Physiology 1:825-847
14. Rugh K S, Garner H E, Hatfield D G, Herrold D 1984 Arterial oxygen and carbon dioxide tensions in conscious laterally recumbent ponies. Equine Veterinary Journal 16: 185-188
15. Severinghaus J W, Stupfel M 1957 Alveolar dead space as an index of distribution of blood flow in pulmonary capillaries. Journal of Applied Physiology 10: 335-348
16. Severinghaus J W 1966 Blood gas calculator. Journal of Applied Physiology 21: 1108-1116
17. Steffey E P, Wheat J D, Norrie R D, McKee J, Brown M, Arnold J 1977 Body position and mode of ventilation influences arterial pH, oxygen and carbon dioxide tensions in halothane-anaesthetized horses. American Journal of Veterinary Research 38: 379-382

18. Stegmann G F 1986 Pulmonary function in the horse during anaesthesia — a review. Journal of the South African Veterinary Association 57: 49-53
19. Stolk P W Th 1982 The effect of anaesthesia on pulmonary blood flow in the horse. Proceedings of the 1st International Congress of Veterinary Anaesthesia: 119-129 Cambridge, England
20. West J B 1979 Respiratory physiology in unusual environments In: Respiratory Physiology — the Essentials Williams and Wilkens, Baltimore

ABSTRACT

SAMEVATTING

PARASITES IN SHEEP GRAZING ON KIKUYU PASTURES IN THE WINTER RAINFALL REGION

Regular worm counts were done post-mortem on sheep that had grazed on Kikuyu pastures at the Elsenburg Research Station near Stellenbosch, a winter rainfall region. Major species were *Trichostrongylus colubriformis, Trichostrongylus axei,* while *Ostertagia circumcincta* was usually present in large numbers.

Minor species were *Haemonchus contortus, Nematodirus spathiger, Dictyocaulus filaria, Oesophagostomum venulosum, Trichuris* spp., *Chabertia ovina* and larvae of the arthropod *Oestrus ovis. Muellerius capillaris* caused the formation of nodules in the lungs but were not counted.

The trial started in April 1982 and was concluded in March 1984. One hundred and four sheep died or were slaughtered and 99 were examined post-mortem during this period. Total worm burdens rose to a peak of 88 763 (range 67 281-124 735) worms in March 1983, i.e. sheep mortality was such that the flock had to be treated with an anthelmintic in April 1983 to prevent further losses.

Kikuyu pastures provide shade, form an excellent mat, the humus layer under the grass retains moisture and is an excellent incubator for preinfective larvae and a protector for infective larvae. If these qualities are combined with more than 100 mm of rain in spring and summer, Kikuyu pastures are a paradise for the free-living stages. (Reinecke, R.K., Kirkpatrick, R., Swart, Lydia, Kriel, Anna M.D. & Frank, F., 1987. Parasites in sheep grazing on Kikuyu *(Pennisetum clandestinum)* pastures in the winter-rainfall region. *Onderstepoort Journal of Veterinary Research,* 54, 27-38 (1987).)

ABSTRACT

SAMEVATTING

A TREMORGENIC MYCOTOXICOSIS OF CATTLE CAUSED BY MAIZE SPROUTS INFESTED WITH *ASPERGILLUS CLAVATUS*

An outbreak of disease affecting a herd of 16 dairy cattle which were fed mouldy, sprouted maize is described. Eight of the cattle were affected, 5 of which died. The clinical signs included muscular tremors, hypersensitivity, ataxia, anorexia and salivation. *Aspergillus clavatus* was the only fungus isolated from the sprouts.

Clinical signs that were indistinguishable from those in the field outbreak were reproduced by dosing the mouldy maize sprouts to a steer and a sheep, and by dosing another sheep with maize inoculated with a pure culture of *A. clavatus* isolated from the mouldy maize on the farm. Light microscopical examination revealed neuronal degeneration and necrosis in the midbrain, medulla oblongata and spinal cord of all 3 of these animals.

The disease is clinically and pathologically indistinguishable from the disease caused by the ingestion of sorghum beer residue, and in certain respects it is similar to toxicoses caused by the ingestion of wheat sprouts and malt sprouts infested with *A. clavatus.* (Kellerman, T.S., Newsholme, S.J., Coetzer, J.A.W. & Van der Westhuizen, G.C.A., (1984). A tremorgenic mycotoxicosis of cattle caused by maize sprouts infested with *Aspergillus clavatus. Onderstepoort Journal of Veterinary Research,* 51, 271-274 (1984).)

ARTICLE ARTIKEL

EFFICACY OF IVERMECTIN AGAINST THE PIG MANGE MITE *SARCOPTES SCABIEI* VAR. *SUIS*

M.D. SOLL* and C.J.Z. SMITH*

ABSTRACT: Soll M.D.; Smith C.J.Z. **Efficacy of ivermectin against the pig mange mite, Sarcoptes scabiei var. suis.** *Journal of the South African Veterinary Association (1987)* **58** No. 1, 29-30 (En) MSD Research Centre, Private Bag 3, 1685 Halfway House Republic of South Africa.

The efficacy of a single subcutaneous dose of ivermectin at 300 μg/kg was evaluated against *Sarcoptes scabiei* in a commercial herd of 146 pigs of which more than 80% were naturally clinically affected with mange. All pigs, except 6 control animals, were treated with ivermectin on Day 0. Scrapings for mite recovery were made from 12 treated animals and 6 controls at the time of treatment and 28 and 42 days later. No mites were recovered from pigs after treatment while live mites were present in scrapings from all control animals. No treated pigs had active lesions at examination on Day 42. The results are discussed in relation to recommendations for a control program for *Sarcoptes scabiei* in pigs.

Key words: Sarcoptic mange, pigs, ivermectin.

INTRODUCTION

Sarcoptic mange or scabies is a highly contagious parasitic disease of pigs caused by *Sarcoptes scabiei* var. *suis*. The disease is usually manifested by generalized pruritis accompanied by focal erythematous skin lesions and resultant traumatization of the skin by rubbing and scratching. This condition is attributed mainly to a hypersensitivity reaction to the mites and lasts for up to 11 weeks[5]. The mites initially multiply unchecked until the hypersensitive response develops whereafter mite numbers decline, except in the so-called 'chronic mange' pigs which either do not become hypersensitized or have become prematurely desensitized[5]. These pigs harbour large numbers of mites and provide a constant source of reinfestation to hypersensitive pigs, keeping them in a pruritic state[6]. The chronic form of the disease is manifested by extensive crust formation, hyperkeratosis, acanthosis and underlying dermatitis.

Growth rate and feed conversion are poorer in *Sarcoptes*-infested pigs than in healthy pigs[6] and improvement in performance and condition has been demonstrated following treatment[1,12].

The current method of treatment is by topical application of an acaricide. Mites frequently survive a single application, probably because they are physically protected under crusts. Furthermore, adequate wetting of certain areas, such as in the ears and on the feet, is difficult to achieve. These treatments are time consuming, labour intensive and may not adequately control the parasite if they are not regularly and correctly applied.

In addition to the pig louse, *Haematopinus suis*[3,13], ivermectin is effective against *Sarcoptes scabiei* in naturally and experimentally infested pigs when administered orally[2,9] or parenterally[7]. Control of sarcoptic mange mite infestation in commercial piggeries has also been reported[8,10].

A trial was conducted in a commercial piggery in South Africa to evaluate the efficacy of a single subcutaneous injection of ivermectin at 300 μg/kg against hyperkeratotic pig scabies.

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MATERIALS AND METHODS

One hundred and thirty-eight adult Large White and Landrace sows and 8 Large White boars, naturally infested with *Sarcoptes scabiei* were included in the trial.

Eighteen sows were selected on the basis of being severely affected with typical chronic hyperkeratotic scabies. Of these, 6 were allocated to an untreated control group and 12 served as treated indicator animals.

All adult animals on the property (with the exception of the 6 sows allocated to the control group) were treated with ivermectin (IVOMEC Injectable, MSD) at 300 μg/kg subcutaneously on Day 0.

Treated animals were either tethered in an open, roofed house, held in a closed farrowing house, were individually penned in sties or ran in open camps. Untreated control animals were individually housed in sties separate from treated animals.

Scrapings from an area approximately 3 cm x 1 cm, were taken from the inside surface of the ears of the control and indicator animals on Day 0 (prior to treatment) and again 28 and 42 days later.

Areas of encrustation and scaling where mites were likely to be found were selected for scraping. The scrapings were made using a sharpened teaspoon and the material collected was transferred to numbered test tubes which were sealed and transported to the laboratory where they were processed for mite recovery by a modification of the technique of Meleney[11] as follows.

Depending upon the amount of material collected, a convenient volume of water (usually 5-7 ml) plus a few drops of liquid detergent were added to the test tube which was placed in a sonicator for 10 minutes to break up the crusts. The mixture was then washed through a coarse sieve (to retain hair and large crusts) onto a ruled black filter paper disc in a Buchner funnel to which light vacuum was applied. This resulted in mites being well-distributed over the surface of the black filter paper

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which was then removed and placed in a petri dish; live mites were counted under a stereoscopic microscope.

The number of clinically affected pigs in the herd was visually assessed on Days 0 and 42.

RESULTS

The numbers of *Sarcoptes scabiei* mites recovered from scrapings taken from treated indicator animals and untreated control animals are given in Table 1.

Table 1: Numbers of mites recovered from pigs

Treatment	Animal No.	Day 0	Day 28	Day 4
Untreated control	401	1350	1638	1485
	483	897	1238	1247
	815	122	163	114
	816	169	249	553
	818	234	178	140
	819	273	97	29
	Mean	507,5	593,8	594,7
Ivermectin 300 µg/kg subcutaneously	214	46	0	0
	218	26	0	0
	405	632	0	0
	406	463	0	0
	421	536	0	0
	482	3	*	0
	486	674	0	0
	801	1106	0	0
	812	229	0	0
	813	1218	0	0
	817	696	0	0
	820	347	0	0
	Mean	498	0	0

* Animal not scraped

Mites were recovered from all animals scraped on Day 0. No mites were recovered from treated indicators on Days 28 and 42 while live mites were recovered from all control animals on these days.

All 8 boars and 85,5% of the sows were clinically affected (crusts, scaliness, erythema, hair loss, hyperkeratosis, scratching and rubbing and ear shaking) at the start of the trial.

After treatment, crusts dried out and lifted, erythema disappeared, hair regrowth began and the incidence of scratching, rubbing and ear shaking decreased. Chronic skin thickening and scaliness were, however, still evident on Day 42, especially on some boars.

Apart from the 6 control animals whose clinical condition deteriorated over the trial period, no pigs had active lesions on Day 42.

Most pigs were sold shortly after conclusion of the trial, making further assessments impossible.

DISCUSSION

This study confirms that ivermectin administered subcutaneously at 300 µg/kg brings about clinical and parasitological cure of pigs naturally infested with *Sarcoptes scabiei*. This disease is, however, usually a herd problem and not confined to single animals.

Under conditions of natural infestation piglets become infested from the sow and thus become sensitized to the parasite between birth and weaning[5]. Chronic mange is relatively rare in growing pigs, but the hypersensitive condition has considerable economic significance[6]. An effective control programme should aim at either reducing the level of infestation in sows or eradicating mites entirely from sows before farrowing[5]. The high efficacy of a single dose of ivermectin allows for a single effective prefarrowing treatment of sows and prevents transmission to their offspring[7]. Treatment of pigs at weaning will also eliminate infection[7].

A control programme could be initiated by treating all breeding animals and could subsequently be based on a single ivermectin treatment of sows before they are moved to the farrowing house[7] or at least 7-14 days prior to farrowing. This treatment would also have additional anthelmintic benefits[4 13 14]. Boars should be treated at least twice a year and breeding sows and gilts 7-14 days prior to breeding. Epizootics in growing pigs could be controlled by treatment of all contact pigs[7]. All new stock should be treated and isolated for at least 21 days before introduction into the herd[8]. A procedure whereby all pigs in a facility are given a single ivermectin treatment on the same day could form the basis of an eradication programme[8 10].

REFERENCES

1. Alva R, Wallace D H, Benz G W, Foster A G, Brokken E S 1984 The effects of ivermectin on the productivity of pigs naturally infested with *Sarcoptes scabiei* var. *suis*. Proceedings of the International Pig Veterinary Society Congress 8 : 207
2. Alva-Valdes R, Wallace D H, Benz G W, Foster A G, Holste J E 1984 Efficacy of ivermectin against the mange mite *Sarcoptes scabiei* var. *suis* in pigs. American Journal of Veterinary Research 45:2113-2114
3. Barth D, Brokken E S 1980 The activity of 22, 23 dihydroavermectin B_1 against the pig louse *Haematopinus suis*. Veterinary Record 106:388
4. Brokken E S, Roncalli R A, Sutherland I H, Leaning W H D 1984 Ivermectin, a new broad spectrum antiparasitic agent for swine. Proceedings of the International Pig Veterinary Society Congress 8:205
5. Cargill C F, Dobson K J 1979 Experimental *Sarcoptes scabiei* infestation in pigs: (1) Pathogenesis. Veterinary Record 104:11-14
6. Cargill C F, Dobson K J 1979 Experimental *Sarcoptes scabiei* infestation in pigs: (2) Effects on production. Veterinary Record 104:33-36
7. Courtney C H, Ingalls W L, Stitzlein S L 1983 Ivermectin for the control of swine scabies: Relative value of prefarrowing treatment of sows and weaning treatment of pigs. American Journal of Veterinary Research 44:1220-1223
8. Hogg A 1984 Eradication of sarcoptic mange in swine with ivermectin. Proceedings of the International Pig Veterinary Society Congress: 8
9. Lee R P, Dooge D J D, Preston J M 1980 Efficacy of ivermectin against *Sarcoptes scabiei* in pigs. Veterinary Record 107:503-505
10. Martineau G P, Vaillancourt J, Fréchette J L 1984 Control of *Sarcoptes scabiei* infestation with ivermectin in a large intensive piggery. Canadian Veterinary Journal 25:235-238
11. Meleney W P 1982 Control of psoroptic scabies on calves with ivermectin. American Journal of Veterinary Research 43:329-331
12. Sheahan B J, O'Connor P J, Kelly E P 1974 Improved weight gains in pigs following treatment for sarcoptic mange. Veterinary Record 95:169-170
13. Stewart T B, Marti O G, Hale O M 1981 Efficacy of ivermectin against five genera of swine nematodes and the hog louse, *Haematopinus suis*. American Journal of Veterinary Research 42:1425-1426
14. Stewart T B, Marti O G, McCormick W C 1981 Efficacy of ivermectin against swine kidney worm, *Stephanurus dentatus*. American Journal of Veterinary Research 42:1427-1428

GEVALVERSLAG **CASE REPORT**

BURSITIS CALCAREA IN 'N HOND

J.S.J. ODENDAAL* en L.B. EVANS**

ABSTRACT: Odendaal J.S.J.; Evans L.B. Bursitis calcarea in a dog. *Journal of the South African Veterinary Association* (1987) **58** No. 1, 31-32 (En) (Afrik.) Department of Zootechnology, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, 0110 Onderstepoort, Republic of South Africa.

A case of bursitis calcarea in a Border Collie showing lameness without acute pain in the right hind leg, is reported. A diagnosis of bursitis calcarea was made on radiological examination. Treatment consisted of anti-inflammatory drugs and rest as far as it was practically possible. After initial improvement, full recovery only took place 2 months after treatment had commenced.

Bursitis calcarea is a rare disease which seldom shows clinical signs. As far as could be ascertained, this is the first report of the condition in South Africa.

Key words: Bursitis calcarea, dog, radiology, rare condition, lameness.

INLEIDING

Bursitis calcarea is beenneerlegging in een van die bursas van die trochanters van die femur, met 'n gepaardgaande holte in die trochanter vanwaar die been gevorm het.

Om die voorkoms van bursitis calcarea in honde vas te stel, is 'n uitgebreide ondersoek van 5 000 gevalle, wat aangebied is vir roetine röntgen-ondersoeke vir heupdisplasie oor 5 jaar, onderneem[2]. Van hierdie honde het slegs 79 gevalle (1,6%) in 'n mindere of meerdere mate tekens van bursitis calcarea getoon. Die meeste honde was tussen 1 - 1½ jaar oud, maar die ouderdomme van die gevalle het gestrek van 5 maande tot 12 jaar. Die verdeling was as volg: 5 gevalle was 5 -11 maande oud; 30 gevalle was 1 - 1½ jaar; 18 gevalle was 2 - 6 jaar; en 26 gevalle was 7 - 12 jaar. Veertig van die honde was tewe en 39 reuns[2].

Die toestand het 'n geneigdheid tot groter rasse getoon, terwyl middelslag rasse ook aangetas was. Die insidens was die hoogste in Rottweilers (27), gevolg deur Duitse Herdershond (16), Poodles (9), Spanjoele (8) en Great Danes (4). Omdat die honde in die eerste plek vir heupdisplasie ondersoeke aangebied is, was dit moeilik om te oordeel wat die insidens van die toestand in kleinras honde is[2].

Bursitis calcarea het in 21 gevalle (di. 0,4% van die totale gevalle en 26,8% van die aangetasde diere) regs voorgekom, in 32 gevalle links en 26 hond het bilaterale aantasting getoon[2].

Die posisie van bursitis calcarea ten opsigte van die trochanters op röntgen foto's wat van die ventro-dorsale posisie geneem is, was in 58 gevalle (di. 1,2% van die totale gevalle en 73,4% van die aangetasde diere) dorsaal en in 8 honde lateraal van die *T. major*; en in 13 gevalle dorsaal van die *T. minor*[2].

Die bursas vorm tussen die trochanters en die pees -aanhegtings van die spiere. By die *T. major* is daar bursas onder die *M. gluteus superficials* en *M. gluteus medius*. Laasgenoemde se bursa strek tot onder die *M. gluteus profundis* en die *M. biceps femoris*. Op die *T. minor* vorm 'n bursa onder die *M. iliopsoas*[4].

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Geen behandelings is uitgevoer of voorgestel in die gevalle nie, omdat die diagnose nie gemaak is op kliniese tekens nie, maar op grond van toevallige bevindings tydens heupdisplasie ondersoeke.

GESKIEDENIS EN KLINIESE ONDERSOEK

'n Dertig-maande oue gesteriliseerde Border Kollie teef, is ingebring vir ondersoek, met die klagte dat die hond in die regter agterbeen oor 'n tydperk mankheid toon. Die mankheid het nie konstant voorgekom nie, was by tye erger, en het die laaste week toegeneem. Andersins was die hond baie lewendig en gesond. Daar was geen geskiedenis van enige trauma in die hond gewees nie.

Nadat 'n algemene kliniese ondersoek gedoen is, is daar gekonsentreer op die regter agterbeen. Die been was met palpasie nie pynlik, warm of geswolle nie en kon buig sonder sigbare pyn. As die linker agterbeen vinnig opgetel is, kon die regterbeen vol gewig neem. Tydens beweging het die hond nie die regter agterbeen gedra nie, maar het tog effense mankheid getoon as sy loop, en was erger mank as sy gehardloop het. Die kruis-ligamente, patella-ligamente, die sooltjies van die poot en been-reflekse is ondersoek, maar geen abnormaliteite is gevind nie.

DIAGNOSE

Omdat daar nie 'n spesifieke diagnose tydens die kliniese ondersoek gemaak kon word nie, is daar besluit om 'n ventro-dorsale röntgenfoto onder algemene narkose van die bekken area te neem.

Die foto het 'n duidelike 5 mm by 4 mm groot, kalkagtige liggaampie dorsaal van die *T. major* getoon. Daar was 'n ooreenstemmende skaduwee op die proksimale oppervlakte van die *T. major*, wat klaarblyklik gedui het op die holte vanwaar die liggaampie sy oorsprong geneem het. (Foto 1)

Metastatiese kalsifikasie word gedefinieer as die neerslag van kalsiumsoute in sagte weefsels, wat nie deel was van die area van 'n vorige besering nie. Anders gestel, distrofiese kalsifikasie word gekenmerk deur die neerlegging van kalsiumsoute in beseerde, degeneratiewe of dooie weefsels. Volgens Morgan[3] mag distrofiese kalsifikasie dus tog van kliniese belang wees. Die radiografiese voorkoms van distrofiese kalsifikasies varieer.

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Fig. 1: Bursitis calcarea in die regter *Trochanter major* van 'n Border Kollie. Die pyltjies toon die donker holte waaruit die kalsifikasie gevorm het en die los beentjie wat 5 mm by 4 mm groot is.

Dit mag amorf en multi-sentries wees, of dit kan duidelik as 'n meer omskrewe, uitstaande gekalsifiseerde digtheid waargeneem word. Laasgenoemde vorm neig om ooreen te stem met die radiologiese bevindings in gevalle van bursitis calcarea. Dit kan daarop dui dat die toestand tog moontlik deur trauma veroorsaak word, kliniese tekens tot gevolg mag hê en as 'n uitstaande kalsifiseerde liggaampie(s) in die aangetasde weefsels waargeneem kan word. Die *M. gluteus medius* se aanhegting op die punt van die *T. major* is 'n baie moontlike area van besering, met gevolglike distrofiese kalsifikasie, en die ontwikkeling van bursitis calcarea. Die besering en gepaardgaande inflammasie van die pees-aanhegting, kan wel inflammasie insluit van die trochanter se bursa, wat tussen die kraakbeen oor die trochanter en die peesaanhegting lê.

In die mens is die tweede algemeenste area van bursitis, ná die skouergebied, die trochanter[1]. Met bursitis is die bursa meesal edemateus, hiperemies en verdik. As die gekalsifiseerde materiaal uit die bursa vrygelaat word, word die pyn dikwels spontaan verlig[1]. Hierdie bevinding in die mens kan dalk direk van toepassing wees in die geval van die Border Kollie wat hier beskryf word.

BEHANDELING

Chirurgie is oorweeg, maar aangesien die graad van simptome nie van 'n ernstige graad was nie, en omdat die los beentjie so klein was, is dit laat vaar. Konserwatiewe behandeling is dus toegepas wat bestaan het uit anti-inflammatoriese middels en rus.

Die hond het 0,75 ml deksametasoon (Dexafort, Panvet) binnespiers gekry, wat na 4 dae opgevolg is met 2 tablette 100 mg fenielbutasoon en 200 mg isopirien (Tomanol, Byk Gulden), twee maal per dag, per os vir 10 dae. Rus is sover prakties moontlik toegepas, aangesien Border Kollies aktiewe honde is. Geen gedwonge oefeninge, spelery, of plesier-stappery saam met die eienaar is toegelaat nie en die hond is so stil moontlik in 'n agterplaas gehou.

Na die 14 dae behandeling was daar 'n merkbare verbetering. Een maand na die behandeling begin is, het die hond nog net van tyd tot tyd effense mankheid getoon as sy gehardloop het. 'n Verdere maand later is die laaste ondersoek gedoen en toe was die mankheid afwesig.

BESPREKING

Bursitis calcarea is 'n raar kondisie in honde en sover as die outeurs kon vasstel, is hierdie die eerste verslag van die toestand in Suid-Afrika.

Faktore wat bydra tot bursitis calcarea is nog onseker, maar daar is moontlik 'n verband tussen die toestand en vinnig groeiende rasse asook aktiewe honde. Trauma speel waarskynlik 'n rol en kongenitale oorsake is nie uitgesluit nie.

Mayrhofer en Olensky[2] se ondersoek het gegaan oor honde wat nie kliniese simptome getoon het nie, terwyl die geval onder bespreking spesifiek ingebring is met die klagte van mankheid. Bursitis calcarea behoort dus as 'n differensiële diagnose van mankheid in honde beskou te word.

Die behandeling was simptomaties, aangesien die mankheid eerder die indruk van ongemak as pyn gewek het. Konserwatiewe behandeling oor 'n tydperk, blyk 'n praktiese benadering te wees.

Volgens Mayrhofer en Olensky[2] kan die volgende radiologiese toestande met bursitis calcarea verwar word: neerslae en corpora libra in die heupgewrig, splinter-frakture, eksostose, ossifikasie punte, miositis ossificans, foute op die röntgenfoto en skaduwees van die papilla mammae in tewe.

BRONNELYS

1. Edeiken J 1981 Röntgen diagnosis of diseases of bone. 3rd edn. Williams and Wilkins, Baltimore: 1105-1106
2. Mayrhofer E, Olensky G 1978 Bursitis calcarea in Hüftgelenksbereich beim Hunde. Wiener Tierärztliche Monatsschrift 65: 193-199
3. Morgan J P 1972 Radiology in Veterinary Orthopaedics. Lea and Febiger, Philadelphia: 371-374
4. Sisson S, Grossman J D 1966 The anatomy of the domestic animals. 4th edn. W B Saunders Co., Philadelphia: 380-385

SOÖTEGNOLOGIE VAN GESELSKAPSDIERE — 'N NUWE BEHOEFTE, 'N NUWE UITDAGING

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ABSTRACT: Odendaal J.S.J. **Zootechnology of companion animals — a new need, a new challenge.** *Journal of the South African Veterinary Association* (1987) **58** No. 1, 33-37 (En), (Afrik.) Department of Zootechnology, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, 0110 Onderstepoort, Republic of South Africa.

The outline and motivation for a new subject in the veterinary curriculum is given. The pregraduate curriculum is divided into ethology, handling, management and care, breeding, behaviour, nutrition, knowledge of breeds, shows, obedience and training of companion animals, the possible clinical implications of normal breed characteristics, the companion animal market, practice management, the study of the human to animal contact and the specialised application of such contacts.

Key words: Zootechnology, companion animals, curriculum.

INLEIDING

Wanneer 'n nuwe vak tot die veterinêre kurrikulum gevoeg word, behoort dit nie net van akademiese belang te wees nie, maar ook vir die hele professie. Die vraag kan tereg gevra word of daar nog plek is vir "nuwe" vakke en studierigtings in 'n reeds oorvol kurrikulum. Sou bykomende kennis werklik van nut wees vir die toekomstige veearts en professie, asook vir die publiek wie die professie bedien.

Om hierdie vrae te beantwoord, is besluit om 'n vakomlyning wat ook as motivering kan dien, aan die professie bekend te stel.

Die doelwitte bly steeds opleiding, navorsing en diens aan die gemeenskap soos dit in die universiteitsbeleid bepaal word. Opleiding sal in hierdie artikel meer volledig bespreek word.

OPLEIDING

Die vak soötegnologie van geselskapsdiere, is 'n studie wat handel oor die tegniek om diere tot voordeel van die mens aan te wend, sonder dat diere daardeur misbruik word. Die spesies hier ter sprake is die sg. geselskapsdiere waarvan die hond en kat klassieke voorbeelde is.

Hierdie is natuurlik nie 'n totaal nuwe vak nie, maar daar is gepoog om die meer bekende dele met nuwe klem en oorgawe te doseer, sowel as om 'n aantal nuwe perspektiewe toe te voeg. Die benadering is oorwegend prakties georiënteerd en toepassing van die biologiese wetenskappe is die uitgangspunt van die leerplan.

Die leerplan word in twaalf afdelings verdeel en elke afdeling word kortliks bespreek. Elke afdeling word in die kursus vooraf gegaan deur 'n aantal doelstellings en daarna word die praktiese voordele wat dit vir die veearts inhou, uitgewys.

1 Geselskapsdiere se gedrag

Die domestikasie-proses van geselskapsdiere is steeds in spekulasie gehul terwyl aanduidings gevind is dat die hond sedert die begin van die geskiedenis 'n noue band met die mens gehad het. Die kat se noue verbintenis met die mens dateer ongeveer 7 000 jaar terug. Alhoewel die domestikasie-proses grootliks onbekend is, is dit egter duidelik dat as hierdie diere se gedrag nie onder die beheer van die mens gebring kon word nie, hulle steeds ongedomestiseerd of "wild" sou gebly het. Die steeds nouer-wordende kontak tussen die mens en geselskapsdier vereis dat diere se gedrag beter verstaan moet word. Die sukses van mense, soos veeartse wat met diere werk, hang reeds nou saam met die kommunikasie wat hulle met die dier kan bewerkstellig asook die beheer wat hulle oor die diere kan uitoefen.

'n Mens kan seker toegee dat daar persone is wat 'n natuurlike aanvoeling, of sesde sintuig het om diere te verstaan, maar 'n basiese boekekennis bly steeds van primêre belang — veral dan vir dié van ons met net vyf sintuie! Tweedens is praktiese oefening ten opsigte van dieregedrag nodig. Dit impliseer dat studente soveel en so lank as moontlik blootgestel moet word aan diere. Gesonde diere moet dus altyd beskikbaar wees sodat studente in die teenwoordigheid van diere kan verkeer.

Diere se gedrag is nie 'n gegewe konstante nie want diere se temperament en aanpassingsvermoë verskil. Daar is dus variasies om te ken en te antisipeer. Verrassingsgedrag kan gevaarlik wees en dit help om voorbereid te wees op sg. onvoorspelbare gedrag. Die veearts wat met 'n groot aantal vreemde diere teen 'n vinnige tempo kontak maak, kan nie bekostig om aanhoudend met onbeheerbare diere te sukkel nie. Daar moet deur die kennis van dieregedrag onderskei word tussen afwykende (abnormale) en vir die mens onaanvaarbare gedrag (maar wat wel normaal kan wees vir die dier). Dit is belangrik as advies gegee moet word ten opsigte van klagtes oor gedrag en die behandeling daarvan.

2 Hantering van geselskapsdiere

'n Saak wat nou aansluit by dieregedrag is die hantering van diere. 'n Mens kan byna nie waag om 'n dier te hanteer voordat die dier se temperament of gedragsreaksies vasgestel is nie. Die hantering van diere vind meesal ten aanskoue van een of meer omstanders plaas. Die wyse waarmee die veearts die dier hanteer laat meesal 'n blywende indruk ten opsigte van die hele pro-

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fessie! Al word korrekte hantering nie altyd opgelet nie, kan swak of onbeholpe hantering die veearts se beeld ernstig skaad.

'n Ander belangrike aspek van dierehantering is beserings. Ondoeltreffende hantering lei nie net tot beserings van die dier nie, maar dikwels ook van die veearts. In sekere sin is die veearts 'n "hande-arbeider". Dis feitlik ondenkbaar dat 'n veearts sy praktyk voortsit sonder twee gesonde hande. Wonde, pyn en sepse opgedoen a.g.v. onbehoorlike hantering van diere, belemmer daaropvolgende hanterings en veroorsaak soms totale ongeskiktheid om die werk voort te sit. Dit kan lei tot 'n verlies aan inkomste, 'n verlies aan waardigheid en soms self 'n verlies van 'n kliënt.

Enige kliniese werk begin met die korrekte hantering van die dier. Dit is dus ideaal dat studente hierdie aspek deeglik onder die knie kry vóórdat hulle die kliniese vakke aanpak. 'n Onhanteerbare dier kan nie ondersoek of behandel word nie. Goeie hantering lei tot 'n beter kliniese ondersoek, diagnose, behandeling en sukses. Suksesvolle behandeling hang ook af van suksesvolle hantering veral as sekere tuisbehandelings van die eienaar verwag word. Die veearts behoort in sulke gevalle die korrekte hantering vir byvoorbeeld doserings te kan demonstreer, anders kan die kliënt dalk nie die behandelings tuis uitvoer nie. 'n Praktyk waar diere nie korrek hanteer word nie, kan nie floreer nie. Alhoewel daar helpers en verpleegsters is wat die veearts kan bystaan, moet hy persoonlik steeds die verantwoordelike persoon wees wat die moeilikste gevalle kan hanteer. By sogenaamde onhanteerbare gevalle het die veearts ook die kennis van, en toegang tot middels en middele om hantering te vergemaklik. Dit is uiteindelik die veearts wat bewondering moet afdwing vir sy korrekte hantering van diere — veral waar ander persone in hierdie opsig gefaal het.

3 Bestuur en versorging van geselskapsdiere

Die bestuur en versorging van geselskapsdiere kry al hoe groter betekenis, namate geselskapsdiere in groepe aangehou word vir toegepaste doeleindes, soos byvoorbeeld die sekuriteitsafdelings van groot instansies. Hier word honde gebruik om te help wagstaan, om onluste te beheer, om patrolliewerk te doen en om misdaad te bestry. Daar is ook die bedryf van dierehotelle rondom stede. Troeteldierwinkels laat dikwels veel te wense oor en dierebeskermingsverenigings moet ook in steeds groter mate diere huisves en versorg. Die nuutste neiging is ook om diere in toegepaste verband by institusies aan te hou. Die bestuur en versorging van hierdie groepe diere is van kardinale belang as daar teenprestasie van die diere verwag word. Indien daar probleme in die verband opduik, en gewoonlik is dit kroniese probleme wat op 'n dag chaos veroorsaak, is dit die veearts wat genader word om die saak reg te stel. Die veearts as kenner moet kan onderskei tussen doeltreffendheid van stelsels al dan nie. Swak bestuur en versorging kan net soos voeding aanleiding gee tot kliniese siektetoestande. 'n Basiese kennis oor behuising, higiëne, sanitasie, parasietbeheer en voorkomende geneeskunde moet deel wees van die veearts se bydrae in hierdie verband. Alhoewel sommige meen dat dit hier maar bloot gaan om die gebruik van gesonde verstand, sal 'n mens verbaas wees juis hoeveel sogenaamde gesonde verstand by leke ontbreek. Die veearts is ook ten nouste betrokke by die bestuur en versorging van siek diere wat in sy sorg geplaas is by dierehospitale en klinieke. Die aanhou van diere en die spanning wat daarmee gepaard gaan, is beslis ook faktore wat in ag geneem moet word. Basiese beginsels, die bestuur en versorging van diere moet neergelê word aangesien dit die beginpunt van die uiteindelike sukses is waarvoor die diere aangehou word. Indien die veearts hier ferme leiding kan neem, kan vele onnodige probleme in die toekoms voorkom word.

4 Telingsgedrag van geselskapsdiere

Hierdie is een onderwerp waar daar by die publiek groot onkunde oor bestaan. Die mees algemene fout is om kennis oor die mens se geslagsfunksies netso op diere toe te pas. Soos in baie ander gevalle word spesie-verskille nie behoorlik begryp nie. Dit lei tot talle navrae in die praktyk oor diere se normale telingsgedrag. Die veearts behoort ook te kan adviseer oor teelprogramme, asook oor probleme met telingsgedrag. Gedragsprobleme moet behandel word sover dit prakties moontlik is en daar moet later onderskei kan word tussen gedragsprobleme en patologies-geslagskundige toestande.

As dit kom by die teel van diere het die veearts 'n eweeens belangrike rol te vervul. Massatelers met winsbejag as primêre doel dra niks by tot 'n beter geselskapsdieropset nie. Diere in hierdie verband word dikwels misbruik en voornemende kopers word dikwels om die bos gelei. Dit het hoog tyd geword dat die professie in hierdie veld aktief betrokke raak en standpunt inneem. Massateling wat net één aspek van teling in berekening bring naamlik winsneming, kan selfs as wreedheid teenoor diere gereken word. Daar is ook aan die anderkant geregistreerde telers wat leiding van die veeartse nodig het. Daar is oral sogenaamde kenners wat slegs van die veearts se dienste gebruik maak indien 'n teelprogram misluk. Telers meen dikwels dat inteling die normale verloop van sake is. 'n Balans tussen verbreding van die genetiese poel en die behoud van ras-egtheid moet egter nagestreef word. Die veearts behoort hierby ten nouste betrokke te raak.

5 Kontak tussen mens en geselskapsdiere

'n Moderne neiging is die herevaluasie van geselskapsdiere. Daar is tans dwarsoor die wêreld studies aan die gang oor hierdie onderwerp. Die band tussen mens en dier is eeue oud en dit lyk asof die intensiteit van hierdie verhouding aan die toeneem is. As daar dus ooit 'n standhoudende simbiose tussen spesies was, dan is hierdie 'n goeie voorbeeld daarvan. Die hond is byvoorbeeld van nature 'n aandagsoeker en hy het hierdie aandag, sowel as kos, water en versorging by die mens gekry. Aan die anderkant het die mens gou die hond tot sy eie voordeel ingespan vir beskerming en jag as 'n getroue vriend en metgesel. Die snel verstedelikingsproses van die huidige eeu, het daartoe gelei dat die kontak tussen mens en dier tot 'n mate verlore gegaan het. Intermenslike spanninge as gevolg van verstedeliking het toegeneem. Van die relatief ontspanne, vrye omstandighede met relatief minder verantwoordelikhede wat die mens ná aan die natuur ondervind het, het min van oorgebly en bevind die mens hom nou in 'n moderne samelewing waar groot getalle mense saamwoon. Eensaamheid bly egter steeds 'n wesenlike probleem. Baie mense hanteer hierdie situasie moeilik en ontvlugting volg dikwels. Neuroses en senusiektes het tot so 'n mate toegeneem, dat pasiënte dokters dikwels moet besoek as gevolg van

psigiese en psigosomatiese probleme. Veral gedurende die tagtigerjare "ontdek" wetenskaplikes van verskeie dissiplines dat daar ook onder andere 'n metode is waarmee die moderne mens sy spanning op 'n gesonde manier kan ontlaai. So word geselskapsdiere op verskeie maniere benut, of dan om letterlik diere "voor te skryf". Hierdie diere kan wees van die huishond of kat wat ons feitlik almal besit, tot in die mees gespesialiseerde toepassings. 'n Pionier op die gebied, dr Boris Levinson, beweer dat ons nog slegs 'n vae benul van die terapeutiese implikasie van troeteldiere het. Hy glo dat ons moontlik in diereterapie, 'n instrument gevind het wat dit moontlik maak om daardie onbegrypbare iets, wat emosionele genesing bevorder, oor 'n lang tydperk en onder 'n vergrootglas te kan ondersoek. Daar bestaan baie geleenthede tot navorsing in hierdie veld. As ons aanvaar dat ons kinders 'n goeie kans het om in 'n chaotiese versteurde wêreld groot te word, en ons besit 'n terapeutiese middel soos die gebruik van troeteldiere as 'n ondersteuning vir geestesgesondheid, dan betaam dit ons om hierdie moontlikhede ten volle te ondersoek en te ontwikkel. Indien ons dit nie doen nie, dan moet ons as volwassenes die volle verantwoordelikheid vir die gevolge van ons nalatigheid dra[6]. Die meeste mense het 'n behoefte aan die natuurlike, omdat die mens self deel is van die natuur. Plastiekspeelgoed en die alles-vervangbare goedere bring nie altyd rus en volledige ontlading nie, inteendeel, dit kan eerder spanning verhoog. Die gevoel vir natuurlike lewe, warmte, aanraking en aanvaarding wat geselskapsdiere kan bied, verskaf 'n kanaal vir spanningsverligting. Hierdie spannings-ontladingsvoorwerpe is beskikbaar soos dit die behoefte hom voordoen en dit vorm in die koper-en-glas omgewing 'n skakel terug na die natuur. Geselskapsdiere het wonderlike aanpassingsvermoë getoon deur hulle saam met die mens in die betonoerwoud te vestig. Die rede daarvoor kan wees die tweerigting voordeel wat mens en dier van mekaar kan kry en so is die eeue-oue simbiose voortgesit. Honde en katte is natuurlik nie die enigste diere wat hulle intrek saam met die mens in die stad ingeneem het nie. Kouvoëls, eksotiese diere en selfs perde rondom stede, speel vandag net so 'n belangrike rol. Die gebruik van die perd was bedreig deur die meganika, maar vandag word selfs in die ontwikkelste lande, groot getalle perde naby stede aangehou om kontak met die natuur te behou.

Ons kan dus aflei dat verstedeliking en spanning vir vandag se mens feite is, en dat hierdie feite vir die mens behoeftes laat ontstaan het om steeds kontak met die natuur te behou en om op 'n gesonde wyse van sy spanning ontslae te raak. Geselskapsdiere kan in beide hierdie behoeftes voorsien en op dié wyse is daar 'n hegte emosionele band tussen mens en dier gesmee[9].

Niemand kan ontken dat die veearts wat betrokke is by geselskapsdierpraktyk 'n groot verantwoordelikheid op hom neem nie — nie net om diere te dokter nie, maar ook om die behoeftes van die eienaar van hierdie diere in ag te neem. Of vandag se veearts so 'n verantwoordelikheid wil ontduik sal eenvoudig nie help nie, omdat die noue band wat tussen geselskapsdier en eienaar gevorm word, 'n realiteit is. Die voornemende veearts sal dus nie net van hierdie aspek moet kennis neem nie, maar hom ook daarvoor deeglik moet voorberei.

De Groot[3] meen dat daar in hierdie opsig 'n nuwe wetenskaplike benadering nodig is wat morele waardes, etiese oorwegings en emosionele betrokkenheid in ag moet neem. Anders kan die realiteite van die natuur nie vasgestel word nie. Die nuwe benadering kyk nie net vas teen oorsaak en gevolg nie, maar werk met 'n oop sisteem waarin die mens wel deeglik betrokke by is.

Kliënte verwag van die veearts hulp en besorgdheid en hulle soek leiding met betrekking tot 'n gepaste verhouding tussen hulle en die troeteldier. Ons moet nie toelaat dat kliënte so versot raak op hulle troeteldiere dat ander naby-mense se behoeftes uitgesluit word nie. Netso behoort ons ook nie 'n gesonde verhouding wat perspektief behou soos tussen 'n seun en sy hond, 'n huisvrou en haar kat, 'n man met sy jaghond of 'n ou weduwee met haar vet Dachshund, te ontmoedig nie, sê McCoy[8]. Hy sien die moderne ontwikkeling in Veterinêre Fakulteite as volg: Lede van Veterinêre Fakulteite moet besef dat in hul najaag van geneeskundige spesialisasies, hulle 'n tonnelvisie kan ontwikkel wat nadelige gevolge vir die studente kan hê. Ons behoort studente aan te moedig om te glo dat 'n gesonde dier *en* 'n gelukkige kliënt genoeg rede is vir volle werksbevrediging. Ons professie selekteer natuurlikerwys persone met sterk medemenslike waardes. In 'n wêreld wat die vermoë besit van selfvernietiging, vol van wrede dade van aggressie teenoor ander mense, kan die navolging van hierdie belangrike waardes dalk aangeteken staan as die veeartse se grootste bydrae tot die gemeenskap.

Hierdie sienings het ook alles te doen met professionalisme as gekyk word na wat Hopkins[5] oor 'n professie te sê het. Die basies etiese oorweging van enige professie moet die besorgdheid en hulp aan ander mense betrek. Soos ons ons spesialisasie in tegnieke verbeter (en dit is vanselfsprekend uiters belangrik), moet ons egter nie hierdie basiese konsep miskyk nie. Met so 'n siening beoefen ons 'n professie, sonder dit bedryf ons 'n ambag.

Herrick[4] onderskryf Hopkins se sienings van 'n professie en verklaar dat 'n professie aanvaar as sy hoofdoel 'n bydrae tot die gemeenskap; dit het 'n standaard van uitnemendheid en 'n kode waardeur sy lede die beste diens aan die publiek kan lewer, dit praat die waarheid, ontken valshede en dien deur gesonde oordeel, en nie emosies nie, asook deur feite en nie verbeelding nie en laastens, 'n professie stel 'n voorbeeld vir die gemeenskap deur sy morele optredes.

Die basiese wetenskap en kliniese agtergrond is eenvoudig nie meer genoeg om veeartse op te lei nie. Die moderne kliënt sal 'n veearts kies wat sy werk goed doen, maar wat ook aan die kliënt se emosionele behoeftes kan voldoen. Uiteindelik is dit tog nie die dier wat die veearts aanbeveel, hom betaal of tevrede is met die dienste gelewer nie, maar wel die eienaar van die dier. Alhoewel die pasiënt met die grootste mate van kennis en sorg behandel moet word, gaan sukses afhang van hoe die veearts met sy kliënte omgaan. Só vervul die veearts belangrike gemeenskapsdiens.

6 Toegepaste gebruike van geselskapsdiere

Met die voorafgaande as agtergrond sal hierdie kursus hom ook moet besig hou met gespesialiseerde toepassings van geselskapsdiere. Hierdie toepassings word veral op die para-mediese terrein aangewend, maar ander dissiplines is ook betrokke. Dit skep 'n buitengewone geleentheid vir inter-dissiplinêre samewerking.

Die mees bekende van hierdie gebruike is gidshonde vir blindes. Van die nuwere ontwikkelings is seinhonde vir dowes, dienshonde vir parapleë, geselskapsdiere in ouetehuise (as gelukbringers, besoekers, of inwonend).

Geselskapsdiere word ook al hoe meer gebruik in inrigtings vir fisiese en geestelike gestremdes asook langtermyn gevangenes. Daar is ook al ondersoek gedoen ten opsigte van die toelating van geselskapsdiere by kroniese en terminaal pasiënte in hospitale. Op onderwysgebied is geselskapsdiere al aangewend vir bewaarskole, weeshuise en selfs in die klaskamer. So word onder andere veeartse, medici, verpleegsters, kliniese sielkundiges, psigiaters, maatskaplike werkers en pedagoge by hierdie saak betrek. In Suid-Afrika word al hierdie velde reeds ondersoek vir behoorlike ontginning en ons glo dat dit ook nuwe beroepsgeleenthede vir die veearts en die veterinêre verpleegster sal skep.

Hoe nouer die kontak tussen mens en dier ontwikkel word, des te meer word die veearts onmisbaar in hierdie opset. As daar probleme kom, sal die veearts genader word. Dit is dus essensieel dat veeartse vir hierdie omstandighede voorbereid en opgelei moet wees ten einde leiding te kan neem. Indien die professie in hierdie saak onbetrokke raak, sal hierdie studieveld grootliks uit hulle hande raak en sal dit soveel moeiliker wees om weer nuwe rigting en insig daar te stel.

Bustad[1] haal uit die veeartseed die volgende aan "Ek wy myself plegtig, en die kennis wat ek besit, toe, tot voordeel van die gemeenskap". Hy vervolg deur te sê dat die veeartseed hiermee nuwe horisonne open in die lig van diere-ondersteunende-terapie, en beweeg van 'n eng tegnies bedrewe praktykvoering na 'n veel wyer professionalisme.

In nog duideliker taal verklaar Levinson dat sedert die verhouding tussen die mens en sy troeteldier verander het, die funksie van die veearts ook van gedaante moet verwissel. Die veearts kan hom nie langer alleen beperk tot die beskerming van die fisiese gesondheid van die gesin se troeteldier nie, hy moet ook betrokke raak by die geestesgesondheid van die gesin wie se dier hy behandel. Hierdie neiging is so onkeerbaar dat veeartse lede van geestesgesondheidsspanne sal word. Mense wie by geestesgesondheid betrokke is, werk nie alleen om emosionele afwykings in die minderheid te behandel nie, maar ook om emosionele gesondheid onder die meerderheid te behou en te versterk. As 'n belangrike deelnemende lid van 'n geestesgesondheidsspan, sal die veearts 'n soortgelyke doel moet nastreef. Namate die veterinêre professie hierdie nuwe fase in hulle praktyke nastreef en ontwikkel, sal die voordele hiervan vir almal in die gemeenskap duidelik word.[7].

7 Die voeding van geselskapsdiere

Namate daar van geselskapsdiere al hoe groter en meer prestasies verwag word, het die voeding van hierdie diere ook belangriker geword. Waar geselskapsdiere byvoorbeeld as kind-substituut aangehou word, het voeding ook uit so 'n oogpunt meer aandag gekry. Dit is noodsaaklik dat die veearts vrae ten opsigte van voeding sal kan beantwoord, ook ten opsigte van die spesiale behoeftes van geselskapsdiere. Dit het dalk ook tyd geword dat ons meer krities sal kyk na die voeding wat vir geselskapsdiere beskikbaar is. In produksiediere kan maatstawwe soos groeitempo, gewigstoename, produkgehalte en markpryse 'n goeie aanduiding van prestasie gee, maar by geselskapsdiere is dit meesal subjektiewe oordeel. Dit is dus veel moeiliker om uit te vind of 'n troeteldier se dieet op die *lang* duur, voor- of nadelig is. 'n Verdere vraag is of dit aanvaarbaar is dat 'n liggaam eenvoudig vol proteïene, energie en vitamine gepomp word sonder om eksterne faktore genoeg in ag te neem? Oefening en dieet gaan hand aan hand en die goed gevoerde troeteldier word dikwels nie die geleentheid tot gesonde oefening gebied nie. In die oorskakelingsproses van natuur ná verstedeliking was die voorsiening van goeie proteïen seker baie nodig. Maar nou dat verstedeliking 'n feit is, is al die proteïen en energie vir die agterplaas- en skoothond nog nodig? Daar is verskille in energie-behoeftes van gesteriliseerde diere, die groeiprosesse van klein en grootras honde, teelhonde en werkhonde, diere met verskillende temperamente, rasgeneigdhede, en diere wat skou.

Daar is ook vrae rondom die vorm van die kos, nat of droog, asook die soort vervaardigingsprosesse. Die voeding van weesdiere en die bepalende effek wat voeding op die eerste 6 - 10 maande van die dier se lewe het, is belangrik om latere probleme te voorkom.

Met geselskapsdiere waar daar 'n noue band tussen eienaar en dier bestaan, kom daar nog 'n belangrike aspek na vore waarvan 'n mens van kennis moet neem nl. antropomorfisme.

Dit is duidelik dat die veearts 'n enorme taak het om gesonde advies t.o.v. geselskapsdiere se voeding te gee. Waar voeding soms die onderliggende probleem van siektes is, behoort die veearts se kennis hom na 'n diagnose toe kan lei. Indien die probleem nie geïdentifiseer kan word en reggestel kan word nie, gaan die simptome bly bestaan ten spyte van talle kragtige chemiese en biologiese middels op die rak.

8 Rassekunde van geselskapsdiere

Dit is 'n groot verleentheid vir die veearts indien 'n eienaar met trots sy dier vertoon en die veearts dan nie die ras kan identifiseer nie. Nuwe rasse word steeds ontwikkel en dit is essensieel dat die veearts sal poog om op hoogte te bly. Die veearts word ook as kenner genader om advies te gee oor die aankoop van sekere rasse. As daar 'n gebrek aan rassekunde bestaan, kan sulke advies eenvoudig nie gegee word nie — veral nie as rasse vir spesifieke doeleindes aangewend word nie. Kennis ten opsigte van die rasse se uitwendige eienskappe, sowel as die ras se temperament, is dus van belang.

9 Beoordeling van genetiese raseienskappe

In hierdie afdeling word beoog om 'n gaping te vul tussen die basiese genetika asook genetiese afwykings, en die kliniese vakke. Dit gaan hier om die uitwys van normale genetiese eienskappe van elke ras, maar wat uiteindelik van kliniese belang kan wees. Die genetiese resultaat van ras-eienskappe wat kan bydra tot kliniese toestande moet dus geïdentifiseer word. So 'n identifikasie kan van onskatbare waarde vir die veearts wees, wat ras na ras op sy konsultasietafel moet ondersoek.

10 Die skou van geselskapsdiere, afrigtingswerk en gehoorsaamheidstoetse in honde

Die hond leen hom uitstekend tot intensiewe afrigting. Hierdie afrigting en gehoorsaamheidstoetse verskaf nie net ontspanning vir die mens nie, maar verskaf ook oefening aan grootrashonde wat dikwels te min geleentheid vir oefening in die stad kry. Die waarde van 'n hond word verder deur afrigting en gedissiplineerdheid verhoog.

Die persone betrokke by hierdie diere is gewoonlik in klubs saamgetrek en hierdie klubs betrek dikwels 'n veearts in 'n ere-posisie. Indien dan nie op daardie wyse nie, is die veearts in praktyk ook intens betrokke by hierdie prestasie-diere se gesondheid, voorbereiding en instandhouding van vorm. Byeenkomste soos skoue is egter die ideale geleenthede vir die veearts om sy betrokkenheid en belangstelling op 'n breër terrein as die konsultasiekamer te toon. 'n Mens kan sê dat dit die regte plek vir openbare skakelwerk is.

11 Geselskapsdiere as bedryf

Geselskapsdierbehoeftes soos slaaphokke, diere-klere, was-en-skeer, wasmiddels, hals- en leibande, kosbakke, voeding, dierehotelle, troeteldierwinkels, dierewelsynsorganisasies, diere by firmas en institusies, diere-tydskrifte en boeke, klubs, koop en verkoop van diere, advertensies en lisensies het daartoe gelei dat geselskapsdiere 'n geweldige bedryf geword het. In die VSA is bereken dat 78,3% van alle gelde vir veterinêre dienste aan geselskapsdiere ("pets") bestee word. As perde bygereken word, is dit 82,2%. Slegs 17,8% van die totale besteding aan veeartse, kom van die landbou[2].

As deel van hierdie bedryf, lewer die geselskapsdierveearts dus 'n belangrike bydrae. Veeartsfooie is dan ook dikwels 'n onderwerp van bespreking. Sonder om uitbuiters van die situasie te wees, sal die professie wel sy deel ontvang. Die goue reël wat egter gaan bepaal of die veearts se dienste in aanvraag bly, gaan nie wees hoe duur of hoe goedkoop hy kan diens lewer nie, maar of die geselskapsdiereeienaar gaan voel dat hy/sy waarde vir geld kry! Dit word weer bepaal deur effektiewe behandeling (sukses) tesame met behoorlike konsultasie (kommunikasie). As sukses en/of kommunikasie ontbreek, sal die eienaar voel dat die diens nie waarde vir geld was nie en behalwe vir noodsaaklikhede, sal geld eerder op ander aspekte van die bedryf spandeer word. Die veearts se wins kan bereken word in terme van *geld* sowel as *werksbevrediging* en *erkenning* deur die gemeenskap. Studente sal bewus gemaak moet word van die waarde van die veearts se bydrae in hierdie bedryf en sy werk ook in terme van die bedryf beoordeel.

12 Praktykvoering en bestuur

Praktykvoering is 'n aspek wat tot nou toe in die geselskapsdierveld agterweë gebly het. Dit is die gedeelte tussen die "belê" en "wins". 'n Mens kan dit opsom as belegging, bestuur en bevrediging (werks, finansieel en erkenning). Sekere beginsels in hierdie verband kan vir die student van groot waarde wees. 'n Mens ontken nie die individualiteit t.o.v. praktykbestuur nie, en elke veearts as bestuurder van sy praktyk kry die geleentheid om sy praktyk te reël na eie wense en behoefte. Maar intussen word die kompetisie hewiger. Sekere foute in praktykbestuur leer 'n mens net deur skade en skande as jy nie vooraf daarvan bewus was nie. Die idee is dus om sekere riglyne so te trek dat probleme in 'n jong praktyk eerder voorkom word as om dit later te probeer regstel. Praktyke moet in hierdie labiele ekonomiese tye so bestuur word dat dit steeds die bes moontlike diens lewer, sonder dat oorhoofse koste of lompe administrasie die praktyk knel. Kort paaie wat na afgronde lei en oorbehandelings moet vermy word. Die knapste veearts kan egter finansieel sukkel indien sy praktykbestuur nie op standaard is nie. Die eerste reaksie is om die fooie te verhoog, maar een fout kan nie deur 'n ander fout reggestel word ie.

Ter wille van oorlewing, is gesonde bestuursaspekte vir die veearts in praktyk 'n noodsaaklikheid.

SAMEVATTING

Uit die voorafgaande is die nuwe afdelings van die vak duidelik. Die gespesialiseerde spesies-benadering verhoed nie dat baie van die beginsels netso van toepassing is op 'n breër veterinêre terrein nie. Dat vandag se student by hierdie byvoeging tot die kurrikulum kan baat, behoort geen twyfel te laat nie.

Aan die begin van hierdie eeu het die land sekere belangrike vraagstukke op die weg van die veearts geplaas, wat opgelos moes word. Pioniers het na vore getree en wêreldroem verwerf op die wyse waarop hulle die land se veestapels en perdepopulasie kon red. Maar tye, omstandighede en behoeftes het verander en nuwe vraagstukke word na vore gebring. Die nuwe uitdagings aan die stadsveearts wat hom op geselskapsdiere toelê, mag dalk minder dramaties en opvallend wees, maar dit is geensins van minder belang nie — want dit raak die mens netso intiem. Die rol wat die geselskapsdier tuis vervul word grootliks onderskat omdat dit dalk te vanselfsprekend is. Geselskapsdiere funksioneer egter vandag as 'n belangrike onderdeel van die moderne mens se sosiale sisteem.

Met die herontdekking van die waarde van geselskapsdiere vir die mens sal daar weer pioniere uit die professie na vore tree om die uitdagings die hoof te bied. Interdissiplinêre studies het noodsaaklik geword en hierin is die veearts onvervangbaar. Netsoos die landbousektor 'n dringende appèl tot die professie gerig het aan die begin van die eeu, netso rig die stedelike-sektor vandag sý appèl tot die professie.

VERWYSINGS

1 Bustad L K, Hines L M, Leathers C W 1981 The human-companion animal bond and the veterinarian. The Veterinary Clinics of North America: Small Animal Practice 11: 807
2 Charles, Charles and Associates 1983 Excerpts of the Veterinary Services Market: Summary Report Part II. California Veterinarian 11: 36
3 De Groot A 1984 Preparing the veterinarian for dealing with the emotions of pet loss. Arkow P (Ed.) The Pet Connection University of Minnesota, Minnesota: 288
4 Herrick J B 1979 What is a profession. Veterinary Medicine, Small Animal Clinician 1: 12
5 Hopkins A F 1980 Our responsibility to understand client needs. Nordon News 3: 12-14
6 Levinson B M 1975 Pets and Environment. In: Anderson R S (Ed.) Pet animals and Society Bailliére Tindall, London: 17
7 Levinson B M 1965 The Veterinarian and Mental hygiene. Mental Hygiene 49: 320-323
8 McCoy P 1984 Student value systems in veterinary colleges. Journal of Veterinary Medical Education 11: 17
9 Odendaal J S J 1981 Die veearts, troeteldiere en psigoterapie. Tydskrif van die Suid-Afrikaanse Veterinêre Vereniging 52: 330-337

ABSTRACT

SAMEVATTING

LEPTOSPIROSIS AS A CAUSE OF "WHITE SPOT" KIDNEYS IN SOUTH AFRICAN PIG ABATTOIRS

The incidence of isolation of *Leptospira* sp. from the kidneys of slaughter pigs with "white-spot" macroscopic lesions was determined. Histology and general bacteriology were performed. *Leptospira pomona* was isolated from 19/21 kidneys showing macroscopic lymphocytic lesions. (Hunter, Pamela, Van der Vyver, F.H., Selmer-Olsen, Anne, Henton, Maryke M., Herr, S. & De Lange J.F., 1987. Leptospirosis as the cause of "white-spot" kidneys in South African pig abattoirs. *Onderstepoort Journal of Veterinary Research,* 53, 59-62 (1987).)

ABSTRACT

SAMEVATTING

OVINE HEPATOGENOUS PHOTOSENSITIVITY CAUSED BY THE PLANT *NIDORELLA FOETIDA*

Following a field outbreak of hepatogenous photosensitivity in sheep, an identical condition was reproduced experimentally by dosing 2 sheep with green, homogenized, *Nidorella foetida* material, collected from the camp where the outbreak occurred. This is a rare, unpalatable, aromatic shrub found in swampy places in the south-western Cape province. The main lesion was a hepatosis, characterized by peripheral coagulative necrosis and midzonal degeneration in the 2 acute cases and mild bile duct proliferation and hepatic regeneration in the more chronic field case. (Schneider, D.J., Green, Jennifer R. & Collett, M.G., 1987. Ovine hepatogenous photosensitivity caused by the plant *Nidorella foetida* (Thunb.) DC (Asteraceae). *Onderstepoort Journal of Veterinary Research,* 54, 53-57 (1987).)

ABSTRACT

SAMEVATTING

RESISTANCE OF SHEEP TO POISONING BY THE PLANT, *MARTICARIA NIGELLIFOLIA* DC.

The plant, *Matricaria nigellifolia,* was dosed to 2 sheep to reinvestigate whether or not sheep are suspectible to pushing disease ("stootsiekte"). Each sheep received a total dose of 370 g/kg of the milled, dried plant from a batch that had caused pushing disease in cattle when given at doses as low as 10 g/kg.

Neither of the sheep developed clinical disease over the trial period of 60 days, and necropsies revealed no macro- or microscopical lesions.

Sheep are apparently resistant to pushing disease, which supports previous findings. (Newsholme, S.J. & Kellerman, T.S., 1984. Resistance of sheep to poisoning by the plant. *Matricaria nigellifolia* DC. *Onderstepoort Journal of Veterinary Research,* 51, 277-278 (1984).)

THE IMPORTANCE OF ESSENTIAL FATTY ACID EVALUATION AND SUPPLEMENTATION IN FELINE DIETS

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ABSTRACT: Davidson B.C.; Traher C.S. **The importance of essential fatty acid evaluation and supplementation in feline diets.** *Journal of the South African Veterinary Association* (1987) **58** No. 1, 39-41 (En). Department of Medical Biochemistry, University of the Witwatersrand Medical School, York Road, 2193 Parktown, Johannesburg, Republic of South Africa.

The importance of essential fatty acids and their supplementation in the diets of obligate carnivores should be an area of concern amongst veterinarians. Certain species of the Felinae have been shown to be obligate carnivores as a result of fatty acid desaturase enzyme deficiencies. Modern dietary practices of processing and pelleting foods can destroy the essential fatty acid (EFA) potency of these foods. Supplementation of the diet with oils high in EFA should be considered, and the possibility of EFA deficiency amongst domestic carnivores should not be overlooked.

Key words: Essential fatty acids, essential fatty acid deficiency, cats, carnivores, dietary supplements.

INTRODUCTION

The obligate carnivore status of a number of animal species has been apparent for many years. However, until recently no evidence was available to pinpoint the exact nutritional requirement satisfiable only by the consumption of other animals. The obligate carnivore nature of a species, or its predecessors, most probably is not the result of such a requirement. Genetic changes in a non-carnivore would be extremely unlikely to provide, within the same generation, the killing instinct and necessary bodily equipment to fulfil the role of carnivore. It is much more likely that there was slow adaptation to becoming efficient carnivores; and that the loss of the ability to produce the necessary nutrients from plant precursors was secondary to this change. Such species, irrevocably, became obligate carnivores.

The first biochemical evidence for the obligate carnivore nature of the domestic cat was published in 1975[1], when cat liver was shown to lack the enzyme Δ-6-desaturase. Further evidence was published later by the same group[2]. This enzyme is responsible for the further desaturation of the polyunsaturated essential fatty acids obtained from plant material, and thus its lack deprives an animal of the ability to produce these longer chain more unsaturated fatty acids. Such fatty acids are, however, still required by the animal for the maintenance and construction of cell membranes, for cell-cell interactions, and for prostaglandin production, amongst other functions. Since these fatty acids are rarely produced by plants, but are produced by animal species without the enzyme lack, then they must be obtained from this source.

Similarly another feline species, the lion, has been investigated by the same group, and parallel results obtained[3]. More recently dietary modification of partially purified cat diets have indicated a requirement for the products of the next two desaturase enzymes in the cascade, Δ-5 and Δ-4-desaturases[4]. These fatty acids are found in large amounts in cell membranes, especially those of the central nervous system, and thus the lack of the enzymes or their production imposes a further dietary constraint on a species. The provision in the diet of only one or the other series of these fatty acids was shown not to be sufficient for the maintenance of normal growth and bodily function[4].

DIETARY POLYUNSATURATES

Polyunsaturated fatty acids are fatty acids with double bonds inserted into the hydrocarbon chain at regular intervals. Such fatty acids are normally named by counting from the methyl terminal or "ω" carbon atom. Therefore a double bond inserted between the carbon atoms ninth and tenth from the ω carbon is termed ω9. Plants are capable of inserting double bonds between the ω terminal and the ω9 carbon atoms, but animals are not able to perform this desaturation step. Animals do, however, require these double bonds for normal metabolism, and plant-derived polyunsaturated fatty acids with such double bonds are therefore "essential fatty acids" (EFA's) (Fig.1.). The two plant EFA's are linoleic acid (cis-C18:2ω6) and α-linolenic acid (cis-C18:3w3). These two compounds and their longer chain more unsturated derivatives cannot be interconverted by animals, thus there are two distinct families of polyunsaturated fatty acids (Fig. 2.).

The double bonds of most naturally occurring polyunsaturates are in the "cis-" stereoisomeric form (Fig. 3.). Many food manufacturing processes convert these "cis-"forms into the opposite or "trans-"forms, and such polyunsaturates do not exhibit EFA activity (Fig. 3.). Indeed many of them compete with EFA's, promote high cholesterol levels, interfere with prostaglandin synthesis, and block desaturase activity[5]. Similarly heat, often generated in food manufacture (rolling, pelleting, etc.), can destroy EFA in the lipid fraction of such foods, but without changing the apparent polyunsaturate level.

High levels of one desaturase reaction product will block the desaturation of other fatty acids of both the same family and the other family of polyunsaturates[6].

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Arachidonic acid (cis-C20:4ω6)[7] rich foods, such as meat, may well be inhibitory of the desaturase enzymes by this mechanism. The three series of prostaglandins are synthesised from dihomogamma-linolenic acid (cis-C20:3ω6), arachidonic acid (cis-C20:4ω 6), and eicosapentaenoic acid (cis-C20:5ω3). These hormones often have antagonistic effects when one series is compared to another[8], and thus the maintenance of a correct balance between them is essential. A high meat intake, providing metabolically high levels of the 2 series precursor (arachidonic acid, 2-8%), may produce an imbalance between the three different series of these compounds.

CLINICAL SIGNS OF EFA DEFICIENCY

Classic-essential fatty acid deficiency include the following[9-11] —

a) Impaired growth.
b) Dry, scaly skin.
c) "Wire" hair.
d) Hair loss.
e) Open skin lesions.
f) Non-healing sores.
g) Anal encrustation.
h) Amenorrhoea.
i) Infertility.
j) Haemorrhage.
k) Spermal abnormalities.
l) Self-mutilation.
m) Distorted birth sex ratio.
n) Death.

The lack of essential fatty acids may pose a serious threat to the life of an individual animal and to future generations. Several of these signs have been observed by various workers in both domestic cats and cheetahs[4,12], especially impaired growth, dry skin, hair loss, skin lesions, and amenorrhoea. Canines do not appear to lack the desaturase enzymes. However, some canines fed processed diets have developed skin erosions (Davidson, unpublished data). This may reflect inhibition of the desaturase enzymes by diet-derived agents, potentially leading to signs of deficiency.

CONCLUSIONS

In the light of the above, considerable care must be exercised when designing diets or manufacturing foods for consumption by carnivores. The elimination of or at least stringent control of all processes involving or generating heat, is of vital importance. A good balance between meat and other animal products (eg. fish, chicken, offal, etc.) must be maintained, and the supplementation of the diet with oils rich in polyunsaturates should also be carefully considered. Sunflower seed oil is extremely rich in linoleic acid (±70%), linseed oil is rich in α-linolenic (± 50%) as is the lipid from spinach (± 60%). Evening primrose oil, whilst very expensive commercially, is especially effective in preventing EFA deficiency, since it contains α-linolenic acid at ± 9%. This fatty acid is the product of a desaturase reaction and thus bypasses any possible inhibition of this enzyme. Chicken is relatively poor in polyunsaturates, but also has practically no arachidonic acid (± 0.5%) and thus may be useful as a means of counterbalancing excessive intake from red meat, whilst offal can provide some of the longer chain more unsaturated fatty acids (eg. docosahexaenoic acid, 4-10%). Fish is also an excellent source of such fatty acids, providing eicosapentaenoic acid at 8-20%, and docosahexaenoic acid at 6-33%. Fish oils, such as cod liver oil, halibut oil, etc. are also exceptionally good sources of such polyunsaturates.

If meat is to be the bulk of the diet of a carnivore, then the supplementation of such with either a mixture of fish, chicken, offal, and one of the above plant oils (these could also be alternated rather than mixed), or the addition of a plant oil:fish oil mixture (± 3:1) should be considered as essential. If solid supplementation, then at a level of 10% of the total diet; if oil mix, then

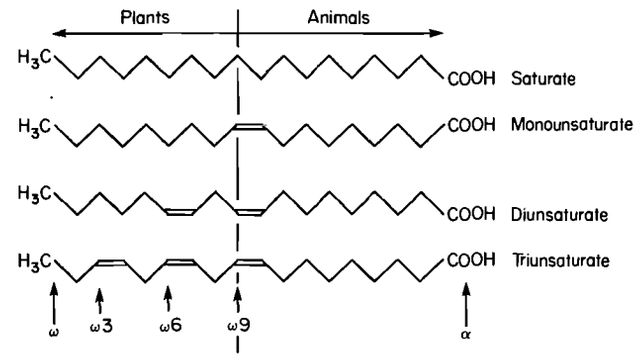

Fig. 1: The capability of animals and plants to desaturate fatty acids between specific carbon atoms.

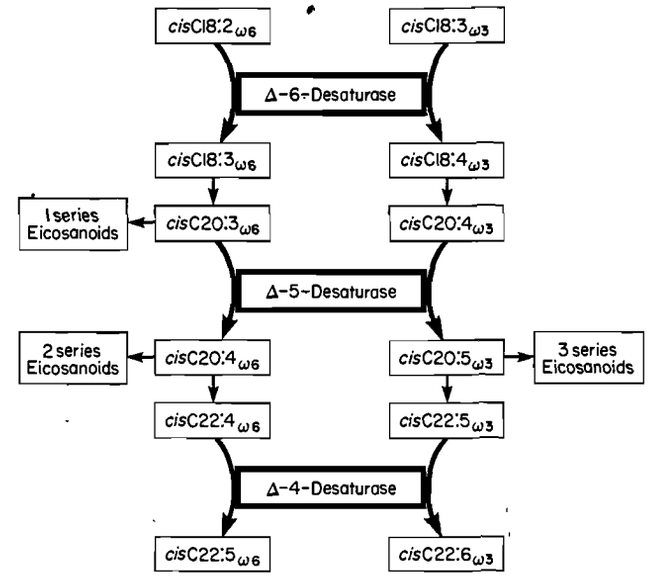

Fig. 2: The desaturation pathway of unsaturated fatty acids in animals.

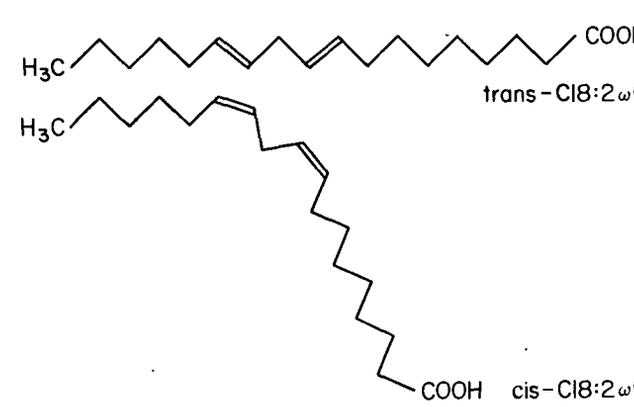

Fig. 3: The different isomeric configuration of fatty acid double bonds and the effects on the shape of the molecules.

2ml (half medicine spoon) per kilogram of diet. A regime along these lines, tailored to suit individual requirements and finances, will suffice to prevent EFA deficiency and associated infertility.

At the same time, because of the increasing popularity of pelleted foods (and their potentially high trans-polyunsaturate content), practising veterinarians would be well advised to consider EFA deficiency as a possibility amongst the domestic carnivores they are seeing during the course of their practice; and to advise the implementation of an oil mix supplement similar to that outlined above.

REFERENCES

1. Rivers J P W, Sinclair A J, Crawford M A 1975 Inability of the cat to desaturate essential fatty acids. Nature 258:171-173
2. Frankel T L, Rivers J P W 1978 The nutritional and metabolic impact of α-linolenic acid (18:3ω6) on cats deprived of animal lipid. British Journal of Nutrition 39:227-231
3. Rivers J P W, Hassam A G, Crawford M A, Brambell M R 1976 The inability of the lion, *Panthera leo*, to desaturate linoleic acid. FEBS Letters 67:269-270
4. Davidson B C, Els M, Kwinda S, Cantrill R C 1986 The effect on the growth of domestic cats of limiting the range of polyenoic fatty acids available from artificial diets. British Journal of Nutrition (in Press)
5. Cook H W 1981 The influence of trans acids on desaturation and elongation of fatty acids. Lipids 16:920-926
6. Garcia P T, Holman R T 1965 Competitive inhibitions in the metabolism of polyunsaturated fatty acids studied via the composition of the phospholipids, triglycerides and cholesterol esters of rat tissues. Journal of the American Oil Chemical Society 42:1137-1141
7. Crawford M A, Gale M M, Woodford M H, Casped N M 1970 Comparative studies on fatty acid composition of wild and domestic meats. International Journal of Biochemistry 1:295-305
8. Hassam A G, Willis A L, Denton J P, Stevens P, Crawford M A 1979 The effect of essential fatty acid deficient diet on the levels of prostaglandins and their fatty acid precursors in the rabbit brain. Lipids 14:78-83
9. Rivers J P W, Davidson B C 1974 Linolenic acid deprivation in mice. Proceedings of the Nutritional Society 33:48A
10. Sinclair A J, Fiennes R N T-W, Hay A W M, Watson G, Crawvord M A, Hart M G 1974 Linolenic acid deprivation in Capuchin monkeys. Proceedings of the Nutritional Society 33:49A
11. Rivers J P W, Crawford M A 1974 Maternal nutrition and the sex ratio at birth. Nature 252:297-298
12. Davidson B C, Cantrill R C, Varaday D 1986 The reversal of essential fatty acid deficiency symptoms in the cheetah. South African Journal of Zoology. 21:161-164

ABSTRACT

SAMEVATTING

AN EXPERIMENTAL MYCOTOXICOSIS IN SHEEP AND GOATS CAUSED BY *DRECHSLERA CAMPANULATA,* A FUNGAL PATHOGEN OF GREEN OATS

Field outbreaks of a syndrome of unknown aetiology associated with the grazing of green oats *(Avena sativa)* in the south-western Cape Province where characterized by diarrhoea, photosensitivity and death in goats and by diarrhoea and a reduction in milk production in cows. A phytopathogenic fungus, *Drechslera campanulata,* was isolated from conspicuous reddish-brown leaf spots on oat plants collected from both outbreaks.

Pure cultures on autoclaved maize of *D. campanulata* isolates from oat leaves implicated in both field outbreaks, as well as a Canadian isolate, proved to be highly toxic to ducklings, goats and sheep. Characteristic clinical signs of the fatal mycotoxicosis caused by *D. campanulata* culture material in goats and sheep were anorexia, apathy, diarrhoea and ruminal stasis. Photosensitivity, however, was not induced. Necrosis of the forestomach mucosa was the most characteristic gross pathological change. Histopathological findings included mild focal erosions to severe, diffuse, coagulative necrosis of the mucosa in the rumen, reticulum and omasum and congestion and haemorrhages in the abomasum. These results provide circumstantial evidence that green oat leaves infected by *D. campanulata* may cause outbreaks of a mycotoxicosis in grazing animals. (Schneider, D.J., Marasas, W.F.O., Collett, M.G. & Van der Westhuizen, G.C.A., 1985. An experimental mycotoxicosis in sheep and goats caused by *Drechslera campanulata,* a fungal pathogen of green oats. *Onderstepoort Journal of Veterinary Research,* 52, 93-100 (1985).)

ABSTRACT

SAMEVATTING

IN VITRO CULTIVATION OF *COWDRIA RUMINANTIUM*

Cowdria ruminatium was cultivated in a calf endothelial cell line after the cells had been irradiated at 45 & 90 GY. Another experiment in which the inoculum and non-irradiated cells were centrifuged together also yielded positive results. In some irradiated cultures, colonies of organisms could be demonstrated microscopically up to 70 days after the cultures had been inoculated with infected tick stabilate. The infectivity of cultures, even after 4 passages and 88 days post-inoculation*, was demonstrated by their intravenous injection in sheep. (Bezuidenhout, J.D., Paterson, Camilla L. & Barnard, B.J.H., 1985. *In vitro* cultivation of *Cowdria ruminantium. Onderstepoort Journal of Veterinary Research,* 52, 113-120 (1985).)

TO THE EDITOR — AAN DIE REDAKSIE

BLOEDNIER IN DIE SUIPLAM

Gedurende Oktober 1986 het daar in die Kaapse Middellande en Suid-Vrystaat bloednier voorgekom by Angoraboklammers en Merinolammers van ongeveer 3-4 weke oud. Die insidens was nooit hoër as 3% nie.

Bloednier in suiplammers is raar in ons land, maar is tog bekend in oorsese lande, aangesien dit reeds beskryf is in Sheep Diseases van Rue Jensen en Brinton L Swift. In die plaaslike gevalle was daar egter verskille in die simptomatologie wat vermelding verdien.

In al 7 gevalle was dit sterk, vinnig groeiende lammers in goeie kondisie wat nog aan hul moeders gesuip het en saam met hulle op besproeide lusernlande of aangeplante weiding geloop het. Party eienaars het slegs skielike vrektes gerapporteer, terwyl ander gesien het dat die lam opgeblaas was en buikpyn ondervind het voordat hulle gevrek het.

Nadoods was die abomasum gebars of redelik opgeblaas. Daar was min of geen tekens van peritonitis wat daarop dui dat die abomasum terminaal gebars het. Daar was heelwat melkstolsels en fyngekoude groen plantmateriaal in die abomasum en dit het 'n skuimerige voorkoms gehad. Die rumen was nog klein, maar het ook baie fyn groen plantmateriaal bevat. In party gevalle was daar kongestie van die dunderm. Verder was daar geen duidelike waarneembare makroskopiese letsels nie. Jensen et al. noem in hulle boek dat petechiale bloedinge kan voorkom in die subserosa van die dunderm, subepi- en endokardiaal, sowel as hidroperikardium met fibrineuse stolsels en longedeem en kongestie.

Bakteriologiese ondersoek van die organe van die lammers het oorheersend *Clostridium perfringens* tipe D organismes opgelewer in die abomasum en dunderm. Die kulture is by die N I V, Onderstepoort ondersoek en die tipering van die organismes is daar gedoen.

Normaalweg word meeste ooie gereeld geënt teen bloednier en een van die 7 eienaars het spesifiek genoem dat sy Angoraooie teen bloednier geënt was gedurende hulle dragtigheidsperiode. Sodoende kan die lam, volgens Jensen et al., ongeveer 5 weke lank passiewe immuniteit hê as dit betyds van die ooi se biesmelk drink.

Waarom daar vanjaar in een seisoen soveel gevalle voorgekom het is moeilik om te verklaar. Moontlik was toestande net baie gunstig vir die lammers om besonder vinnig te groei en selfs te veel te suip en te vreet. Dit is die reeds bekende vereistes vir bloednier. Verder is dit interessant dat Jensen et al. noem hoe die toestande minder gunstig gemaak kan word vir bloednier deur die lammers op 'n vroeër ouderdom te merk, kastreer en sterte af te sny. Die klein operasies veroorsaak net genoeg ongemak by die lammers om hulle voer- en melkinname te verminder sodat die bloednier-gunstige toestande vermy word.

VERWYSING

Jensen R, Swift B L 1982 Sheep Diseases 2nd edn Lea & Febiger, Philadelphia: 67

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ABSTRACT

SAMEVATTING

HEARTWATER IN ANGORA GOATS

Pathological lesions in untreated Angora goats infected with the Ball$_3$ strain of *Cowdria ruminantium* corresponded with those previously reported. A severe nephrosis was the most prominent pathological lesion in the animals treated after the 1st day of the febrile reaction. Renal ischaemia appears to be central to the pathogenesis of the kidney lesions. (Prozesky, L. & Du Plessis, J.L., 1985. Heartwater in Angora goats. II. A pathological study of artificially-infected, treated and untreated goats. *Onderstepoort Journal of Veterinary Research,* 52, 13-19 (1985).)

ABSTRACT

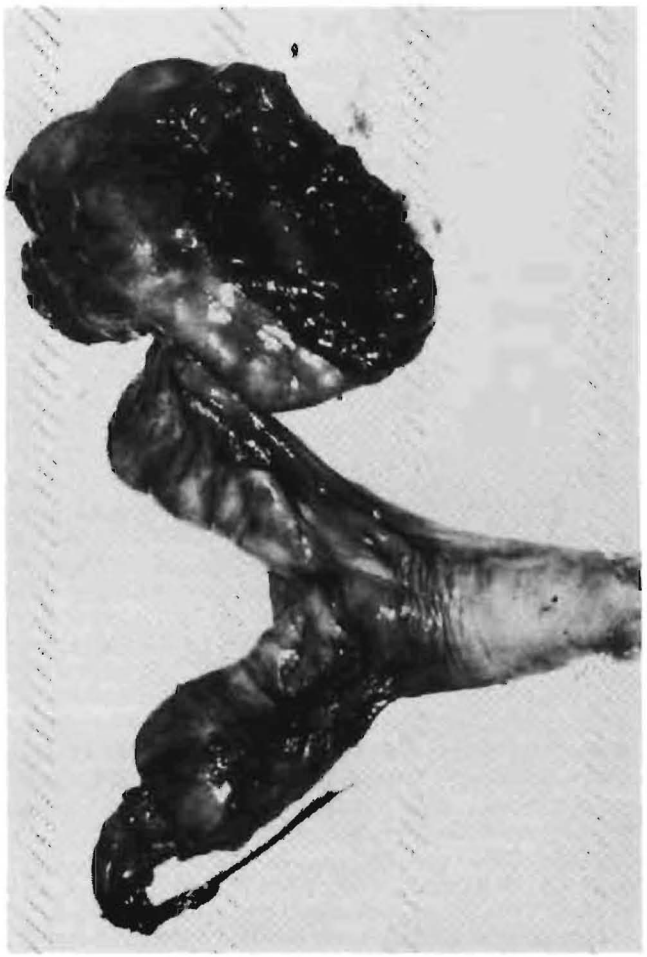
SAMEVATTING

RESISTANCE BY THE BLUE TICK *(BOOPHILUS DECOLORATUS)* TO THE SYNTHETIC PYRETHROID, FENVALERATE

The shaw larval test, in conjunction with adult tick immersion and stall tests, was utilized to confirm that a field strain of *B. decoloratus,* from Natal, is highly resistant to the ixodicide fenvalerate (Factor of resistance 4 744). This resistance developed over a reported 18 months of usage for cattle dipping. (Coetzee, Bridgette B., Standord, G.D. & Davis, D.A.T., 1987 Resistance by the blue tick *(Boophilus decoloratus)* to the synthetic pyrethroid, fenvalerate. *Onderstepoort Journal of Veterinary Research,* 54, 83-86 (1987).)

FATAL OVARIAN HAEMORRHAGE IN A HEIFER

A twelve-month-old Holstein heifer was found dead two days after being noticed to be in oestrus. During oestrus she had left the heifer group, jumped a fence, and joined the milking cow group. No signs of illness had been observed prior to her death and the heifer had not been inseminated or examined rectally. At post mortem examination the abdominal cavity was found to be filled with clotted blood. The source of haemorrhage was apparently the right ovary, which contained a 15 cm diameter blood-filled cavity which had ruptured. Histological examination of representative sections of the right ovary revealed only a small area in which the normal architecture had been preserved. Adjacent to the area of presumed haemorrhage was a zone of immature fibroblasts suggesting attempts at organization. The remainder of the sections consisted of fibrous tissue containing many small and large blood vessels. There was no suggestion of an infiltrating neoplastic mass, or of a primary inflammatory process. Although fatal haemorrhage following manual enucleation of corpora lutea has been recorded, spontaneous haemorrhage of this magnitude following oestrus in a heifer without any intervention must be regarded as extremely unusual[1,2]. It is possible that the heifer was roughly treated by the herd of larger and unfamiliar animals which she joined during her oestrous activity and that this provoked rupture of normal, or possibly abnormal ovarian structures, or that this represents an extreme case of spontaneous follicular haemorrhage[1]. The protracted existence of an abnormal endocrine milieu is suggested by the appearance of the uterus which was unusually large and flaccid for a heifer.



FATALE EIERSTOKBLOEDING IN 'N VERS

'n Twaalf maande oue Holsteinvers is twee dae nadat sy as bronstig waargeneem is, dood aangetref. Gedurende bronstigheid het sy die versgroep verlaat, oor 'n heining gespring, en by die melkkudde aangesluit. Geen siektetekens is voor haar dood by die vers waargeneem nie, en sy was ook nie rektaal ondersoek, gedek of kunsmatig geïnsemineer nie. Nadoodse ondersoek het getoon dat die buikholte met gestolde bloed gevul was. Die regter eierstok, waarin 'n oopgebarste, bloedgevulde holte met 'n deursnee van 15 cm aangetref is, was blykbaar die bron van die bloeding. Histologiese ondersoek het getoon dat slegs 'n klein gedeelte van die ovarium die normale argitektuur behou het. Langs die rand van die vermeende punt van bloeding was 'n laag jong fibroblaste, wat pogings tot organisasie aangedui het. Die weefsel het verder uit fibreuse weefsel met 'n groot aantal bloedvate van verskeie groottes bestaan. Daar was geen aanduiding van 'n infiltrerende gewas of van 'n inflammatoriese proses nie. Alhoewel fatale bloeding wel na enukleasie van corpora lutea beskryf is, moet spontane bloeding van hierdie aard na bronstigheid sonder enige ingrepe as hoogs buitengewoon beskou word[1,2]. Dit is moontlik dat die vers deur die groter, vreemde koeie, waarby sy tydens bronstigheid aangesluit het, rof behandel is, en dat ruptuur van die normale, of moontlik abnormale, eierstokstrukture sodoende veroorsaak is, of dat hierdie geval 'n erge spontane follikulêre bloeding verteenwoordig[1]. Die voorkoms van die baarmoeder, wat buitengewoon groot vir 'n vers was en 'n swak tonus gehad het, dui moontlik op 'n abnormale endokriene profiel.

REFERENCE/VERWYSINGS

1. Jubb RVF, Kennedy PC, Palmer N 1985 Pathology of Domestic Animals 3rd edn Vol. 3 Academic Press, New York: 311-314.
2. Roberts JS 1971 Veterinary Obstetrics and Genital Diseases 2nd edn Published by the author, Ithaca: 443-445.

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ABSTRACT

SAMEVATTING

PATHOLOGY OF A NERVOUS DISORDER (PUSHING DISEASE OR "STOOTSIEKTE") IN CATTLE CAUSED BY THE PLANT. *MATRICARIA NIGELLIFOLIA* DC.

Brains from 10 bovine field cases of pushing disease, a nervous disorder caused by the plant, *Matricaria negellifolia*, were examined by light microscopy. Moderate to marked encephalitis, characterized by predominantly perivascular microgliosis, perivascular lymphocytic infiltrates and reactive changes in astrocytes, was present in all the brains. The lesion was concentrated in the white matter throughout the forebrain and midbrain.

Dried, milled *M. nigellifolia* was dosed to 6 steers. Clinical signs of pushing disease, which included docility, clumsiness and pushing against objects, appeared abruptly in 5 of the steers after a latent period that varied from 16-44 days. The lowest total dose of plant that proved toxic was 10 g/kg. The length of the latent period appeared to be related inversely to the total dose. Encephalitis, which was similar in nature and distribution to those in the field cases, was demonstrated in the 5 affected steers. The lesion was minimal in the brain of the steer that did not develop pushing disease.

The cerebral lesion is suficiently consistent and distinctive to be useful in establishing a diagnosis of pushing disease. The perivascular distribution of microgliosis suggests that the site of the toxic insult is the cerebral vasculature. Botanical information is presented. (Newsholme, S.J., Kellerman, T.S. & Welman, Wilhelmina G., 1984. Pathology of a nervous disorder (pushing disease or "stootsiekte") in cattle caused by the plant, *Matricaria nigellifolia* DC. (Asteraceae). *Onderstepoort Journal of Veterinary Research*, 51, 119-127 (1984).)

BOEKRESENSIE — BOOK REVIEW

MEAT HYGIENE

J F GRACEY

Eight edition. Bailliere Tindall, W.B. Sauncers, 1 St Annes Road, Eastbourne, East Sussex BN21, 3UN, England, 1986 pp X and 517. Illustrations 69 Tables 39. Price £25-00 (ISBN 0 7020 1162 2).

This edition replaces "Thornton's Meat Hygiene" by J F Gracey which appeared in 1981 and which itself was a thorough revision of "Textbook of Meat Inspection" by H Thornton (first published in 1949) incorporating scientific and technical advances and an update on legislation. Further consideration has been given to environment considerations and the scope of the book has been widened, with more insight into modern agricultural practices, an improved coverage of the role of the poultry industry, sufficient emphasis on the important animal welfare aspects in the production of a quality end product and due regard being paid to the demands of the consumer in the section on meat composition and quality. Two new chapters have been added — one on metabolic diseases and nutritional deficiencies and one on diseases due to extraneous poisons: an emerging problem in meat hygiene.

The new edition has reduced figures and illustrations from 106 to 69 and increased tables from 20 to 39. The elimination of many photographs of specimens is regretted, but overall the photographs, figures and illustrations have been improved. As in the previous edition, there are four plates of coloured photographs, but in this edition six larger photographs of an excellent quality are included on each plate to replace the previous four per plate.

Editorial improvements are pleasing — the text is fractionally larger, tables are not split between pages, basic spelling mistakes have been corrected and many open blank areas eliminated or reduced in size. A useful introduction is the list for further reading supplied after important chapters and relevant UK and EEC legislation references. This has eliminated a dull appendix which has been replaced by some useful ideas on meat inspection recording. References are included after each chapter, many of which are recent work in the relevant sphere.

This new book is a most useful edition for both the student in meat hygiene and the veterinarian in Veterinary Public Health with its additional information on chemical residues, its introduction to analytical epidemiology, a more concise approach to judgements in the section on Pathology, an extensively reviewed chapter on Bacterial, Viral and Fungal Diseases and the introduction of life-cycle diagrams in the more important parasitic diseases. If anything, more useful than previous editions in Southern Africa and recommended.

C M VEARY

DISEASES OF SWINE

A D LEMAN, BARBARA STRAW, R D GLOCK, W L MENGELING, R H C PENNY AND E SCHOLL (EDITOR)

6th Edn. Iowa State University Press, Ames, Iowa, 50010; 1986 pp xiii and 930, illustrations 382 and numerous tables, Price £75.00. (ISBN 0-8138-0441-8).

The latest edition of what has become "the best single source of information on swine diseases" is an excellent co-operative effort by 99 contributors to document the ever-increasing new information on swine medicine. The book retains its strong North American character with 70 contributors having U.S. or Canadian addresses. Missing from among the 15 distinguished U.K. contributors are any representatives of the Cambridge *Mycoplasma* research group, to your reviewer's regret.

Extensive updating has been restricted to 100 additional pages compared to the previous edition, and includes 4 new chapters plus considerable rewriting elsewhere. No less than 18 newcomers have been involved here and the results are excellent. Particularly welcome are the increased emphases placed on herd medicine and production efficiency, coupled with an extensive database to guide the inexperienced. This recognition of the primary veterinary input to the pig industry greatly increases the book's value as a working manual.

The new chapters include one on differential diagnosis of swine diseases arranged in 35 useful tables plus a welcome chapter (not listed in the table of contents) on atrophic rhinitis clearly explaining recent research results in this area. All in all the sixth edition clearly embodies the changing concepts of modern swine medicine and is a worthy successor to its long line of predecessors. It is strongly recommended to all with an interest in this field.

R K LOVEDAY

0038-2809/87/01/0047-0051

HEARTWORM DISEASE IN DOGS AND CATS

CLARENCE A. RAWLINGS

W.B. Saunders Company, Philadelphia. 1986 pp vi and 329, numerous illustrations, Price £29-95 (ISBN 0-7216-1221-0)

Professor Rawlings's book on heartworm disease is a comprehensive account of the pathophysiology, clinical signs, evaluation, diagnostic approach, treatment and pathology of heartworm infestation in the dog and cat. The book is essentially clinically orientated and contains virtually no information on epidemiological factors such as worldwide occurrence of the disease and geographical distribution of possible vectors. Heartworm disease is perhaps mainly of academic interest (only one case has been reported in the literature; most suspected cases turn out to be *Dipetalonema reconditum*) and the book would therefore be of very little practical value to veterinarians in the Republic of South Africa.

J VAN HEERDEN

NOTES ON CANINE INTERNAL MEDICINE

P G G DARKE

2nd Edn. Wright, Bristol, 1986 pp xii and 286, figures 13 (ISBN 0 7236 0887 3) Price not given

"Notes on Canine Internal Medicine" is a concise presentation of mainly the clinical signs, differential diagnosis, diagnostic approach and treatment of medical conditions of the respiratory, cardiovascular, gastro-intestinal, urogenital and endocrine systems. The differential diagnoses and diagnostic approach to syndromes such as polyuria, coughing and weight loss are presented in the same manner.

The book allows for quick reference and is aimed primarily at a differential diagnostic approach. Brevity does however not always allow for clarity, and statements like: "The presence of cardiac disease is easily detected. However, much cardiac disease may not be very relevant in pet dogs", and "The presence of cardiac disease is readily detected by auscultation" are open to misinterpretation.

Locally-important conditions like babesiosis, ehrlichiosis and verminosis receive extremely limited attention. Babesiosis is not even mentioned as a cause of splenomegaly and faecal flotation does not feature as a diagnostic test in the investigation of thinness/weight loss in the dog.

The numbering system and use of boldface print as headings to sections and subsections is at times confusing; for example in the listing of pituitary endocrine disorders. It is also a pity that some of the typographical errors of the first edition are repeated in this edition eg. *Dirofilaria* imitis instead of *Dirofilaria immitis*.

"Notes on Canine Internal Medicine" is however a useful aid to the promotion of an intelligent approach to the diagnosis of canine internal disease. The book should fulfill the author's wishes that it may help to save lives or protect against prolonged investigation or inappropriate therapy.

J VAN HEERDEN

CLINICAL AVIAN MEDICINE AND SURGERY

G J HARRISON AND L R HARRISON (ED)

1st Edn. W B Saunders Company, Philadelphia, P A 19105, 1986 pp XVIII and 717, Figs 358, Tabs 87 ISBN 0-7216-1241-5. Price not quoted.

This compilation of 53 chapters by 33 contributors is set out into 8 major sections. Section 1 deals with basic husbandry, the behaviour of a captive bird and modifications to its behaviour. Section 2, "The Normal Bird", describes clinical aspects of anatomy and physiology. Section 3 and 4 deal with the clinical examination and evaluation of the patient, diagnosis, differential diagnosis and diagnostic procedures. The chapters involving laboratory techniques and examinations are amongst the most comprehensive the reviewer has seen. The problem — specific defined data base section and the differential diagnosis section is presented in an easy to use tabulation format. The radiographs are of high standard, reflecting the clinical approach of the book.

Section 5 concerns therapy and discusses avian immune mechanisms, the pharmacology of antibiotics and emergency medicine. Section 6, "Diseases", succinctly discusses the major causes of pathology. Section 7 deals with anaesthesiology and selected surgery of birds, with particular emphasis on micro-surgery and the use of an operating microscope. Section 8, Aviculture, discusses the management of large collections, laparoscopy and reproductive and pediatric medicine.

The integration of the various sections and chapters is excellent, and the information supplied is detailed and generally up to date, with comprehensive product information. A reference or reading list is included at the end of each chapter.

The book deals largely with psittacine birds, and gives relatively little information on other avian groups which are kept as pets or semi-domesticated birds. However, most of the information given can be extrapolated to include these groups. A standardised use of either S.I. or Imperial units in the Therapeutic table has not been made. Indiscriminate mixing of doses using both sets of units has occured, which becomes confusing and necessitates recalculation into applicable units by the users of the table.

These objections aside, the book sets out to achieve a clinical approach to avian medicine and surgery, and succeeds admirably. The abundance of figures (black and white photographs and diagrams) and tables make this an easily accessible text, for practitioner and student alike. This book is to be highly recommended for all interested in avian practice.

R E J BURROUGHS

CURRENT VETERINARY THERAPY IX SMALL ANIMAL PRACTICE

ROBERT W KIRK (ED)

W.B. Saunders Company, West Washington Square, Philadelphia, PA 19105. 1986 pp xxx and 1346, numerous tables and illustrations. Price not given (ISBN 0-7216-1500-7)

"Current Veterinary Therapy IX, Small Animal Practice" is a collection of contributions from more than 240 authors from different continents of the world, covering a wide variety of subjects ranging for example from regurgitation in snakes to wheat-sensitive enteropathy in Irish Setters. The setting is formidable and it is hardly possible to do justice to the text in the scope of a short review.

Consulting editors were appointed for each of the 14 Sections and Appendices contained in the book. The titles of these various sections do not, however, always reflect their contents and it is almost with surprise that one finds for example a chapter on nuclear imaging under "Special Therapy" and a chapter on salicylate toxicity under "Immunology, oncology and haematology". It soon became clear to me that one has to study the contents pages very carefully if you want to appreciate the full scope of what is offered by this latest edition of Current Veterinary Therapy.

The first section, "Special Therapy", deals not only with specific therapeutics such as hemapharesis and the treatment of adverse reactions to radiopaque contrast agents, but also with specific and sometimes very sophisticated diagnostic techniques such as nuclear imaging and ultrasonography. South Africans will unfortunately not benefit by the listing of nuclear imaging referral centres in North America. This section also includes interesting chapters on the prophylactic use of antimicrobials, the control of nosocomial infections, algorythms, acupuncture and therapy by referral. Chapters in this section, as elsewhere in the book, vary, in my opinion, considerably in the detail by which the relevant material is handled. Most chapters, however, allow for easy reading, sometimes even with a touch of humour: (on therapeutic use of vitamins) "Vitamin supplementation only enhances the value of the animal's urine".

Section two deals with poisonings in small animals and includes chapters on prevalence, information resources, clinical signs, treatment, adverse drug reactions and their treatment, drug-related diagnostic errors as well as a chapter on contaminated food and water. The tabulated form in which information is presented allows for quick reference and optimal use by the busy practitioner. Chapters on specific toxicological substances like lead, rodenticides and aspirin are also included.

The third section is on respiratory disorders which begins with an account of non-immunologic and immunologic defense mechanisms of the lung and continues with pathological reactions within the lung, cytological diagnosis of respiratory disorders and radiography of the chest. The section further includes detailed chapters on specific therapeutics such as bronchodilator therapy and ventilatory support as well as chapters on specific entities such as hypersensitivities, chylothorax and bronchitis.

The section on cardiovascular diseases starts with the mnemonical ("DAMN IT") presentation of the aetiology of cardiovascular disease in dogs and cats. The section contains a lot of information on the therapy of cardiovascular disorders, with chapters on positive inotropic drugs, vasodilator therapy, captopril, calcium antagonists and anti-arrhythmic drugs. Systemic hypertension, which up to now has essentially been a non-entity amongst most veterinarians, is brought to the attention of clinicians and renal disease is mentioned as the most frequent disorder associated with hypertension. Very useful chapters on myocardial conditions in the dog and cat, for example Boxer cardiomyopathy, are included.

Section five deals with immunology, oncology and haematology. The chapters on immunology present a broad outline of mechanisms of immune-mediated disease, immunodiagnostic tests, feline leukemia virus as a cause of severe immunosuppression in cats, and various hypersensitivities. In the capters on oncology, the veterinarian is introduced to various concepts, amongst others that of precancer by way of a short description of some precancer-cancer sequences in small animals. The selection and use of chemotherapeutic agents by private practitioners should be facilitated by the tabulated presentation of these drugs and their adverse effects. In the section on haematology, the veterinarian is introduced to the abbreviation "PIVKA" which, as the author claims: "once understood," the veterinarian will undoubtedly desire to apply a test to determine PIVKA's presence".

The section on dermatology is a bonus for those who do not wish to invest in a separate book on skin diseases. Section seven on opthalmologic diseases includes chapters on opthalmology of reptiles and caged birds. Several tables on infectious causes of eye problems allow for quick reference. The next section includes more exotics: various aspects of veterinary medicine in various species like snakes, turtles and tortoises are dealt with. The subjects are too numerous to list but the practising veterinarian faced with a broken shell in a tortoise or toothache in a rabbit should try this book: one is likely to find some useful information.

The section on neurology and neurologic disorders, although far from replacing a textbook on neurology, contains chapters for example on feline dysautonomia which most South African practitioners may not be familiar with. Most local veterinarians would probably also recognize the "Generalized tremor syndrome" as Maltese Shakers.

The chapter on endocrine disorders is begun with a presentation on principles of glucocorticoid therapy in non-endocrine disease which should be read by all veterinarians. It should help to curb the unnecessary and excessive use of depot forms of glucocorticoids. The chapters on feline hyperthyroidism and canine pheochromocytoma should help to increase awareness of these conditions locally. Feline hyperthyroidism is reported as being "a common and extremely important disorder of cats". This section also includes an informative chapter on the diagnostic approach to lipaemic serum.

Most local veterinarians will probably find the section on infectious diseases wanting: the chapter on canine babesiosis for example includes such statements as "cases with mild to moderate anaemia may only require iron supplementation", and "cases with concurrent ehrlichiosis are difficult to treat, particularly if only babecidal drugs are used".

Assessment of renal function is covered in several chapters. The chapters on medical dissolution and prevention of struvite uroliths should be read by all veterinarians who still feel that detection of a urolith is an indication for surgical intervention.

The section on reproductive disorders includes a clear representation of major reproductive events in the bitch, a diagnostic approach to reproductive disorders, prostaglandin therapy and the freezing of canine semen.

The appendices present tables on a variety of measurements like haematological data, blood chemical parameters, conversion of weight to body surface area, electrocardiographic data, recommended nutrient allowances and approximate dosages of common drugs.

"Current Veterinary Therapy IX" covers a vast scope of subject material. The practising veterinarian could only benefit by reading and using this contribution to continuing education. The book is definitely not going to fulfill all the needs or answer all the questions but it may in many instances go a long way towards it. I have no hesitation in strongly recomending this publication to all veterinarians in small animal practice.

J VAN HEERDEN

THE CAMEL IN HEALTH AND DISEASE

ANDREW HIGGINS (ED.)

1st Edn. Baillière Tindall, 1986 pp VIII and 168, numerous photographs and tables. (ISBN 0-7020-1167-3)

This compact, up to date, scientifically sound guide to all aspects of camel care must be welcomed by every veterinarian dealing with camels — whether in the field or in zoological gardens around the world. Eight of the ten chapters were first published as papers in the British Veterinary Journal from 1984 - 86.

Experts in their particular field were responsible for reviewing the up to date available literature and to present data. The chapter headings cover the following: husbandry and management, clinical examination and restraint, protozoal diseases, helminths, extoparasites and their control, infectious diseases, reproduction, surgery, anaesthesia and the management of camels under intensive husbandry.

Not since the publication in 1927 of Leese's classic work on the camel in health and disease has such a useful, scientific and practical book become available.

All honour to the editor and his 14 contributors.

MALIE M S SMUTS

A COLOUR ATLAS OF EQUINE PARASITES

DENNIS E JACOBS

1st Edn. Gower Medical Publishing Ltd, 34-42 Cleveland Street, London WIP 5 FB, U.K. 1986 pp VII + 172 Figs-199 Price £19-95 (ISBN 0-7020-1175-4)

This colour atlas is a dream come true for the veterinary student, horse practitioner, parasitologist and horse lover. In an easy practical style the parasites of horses become alive. The photographs are excellent and were contributed by colleagues around the world.

The parasites are grouped into 8 chapters according to their habitat: Dipteran parasites of the skin, non-Dipteran parasites of the skin, the blood, the stomach, the small intestine, the large intestine, the liver and the lung and other tissues.

The book is in a handy format (200x150)mm which fits easily into a coat pocket to assist you in arriving at the correct diagnosis.

This atlas, in the words of the author is not intended to replace the standard textbooks but rather to bring the detailed factual information contained therein alive.

F S MALAN

VETERINARY NEUROLOGY

J E OLIVIER, B F HOERLEIN and J G MAYHEW'

W B Saunders Company, Philadelphia, PA 19105 1987, pp xvii and 554, numerous illustrations and tables, Price not given (ISBN 0-7216-1314-4)

As stated in the preface, Veterinary Neurology is built on the foundation of Hoerlein's Canine Neurology. A major change is the inclusion of material on all domestic animal species. Apart from the editors which are well recognised specialists in the field of neurology, the book also includes contributions by 15 other veterinary scientists.

Veterinary Neurology is a well-presented attractive dissertation on medical and surgical aspects of neurology. Although the major emphasis is still on the dog, the book includes information on all domestic animals. It does for example include well-illustrated information on spinal cord problems in horses.

The text starts with an introductory chapter which includes amongst others interesting figures on the incidence of neurologic disease in the various species.

The next chapter deals with the fundamentals of the clinical diagnosis of neurologic disorders: a basic understanding of morphology and function, and a sound diagnostic approach to enable the clinician to localize the lesion in the nervous system and/or to arrive at an aetiological diagnosis.

Several chapters are devoted to the presentation and discussion of diagnostic aids which range from procedures within reach of practitioners such as cerebrospinal fluid analysis and survey radiographs to more sophisticated techniques such as cerebral angiography, scintigraphy, computer analyzed tomography, electroencephalography, electromyography, recording of evoked potentials, tympanometry, cystometry and uroflowmetry. Some of these diagnostic tools are also available at referral centres in the Republic.

Brief accounts of clinical diseased as caused by specific aetiologica agents are presented in several chapters. Disease conditions are grouped together on the basis of aetiology. Each chapter is backed up by an extensive list of referrences. Seperate chapters are devoted to seizures, brain and spinal cord injuries, intervertebral disc disease, disorders of micturition, neuro- and myopathies.

The final chapters of the book are devoted to therapeutics: medical and surgical. Proposed medical treatment of central nervous system oedema, pain and infectious are substantiated by brief discussions of the mode of action of proposed drugs. The presentation of the principles of anaesthesia, patient positioning and monitoring is followed by detailed and well-illustrated accounts of amongst others, the techniques of hemilaminectomy, fenestration, vertebral stabilization, craniotomy and peripheral nerve surgery in small animals and horses.

The book is concluded with an appendix which has been designed as a quick-reference system to assist the reader in identifying the diseases most likely to cause a particular presented clinical sign.

Although the book is unlikely to solve all the neurological problems that a clinician may encounter, it should go a long way towards fostering a sound diagnostic approach and stimulating an interest in the fascinating field of veterinary neurology. The book should be useful to both small and large animal practitioners, generalists and specialists. If you have never bothered to buy a textbook on neurology, this text would definitely complement your private collection.

J. VAN HEERDEN