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TYDSKRIF VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING

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BERIA

ADDRESS **VOORLESING**

VETERINARY EDUCATION

J.M. ERASMUS*

Mr President, honoured guests, colleagues, ladies and gentlemen, I have been requested by the congress organisers to address you on veterinary education as it affects the Directorate of Veterinary Services which functions within the Ministry of Agricultural Economics and Marketing.

In this talk I will give you a summary of our responsibilities, the value of the animal industry we are involved with and the educational requirements for veterinarians in the employ of my directorate as well as for those private practitioners active in this field.

The Directorate of Veterinary Services is responsible for the administration of the Animal Diseases and Parasites Act, No. 13 of 1956 and the Animal Slaughter, Meat and Animal Products Hygiene Act, Number 87 of 1967. This has lead to the establishment of a subdirectorate for animal health and another one for meat hygiene. The Animal Diseases Act provides for the control of animal diseases and -parasites, for measures to promote animal health and for matters connected therewith whereas the Meat Hygiene Act provides for the maintenance of proper standards of hygiene in the slaughtering of animals and the handling of meat and animal products and for the prevention of the transmission of disease to humans and animals by such products. It also provides for the prevention of cruelty in the slaughtering of animals at abattoirs and the control of imported meat.

In the subdirectorate of animal health a laboratory diagnostic service has developed to the extent that it can be regarded as a subdirectorate of its own although this has not been officially approved yet.

We have in our employ 130 veterinarians excluding myself and my two deputies. Of these 63 are involved in animal health, 39 in meat hygiene and 28 in diagnostic laboratories. We also have contracts with 413 private practitioners involving them in our animal health schemes and another 75 colleagues are authorised to act on my behalf in meat hygiene matters. This adds up to a total of 621 veterinarians involved in various aspects of regulatory service aimed at an industry of which the gross value of animal products is R4 394 089 000 produced on 70 000 farm units. The value of exported animal products only, needing veterinary certification was R660 000 000 in 1984. In our laboratories we handled: 1 400 141 serological tests, 129 944 somatic cell counts, 149 934 other tests and 16 017 post mortems of which 12 986 were on poultry carcases.

The carcass value of animals (red meat) slaughtered last year (1985) was R1 806 161 100. Officials of the subdirectorate of meat hygiene and authorised veterinarians were involved in the inspection of 85% of these carcases with a market value of R1 535 236 900. I have quoted these figures to illustrate the responsibility carried by a large group of veterinarians in our profession and the importance for veterinary educational institutions to supply suitably qualified manpower to deal with this task. From what I have said earlier it is clear that our veterinary operations are spread over a wide field. We therefore require veterinarians who have received a broad-based training during their intern years. As far as my requirements are concerned, I am reasonably satisfied with the graduates produced by the faculty at Onderstepoort and I am sure the same will apply to graduates from the faculty at Medunsa when they become available, although I would like to see undergraduates receive some training in communication, the principles of business management and data processing.

Until recently we encountered problems with graduates leaving college with insufficient practical knowledge in handling herd health schemes, diagnostic procedures and hygiene management in abattoirs. After consultations with the faculty concerned and through mutual cooperation the situation has improved to a certain extent. The recent establishment of a course in epidemiology will also be of great value to the profession and our directorate in particular.

It is also interesting to note that since we began exposing students to laboratory diagnostics and abattoir management during their two week practical training with my directorate more graduates have shown an interest in entering these fields. They began to realise that the veterinarian in the laboratory is not bench bound but he experiences tremendous job satisfaction in that he can investigate a problem on a farm, examine his specimens in the laboratory, make a diagnosis and return to the farm with advice.

The same applies to the veterinarian in meat hygiene who now realises that inspection and hygiene control involves more than knowing where the inspection sites on a carcass are.

During the two week period we only have time to expose students to the most salient features and try to stimulate their interest. With the limited time available we can do no more.

In order to prepare graduates for their fulltime careers in state service we resolve to inservice training but as far as contracted and authorised colleagues are concerned, we are not equipped to present them with specialised courses. I also regard it as not one of the functions of my directorate. The most we can do is to offer crash courses lasting a few days each as we have now been doing in meat hygiene in conjunction with our faculties. This must be regarded as a temporary measure to fill a gap until our educational institutions can offer

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post graduate courses on the subjects concerned such as epidemiology, microbiology, meat hygiene etc., as well as species orientated training. What I have in mind is a short course where a candidate can receive specialised training over a limited period of time without necessarily obtaining a diploma or degree. These courses can be presented on a decentralised basis so as to enable practitioners and state veterinarians to attend them more freely.

From my side I will fully support such training. At this moment one of my state veterinarians is completing a specialised course on meat hygiene in Bristol UK., and it is going to cost my organisation R60 000. With the modern training facilities available at the new Pyramid abattoir equivalent courses can be presented by our own faculties.

While on meat hygiene, it is important to note that local authorities are required by law to employ veterinarians to supervise meat inspection and hygiene in abattoirs owned by them. For various reasons we exempted them from this requirement in the past. These exemptions are now being withdrawn and it is up to our colleagues to qualify themselves in this field. Some local authorities are already arguing that the meat inspector knows more about meat inspection and hygiene than the veterinarian. It is therefore up to the veterinarian to prove himself in this situation.

In the diagnostic laboratory I require veterinarians with specialised knowledge on various subjects such as epidemiology, pathology, bacteriology, toxicology etc. in order to advise colleagues and animal owners on problems encountered. Fortunately this type of service is available from some of our laboratories. These colleagues have acquired their knowledge through experience and selftuition. The availability of formal courses on the subjects concerned here will fulfill a great need. The same applies to state veterinarians and rural practitioners concerned with epidemiology of which our animal health schemes form an integral part.

In conclusion I would like to summarise that 133 fulltime state employed veterinarians and 488 contracted and authorised private practitioners are involved in various animal health services to an animal product industry with a gross value of 4,4 thousand million rand. These veterinarians are receiving an acceptable basic training and I acknowledge the willingness of faculties to accommodate our needs within their limitations. Serious consideration must be given to the establishment of formal specialised post graduate courses on various subjects on a decentralised basis. The contents and format of such courses must be well planned by our faculties in liaison with the veterinary groups concerned.

A responsibility lies with all of us, from the veterinary teacher to the practitioner in whatever field he may be active, to contribute towards the well being of the peoples of our country. Let us ensure that we are suitably qualified to undertake this task.

ADDRESS **VOORLESING**

AFRIKAANS EN DIE VEEARTSENYKUNDIGE*

J.M.W. LE ROUX**

"Het is opmerkelijk dat in deze strijd voor de vervlaamsing van het diergeneeskundig onderwijs, de bijdrage vanwege sommige buitestaanders zeker niet geringer, soms zelfs hardnekkiger was dan deze vanuit het beroep, de beroepsorganisatie en sommige academische kringen. Het is zeker niet overdreven te beweren, dat deze vervlaamsing sommige niet-dierenartsen nauwer aan het hart lag dan een groot gedeelte van de toenmalige dierenartsen die sich in het burgerlijk kringetje der franstaterende notabelen van hun dorp zeer gelukkig voelden en dan ook geen behoefte hadden aan de vervlaamsing". Hierdie wrange uitspraak is in 1984 by geleentheid van die herdenking van 50 jaar Nederlandstalige veeartsenykundige onderwys aan die Rijksuniversiteit Gent gelewer. Alhoewel dit nie daar oor die vaktaal gegaan het nie is dit nogtans van toepassing op die volslae gebrek aan georganiseerde denke, geesdrif en handeling van die kant van die Fakulteit Veeartsenykunde as 'n akademiese instelling om die Afrikaanse veeartsenykundige vaktaal te bevorder. Ander doen dit vir ons. Ek huiwer om van 'n agterstand te praat want die dokumentasie wat beskikbaar is, is nouliks meetbaar. Daarmee wil ek nie die bydraes van individuele fakulteitslede vir dit wat sober en helder en eerlik in ons taal is verkleineer nie. Hulle pogings, hulle eensame stryd, is 'n onbaatsugtige diens ten bate van die geskrewe Afrikaanse vaktaal.

Die hoofgedagte van my bydrae is: skryf en publiseer in Afrikaans, met die klem op publiseer. As 'n mens dit wat jy skryf sigbaar wil maak en bekend stel op gevaar af dat dit kritiek kan uitlok en aanvegbaar is, begin jy ernstig nadink. Die gesindheid moet ook reg wees, naamlik die begeerte en behoefte om in jou daaglikse handel en wandel, in amptelike en persoonlike korrespondensie die reëls van die geskrewe taal te eerbiedig.

Die bydrae wat die Fakulteit Veeartsenykunde van hierdie Universiteit kan lewer moet teen die agtergrond van die beskikbare mannekrag, sy geskiedenis en die invloed van die internasionale wetenskaplike taal beoordeel word: Sedert die stigting 65 jaar gelede in 1920 het die Fakulteit ongeveer 1400 veeartse opgelei. Ons is 'n tweetalige Fakulteit. Die meeste van ons studente kom uit stedelike gebiede met 'n oorwig van Engelstaliges: ongeveer 58 Engelstalig en 42 Afrikaanstalig. As dié aanname korrek is, is daar 588 Afrikaanssprekende veeartse in Suid-Afrika. Dit is interessant om daarop te let dat die verhouding van Afrikaanssprekende tot Engelssprekende dosente in die Fakulteit gemiddeld 64%: 36% is. In 1931 was daar 16 dosente, 31 in 1959, 46 in 1974 en 67 in 1985. Oor 'n tydperk van 65 jaar het die Fakulteit dus gemiddeld oor 40 dosente beskik waarvan 25 Afrikaanssprekend was. Die verhouding van Afrikaanssprekende tot Engelssprekende dosente is tans 58%: 42%, d.w.s. 39 Afrikaanssprekende dosente. Baie van hulle is nog onervare en begin publiseer — nie noodwendig in Afrikaans nie. Die mannekragpotensiaal in die akademie vir die bevordering van die Afrikaanse veeartsenykundige vaktaal is daarom uiters beperk.

Vanaf 1920 tot einde 1957 was die Fakulteit deel van die staatsdepartement landbou. Navorsing was hoofsaak; onderrig bysaak. In 1958, hoewel nog steeds onder die vleuels van daardie Departement, het 'n nuwe bedeling ingetree en kon aandag aan akademiese ontwikkeling bestee word. Dié ontwikkeling het gedurende die sestigerjare plaasgevind en die Fakulteit het sy beslag as 'n suiwer akademiese instelling gekry. In 1973 het ons 'n selfstandige en volwaardige Fakulteit van die universiteit geword. As sodanig is die Fakulteit 12 jaar oud. As dit as 'n maatstaf vir die bevordering van die Afrikaanse veeartsenykundige vaktaal gereken kan word, het ons goed gepresteer: van 1920 tot 1973 is een Afrikaanse boek gepubliseer, van 1973 tot 1984 het ses verskyn — vyf daarvan sedert 1980.

Daar is drie internasionale wetenskaplike tale, naamlik Engels, Duits en Frans. Sedert die Tweede Wêreldoorlog het Engels 'n steeds belangriker rol in biomediese geskrifte gespeel. Meer as die helfte van die totale biomediese literatuur verskyn tans in Engels en artikels wat in ander tale in vaktydskrifte verskyn, gaan altyd deur 'n opsomming in Engels vergesel. Binnelands beskik ons oor twee joernale met internasionale verspreiding wat veeartsenykundige navorsingsartikels publiseer — een suiwer Engels, die ander tweetalig. Dosente wil hulle navorsingsresultate internasionaal bekend stel en moet daarom in Engels publiseer, trouens, owerheidsbeleid m.b.t. finansiële ondersteuning vir navorsing skryf publikasie in internasionale joernale voor.

Daar is 'n nypende tekort aan sowel Afrikaanse as Engelse handboeke wat op Suid-Afrikaanse toestande gerig is. In my amptelike hoedanigheid as dekaan van die Fakulteit kan ek nie dosente aanmoedig om Afrikaanse handboeke te skryf nie. Die publikasie van Afrikaanse handboeke vir onderrigdoeleindes plaas 'n groot finansiële las op die skrywers as hulle besluit om dit self uit te gee en daarmee die verkoopprys beheer. Enersyds is die afset tot hoogstens 100 eksemplare per jaar beperk en andersyds is die drukkoste 'n afskrikmiddel. Om in die omstandighede en in die lig van die persoonlike finansiële opoffering die geesdrif ter wille van die vaktaal te behou is gewis knyp sonder lag.

Die agtergrond wat ek kortliks hierbo geskets het, skep vir ons verleenthede vir die bevordering en ontwikkeling van die vaktaal. Dit is egter nie rede tot gevoel-

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loosheid en pessimisme nie. Die uitdagings is te opwindend, die ruimte vir skeppende werk te groot en die bevrediging wat daaruit geput kan word, te kosbaar. Laat ons na die positiewe kant van die saak kyk.

Die woordeskat van die moderne mediese wetenskap kom op minstens 'n halfmiljoen te staan. Die veeartsenykundige student se studieveld is nog wyer as dié van die mediese student. Sy leerplan behels die vergelykende studie van vyf huisdiersoorte en hulle siektetoestande teenoor die enkele spesie waarop die gewone medikus konsentreer. As praktiserende veearts kan hy verwag dat feitlik enige soogdierspesie as pasiënt by sy spreekkamer kan opdaag. Bykomende vakke, o.a. Vergelykende Anatomie, Entomologie, Helminitologie en Geslagskunde is by sy leerplan ingesluit.

Die verskeidenheid aandoenings waaraan hierdie diere kan ly, verg onvermydelik ook 'n groter verskeidenheid in die veearts se woordeskat. Danksy die feit dat die basiese woordeskat van die verskillende biologiese subwetenskappe in 'n groot mate ooreenstem en daar ook groot oorvleueling van terminologie tussen vakke soos Anatomie/Fisiologie en Biochemie/Farmakologie voorkom, is die globale veeartsenykundige terminologie aansienlik kleiner as wat dit andersins die geval sou wees.

Die name van anatomiese strukture, wat die basis vorm van alle studie van geneeskunde, maak 'n aansienlike persentasie van die totale mediese terminologie uit. Hoe belangrik wetenskaplikes die bestaan ag van so 'n terminologie met behulp waarvan vakmanne met mekaar kan kommunikeer sonder die gevaar van misverstand, word pragtig geïllustreer deur die ontstaan van Nomina Anatomica Veterinaria in 1967. Die internasionale aard en algemene bruikbaarheid van hierdie terminologie kan vergelyk word met die binomiale stelsel van taksonomie wat wêreldwyd in die Plant- die Dierkunde gebruik word. Die gebruik van hierdie terme skep nie enige probleme vir die Afrikaanssprekende studente of vakman nie. Ons gebruik hulle in die voorgeskrewe vorm. Ek wil graag met 'n paar voorbeelde uit my eie vakgebied, Anatomie, aantoon hoe ons die amptelike benamings gebruik en watter moontlikhede daar vir skeppende werk bestaan.

By geleentheid van 'n anatomiese kongres is die vraag aan my gestel: "Wat maak julle Afrikaners met die term: *M. sternocleidomastoideus?* Die vraesteller wou klaarblyklik hê ek moes antwoord: die *borsbeen-sleutelbeen-tepelvormige uitsteekselspier.* Asof die Engelse taal die alleenreg op Latyn besit. Die letterlike vertaling is korrek maar as sodanig onsinnig. Die amptelike term is in enige spreek- of skryftaal verstaanbaar en aanvaarbaar, ook in Afrikaans.

Die Angel-Saksiese woord, *gape*, beteken die mate waartoe 'n dier sy bek kan oopmaak, m.a.w. die afstand tussen die bo- en onderkakebeen as die bek op sy wydste oop is. Daar is in die opsig aansienlike verskille by ons huissoogdiere en dit speel 'n belangrike rol by die kliniese ondersoek, geneeskundige behandeling en toediening van geneesmiddels. Alhoewel dit nie 'n anatomiese struktuur is nie, is dit 'n begrip wat suiwer op vorm en funksie gebaseer is. Ons gebruik die benaming, *bekspan*, wat die begrip presies weergee.

Die term, *Lig., digitale anulare proximale* leen hom tot sinryke en skeppende vertaalwerk. Die letterlike vertaling, *proksimale ringband van die toon,* is sonder meer aanvaarbaar. By kritiese ontleding van die diep bindweefselstrukture in die toonstreek van die perd, blyk dit dat hulle almal in elk geval soos 'n ring om die toon gerangskik is. Dit is breë bindweefselbande en lyk soos plate. Die band onder bespreking is in dié gedeelte van die toon wat as die koot bekend staan, geleë. Ons noem dit daarom die *kootplaat-* 'n benaming wat met een treffende Afrikaanse woord presies alles sê waarvoor die amptelike Latynse term vier woorde gebruik.

Die Engelse woord, *lock* of *tie*, het betrekking op 'n stadium gedurende die paringsproses van honde waarby die manlike geslagsorgaan letterlik in die vroulike geslagsorgaan vasgeklem word. Vir die vakman gaan dit suiwer oor die anatomiese bou van die geslagsorgane. Met dié agtergrond het ons die term *paringsklem* geskep wat enige misverstand of verkeerde vertolking uitskakel. Die verskillende betekenisse en skakerings wat woordeboeke aan *koppeling* toeskryf maak hierdie woord haas onaanvaarbaar vir die situasie by honde.

Die voorbeelde wat ek hierbo genoem het illustreer die skeppende omgang met taal op die vakterrein. Dit vereis deeglike vakkennis, kritiese waarnemingsvermoë, die aktiewe medewerking van taalkenners en les bes die regte instelling, naamlik om die vaktaal te bevorder deur Afrikaans te skryf en in Afrikaans te publiseer.

Die veeartsenykundiges is berug vir hulle traagheid om te publiseer. Dit is tewens ook 'n eienskap van baie ander natuurwetenskaplikes en bioloë. Hulle vind die grammatika, sinsbou, styl en idioom van die geskrewe taal te ingewikkeld — dit wat inwerk, terugwerk, uitwerk, teenwerk en meewerk is verwarrend en te tydrowend om uit te pluis. Dit het 'n remmende invloed op publikasie. Daar word reeds voorspraak gemaak vir die aanstelling van taalgeleerdes om wetenskaplike artikels en boeke taalkundig te versorg. Dit is nie so eenvoudig as wat dit op die oog af lyk nie. Dit sal nou samewerking tussen wetenskaplike en taalgeleerde vereis. Daar is gebreke aan beide kante: 'n gebrek aan wetenskaplike kennis by die taalgeleerde en 'n gebrek aan taalkennis by die wetenskaplike. Daar is baie natuurwetenskaplikes wat 'n aanvoeling vir die geskrewe taal het. Die basiese opleiding en afronding ontbreek. Eweneens is daar baie taalkundiges met 'n aanvoeling vir die natuurwetenskappe. Ook by hulle ontbreek die basiese opleiding en afronding. Ek pleit vir die instelling van 'n 5-jarige BA-BSc-graad aan ons universiteit. Dit sal studente in staat stel om natuurwetenskaplike en geesteswetenskaplike vakke tot op derdejaarvlak te bestudeer. Op die lang termyn sal die vormende invloed van so 'n graadkursus van onskatbare waarde wees. Dit kan inderdaad 'n versoening tussen die kuns en die wetenskap bewerkstellig — 'n probleem waaroor soveel intelligentsia hulle denke kwel.

Die Afrikaanse veeartsenykundige vaktaal staan in sy kinderskoene. Die moontlikhede wat die veeartsenykundige wetenskap vir die akademie bied om die Afrikaanse vaktaal te ontwikkel, is onbeperk. Daar is die basiese vakgebiede: Anatomie, Histologie, Embriologie, Fisiologie, Etologie en Toksikologie; die kliniese vakgebiede: Geneeskunde, Geslagskunde en Chirurgie; die belangrike parakliniese vakgebiede: Patologie, Parasitologie en Tropiese Siektes. Die geskiedenis van veeartsenykunde in Suid-Afrika moet ook nog geskryf word. Dit is veeartsenykunde se braaklande wat daar lê om geploeg, gesaai en geoes te word — vir Afrikaans! Is dit ons erns?

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ARTIKEL **ARTICLE**

GEDISSEMINEERDE INTRAVASKULÊRE STOLLING IN PERDE: 'N VERSLAG VAN SES GEVALLE

S.R. VAN AMSTEL*, G.C. OLIVIER* en F. REYERS*

ABSTRACT: Van Amstel, S.R., Olivier, G.C., Reyers, F. **Disseminated intravascular coagulation in the horse. A report on six cases.** *Journal of the South African Veterinary Association* (1987) **58** No. 3, 109-112 (Afrik.). Department of Medicine, Faculty of Veterinary Science, University of Pretoria, P/Bag X04, 0110 Onderstepoort, Republic of South Africa.

Six cases of disseminated intravascular coagulation in the horse are reported. They were characterised clinically by severe subcutaneous oedema of the head and neck, and clinocopathologically by abnormal partial tromboplastin and prothrombin times, a thrombocytopaenia and an increase in fibrin degradation product concentrations. Various aetiologies played a role in the pathogenesis of the condition including African horsesickness, babesiosis, lymphosarcoma and verminosis.

Key words: Disseminated intravascular coagulation, African horsesickness, equine babesiosis, horses

INLEIDING

Gedissemineerde intravaskulêre stolling (DIS) is 'n term gebruik om 'n komplekse patofisiologiese sindroomentiteit, wat sekondêr tot onderliggende siekte kan plaasvind, te beskryf. DIS ontwikkel as gevolg van 'n oormatige stimulasie van bloedstolling en fibrinolise *in vivo,* gevolg deur die konsumpsie van trombosiete en stollingsfaktore, die produksie van fibrienstolsels in die lokale of algemene mikrosirkulasie, hulle afbraak en derhalwe die ophoping van fibrienafbraakprodukte wat in die sistemiese sirkulasie as kragtige antistolmiddels optree[5,7,8].

Ses perde met DIS is oor 'n ses maande periode geïdentifiseer deur die Fakulteit Veeartsenykunde, Onderstepoort. Alhoewel 'n verskeidenheid onderliggende etiologiese faktore betrokke was, was die kliniese ooreenkoms tussen die gevalle opmerklik. Bevestiging van die teenwoordigheid van DIS was gebaseer op kliniese patologiese toetse.

MATERIALE EN METODIEK

Kliniese gevalle

Ses perde, waarvan vyf kruistipe saalperde en een 'n ponie was. Al ses perde was afkomstig van kleinhoewes in die Onderstepoort omgewing. Die perde was deurgaans aangehou onder swak toestande van voeding en versorging.

In al die gevalle was die eienaars se klagte die verskyning binne 48 uur, van milde tot erge onderhuidse edeem van die kop en nek, wat nie onderskei kon word met die van dikkopperdesiekte nie. In geen gevalle kon 'n onlangse entingsgeskiedenis verkry word nie.

Gevalle 1, 2 en 4 (sien Tabel 1) was gehospitaliseer in die Departement Geneeskunde, Fakulteit Veeartsenykunde, Universiteit van Pretoria, terwyl die ander drie perde deur hulle eienaars aangehou was gedurende die ondersoek en behandeling.

Al ses gevalle was onderwerp aan 'n volledige kliniese ondersoek, insluitende 'n urienanalise sowel as 'n fekale flotasie toets. Al ses perde het spesifieke en/of ondersteunende behandeling ontvang (sien behandeling).

Gevalle 2, 4 en 6 is dood en daarna onderwerp aan 'n volledige nadoodse ondersoek.

Laboratorium toetse

Heelbloed in EDTA en serum versamel op die eerste dag van aanbieding van hierdie gevalle was onderwerp aan die volgende toetse: hematologiese ondersoek, serum protein bepaling; serum lewerensiem aktiwiteit (Sorbitol dehidrogenase SDH) asook 'n stollingsprofiel insluitende protrombientyd, gedeeltelike tromboplastientyd, plasma fibrinogeen vlakke, sowel as die konsentrasie van fibrienafbraakprodukte.

Hematologie was gedoen met die gebruik van 'n coulterteller model Fn (Coulter Electronics Inc., Hialeah, Florida, USA) en die mikroskopiese ondersoek van bloedsmere gekleur met Harleco's Diff. Quick (C.A. Milsch Pty. Ltd., P.O. Box 943, Krugersdorp, South Africa).

Totale serum protein bepaling was gedoen deur gebruik te maak van die biuretmetode in 'n RA 1000 geoutomatiseerde analiseerder (Technicon Corporation, Tarrytown, USA) met 'n gesette punt RA 1000 kalibrator (T13-1291) 64 $g\ell^{-1}$ protein standaard. Die albumien was gedoen met bromocresyl-groen ook in die RA 1000 (metode TO1-1373) met 'n gesette punt RA 1000 kalibrator (T13-1291) 36 $g\ell^{-1}$ protein standaard.

SDH bepalings was gedoen met behulp van 'n LP 6 fotometer (dr Lange fotometer, Wes Duitsland).

Die protrombientyd was gedoen deur gebruik te maak van Boehringer Mannheim se toetsstel (Katalogus Nr 126551) en was uitgevoer volgens die vervaardigers se aanwysings. Vir elke bepaling is 'n normale perd ingesluit as 'n kontrole.

Die gedeeltelike tromboplastientyd was ook bepaal met behulp van 'n Boehringer Mannheim toetsstel (Katalogus Nr 126551) en weereens is die vervaardigers se aanwysings gevolg. 'n Normale perd is ingesluit as 'n kontrole vir elke bepaling.

Fibrienafbraakproduk-konsentrasies was bepaal met die gebruik van die Trombo-Wellco (Rapid Latex) toets HA 13 (Wellcome Laboratories).

Fibrinogeenvlakke was bepaal volgens die verskil

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tussen die plasma en serum elektroforetogram beta globulien bande.

Behandeling

'n Opsomming van die behandeling van indiwiduele gevalle verskyn in Tabel 1. Perde wat positief was vir babesiose (Tabel 2) was behandel met 20 ml euflavien i.v. (Euflavine, Centaur). Aangesien die teenwoordigheid van DIS en/of perdesiekte vermoed was is die volgende middels gebruik: furosemide (Lasix, Hoechst) teen 1 mg kg^{-1} subkutaan; heparien (Pularin, Allen & Hanburys) teen 25 i.e. kg^{-1} subkutaan; kortisoon (Solu-Delta Cortef, Upjohn) binneaars toegedien teen 'n dosis van 1 mg kg^{-1} en flunixin (Banamine, Centaur) was binnespiers gegee teen 1 mg kg^{-1}. Verder is penicillin (Procaine penicillin G, Milvet) teen 30 000 i.e. kg^{-1} as 'n antibiotiese dekking gebruik. In een perd (geval 2) was 6ℓ bloed toegedien agv die teenwoordigheid van 'n erge anemie.

In gevalle waar die fekale flotasie ondersoek die teenwoordigheid van wurmeiers aangedui het is ivermectin (Eqvalan, MSD) of fenbendazole (Panacur, Hoechst) gebruik.

RESULTATE

'n Opsomming van die belangrikste kliniese tekens word gegee in Tabel 1. Subkutane edeem van die kop en nek wat in sommige gevalle (1, 2 en 3) tot tussen die voorbene afgestrek het, was die mees uitgesproke tekens. Hierbenewens was dispnee ook opvallend in al die gevalle. In sommige was die dispnee geassosieer met sianotiese slymvliese (Geval 4 en 6) en 'n opsigtelike naatlyn (heaveline) van die abdominale spiere gedurende ekspirasie (Gevalle 1, 2, 4, 5 en 6). Uitgesproke lusteloosheid en anoreksie was ook teenwoordig by al die gevalle. 'n Matige tagikardie was deurgaans teenwoordig.

Resultate van die kliniese patologiese ondersoek verskyn in Tabel 2. 'n Erge anemie was teenwoordig in Geval 2 wat toegeskryf was aan die teenwoordigheid van 'n *B. equi* besmetting. Soortgelyke besmettings was ook teenwoordig in Gevalle 1 en 6 terwyl Geval 3 'n *B. caballi* positiewe bloedsmeer gehad het. Verder het meeste gevalle 'n leukositose getoon wat te wyte was aan 'n neutrofilie en 'n links verskuiwing. 'n Hipoalbuminemie was teenwoordig in al 6 gevalle. Hierdie verandering was heelwaarskynlik te wyte aan 'n kombinasie van swak voeding en verminose aangesien slegs een perd (Geval 6), verhoogde serumvlakke van SDH getoon het.

Afwykings in die stollingsprofiel is duidelik sigbaar in Tabel 2. In alle gevalle was daar 'n groot afname in die hoeveelheid sirkulerende trombosiete. Verlengings in beide die protrombientyd en die tromboplastientyd het hiermee gepaard gegaan terwyl daar ook aansamelings was van fibrienafbraakprodukte. Hierdie veranderinge was aanvaar as 'n aanduiding van die bestaan van DIS in hierdie gevalle.

Resultate van die patologiese en virologiese bevindinge kan gesien word in Tabel 3. In Gevalle 1 en 3 wat herstel het, kon die laboratoriumaanduidings van DIS moontlik gekoppel gewees het met die teenwoordigheid van die babesiose terwyl Geval 5 wat ook herstel het, slegs 'n swaar wurmbesmetting gehad het volgens die fekale flotasie bevinding. 'n Etiologiese diagnose van limfosarkoom was gemaak in Geval 2 op nadoodse ondersoek terwyl 'n diagnose van DIS gestel was in Gevalle 4 en 6 gebaseer op die makropatologiese veranderinge. Virologiese ondersoek in beide hierdie gevalle het getoon dat perdesiektevirus heelwaarskynlik as sneller gedien het vir die ontstaan van die DIS.

BESPREKING

Dit word aanvaar dat die laboratoriumbevindings wat nodig is om die diagnose van DIS te staaf, drie van die volgende parameters moet insluit; naamlik verlengde protrombientyd, verlengde gedeeltelike tromboplastientyd, verlaagde fibrinogeenkonsentrasie, 'n trombositopenie en 'n verhoogde fibrienafbraakprodukte waarde[3 6 7]. In die gevalle bespreek is daar nie altyd vir al vyf hierdie parameters getoets nie, maar uit die beskikbare abnormale kliniese patologiese inligting en ooreenstemmende kliniese tekens en nadoodse bevindinge, word die afleiding gemaak dat al ses gevalle DIS gehad het.

Tabel 1: Opsomming van die belangrikste kliniese tekens en behandeling van 6 perde met DIS

	Geval 1	Geval 2	Geval 3	Geval 4	Geval 5	Geval 6
Ouderdom (jaar)	3,5	18	3	11	5	4
Algemene kondisie	redelik	vermaer	vermaer	redelik	vermaer	redelik
Lusteloosheid	erg	erg	erg	erg	matig	erg
Bosluisbesmetting	lig	lig	lig	lig	swaar	swaar
Onderhuidse edeem (kop en nek)	erg	erg	matig	erg	matig	erg
Slymvliese	ligte geelsug puntbloedinge	ligte geelsug erge anemie	puntbloedinge kongestie	puntbloedinge sianose	kongestie	kongestie sianose
Dispnee	matig	matig	matig	erg	matig	erg
Hartspoed (min^{-1})	64	84	56	90	40	84
Respirasiespoed (min^{-1})	24	20	26	56	20	54
Wurmbesmetting*	swaar	negatief	matig	lig	swaar	swaar
Behandeling	Euflavien Penisillien Furosemide Fosfolipiede Vit B Fenbendazole	Euflavien Penisillien Furosemide Heparien Flunixin Bloed	Euflavien Penisillien Furosemide Ivermectin	Penisillien Furosemide Heparien Kortisoon Ivermectin	Penisillien Furosemide Ivermectin	Euflavien Penisillien Furosemide Kortisoon
Oorlewing	herstel	dood	herstel	dood	herstel	dood

* Beoordeling volgens fekale flotasie ondersoek

Tabel 2: Resultate van die kliniese patologiese ondersoek van gevalle met DIS

Kliniese patologiese parameter	Gevalle 1	2	3	4	5	6	Normale waarde Schalm O.W.[10] Veterinary Hematology
Hemoglobien g/ℓ	81	31	128	178	139	219	80-240
Hematokrit ℓ/ℓ	0,24	0,09	0,34	0,48	0,38	0,61	0,24-0,44
Rooiseltelling x 10^{12}/ℓ	4,96	1,44	6,33	9,17	7,64	12,04	5,5-9,5
Witseltelling x 10^9/ℓ	21,5	26,5	10,6	8,6	14,1	15,3	7,25-10,85
Neutrofiele volwasse (verhouding van 1)	0,55	0,75	0,44	0,55	0,62	0,34	0,52 ± 0,08
Neutrofiele onvolwasse (verhouding van 1)	0,13	0	0,17	0,33	0,18	0,29	0,003 ± 0,009
Limfosiete (verhouding van 1)	0,30	0,10	0,20	0,70	0,12	0,26	0,38 ± 0,08
Monosiete (verhouding van 1)	–	0,15	0,19	0,5	0,8	0,11	0,43 ± 0,02
Trombosiete x 10^9/ℓ	90	»	40	39	70	65	200-600
Aktiewe monosiete – tot 6 +	2+	2+	–	3+	2+	4+	
Babesia negatief/teewoordig	*B. equi*	*B. equi*	*B. caballi*	Neg	Neg	*B. equi*	
Albumien g/ℓ	20,8	17,5	20,3	18,8	21,9	19,5	22,7-41 Boehringer = Mannheim[2]
Sorbitol dehidrogenase/ℓ@25°	0,9	3,7	1,7	3,2	3,8	22,4	< 1,3 Boehringer = Mannheim
Protrombientyd sekondes (sek.)	24(8)	–	*26(9)*	*20(15)*	*26(15)*	33(15)	
Gedeeltelike tromboplastientyd (sek.)	35(30)	–	38(15)	45(35)	55(35)	48(30)	
Fibrienafbraakprodukte µg/mℓ	10	40	40	10	10	10	< 10
Fibrinogeen g/ℓ	–	–	4	1	1	8	2-4[10]

» = Mikroskopiese beoordeling. Minder as 6 trombosiete per olieveld
( ) = Waarde van 'n normale kontrole perd

Tabel 3: Resultate van die patologiese en virologiese ondersoeke

Geval	Belangrikste patologiese veranderinge	Virologiese ondersoek
1	Geen (oorleef)	Negatiewe perdesiekte-virus isolasie
2	Limfosarkoom van milt Onderhuidse edeem Hidrotoraks; hidroperikardium	Nie gedoen
3	Geen (oorleef)	Verdagte positiewe komplementbindingstoets vir perdesiekte
4	Wydverspreide bloedinge Erge kongestie Hidrotoraks; hidroperikardium Onderhuidse edeem Verminose	Positiewe perdesiekte-virus isolasie
5	Geen (oorleef)	Nie gedoen
6	Wydverspreide bloedinge Erge kongestie Longedeem Onderhuidse edeem Kroniese endokarditis Verminose	Positiewe perdesiekte-virus isolasie

Sover die skrywers kon vasstel is hierdie die eerste publikasie waar kliniese perdesiektegevalle gekoppel word met DIS betrokkenheid. Of ongekompliseerde eksperimentele perdesiektegevalle DIS ontwikkel, is skynbaar nie in die verlede kliniespatologies voor getoets nie. Daarby verdwyn histopatologiese bewyse van DIS redelik gou na dood weens die vinnige aktivering van fibrinolise pre- en post-mortaal[9]. Hierdie kan waarskynlik ook die rede wees waarom tekens van intravaskulêre stolling ook nie in die nadoodse gevalle, histologies waargeneem was nie. Wat ook al ten opsigte van perdesiektegevalle, DIS kon sneller, is spekulatief van aard. Ultrastrukturele endoteelveranderinge is waarskynlik nie betrokke nie[8]. Antigeen teenliggaam kompleksreaksies soos wat aan viremieë toegeskryf word, kan egter DIS sneller[3]. Weefsel- en orgaanhipoksie soos met bloedsirkulatoriese veranderinge en suurbasis versteurings sal in gevorderde perdesiektegevalle voorkom en deur die vrystelling van vaso-aktiewe stowwe, die DIS proses aan die gang kan sit[3].

Reeds so vroeg as in 1890 het die ooreenkoms tussen erge galkoorsgevalle met supra-orbitale edeem en milde perdesiektegevalle met dikkop, verwarring veroorsaak sodat hierdie twee toestande selfs as dieselfde siekte bestempel is[5]. Die feit dat ongekompliseerde babesiose gevalle wel supraorbitale edeem kan ontwikkel is welbekend[11 12]. Dit word gespekuleer dat die gelyktydige voorkoms van galkoors en perdesiektevirusbesmettings in swakversorgde ongeimmuniseerde dier, nie 'n seldsame verskynsel is nie. Die rol wat 'n subkliniese galkoorsbesmetting mag speel in die ontwikkeling van perdesiekte is tans egter onbekend.

Ongekompliseerde galkoorsgevalle in perde is sover nog nie met DIS geassosieer nie. So kon onlangs, in gekontroleerde babesia besmettings in perde, DIS se betrokkenheid nie bewys word nie. Hoewel die trombosiettellings in hierdie gevalle tipies verlaag was, kon abnormale bloedstollingstye of verhoogde fibrinogeen afbraakprodukwaardes nie gedemonstreer word nie[1] (J. van Heerden, persoonlike mededeling, Fakulteit Veeartsenykunde, Medunsa, Suid-Afrika). Dit wil dus voorkom dat vir DIS om by babesiose infeksies in perde te ontwikkel, ander kompliserende faktore teenwoordig moet wees.

Die rol van wanvoeding, ekto- en endoparasiete soos in meeste van hierdie gevalle voorgekom het, is slegs spekulatief van aard.

Die onderliggende patologie in Geval 2 wat bygedra het tot die ontwikkeling van DIS, is 'n limfosarkoom van die milt. Neoplasieë is welbekende oorsake an DIS in mens en dier[4 7].

Hipo-albuminemie was 'n konstante bevinding in al ses gevalle. Die werklike oorsaak hiervan is onbekend maar kon multifaktoriaal wees. Al ses perde was in 'n swak kondisie en 'n voedingsproteintekort, verminose, en die ektoparasietbesmetting, kon daartoe bygedra het. 'n Ander belangrike meganisme vir albumienverlies is 'n verhoogde kapillêre deurlaatbaarheid. Edeem vloeistof in perdesiekte gevalle bevat feitlik dieselfde protein inhoud as die van plasma. (Littlejohn A, Erasmus B J, Newsholme S, Reyers F, 1984. Ongepubliseerde data, aangebied by die 1984 Fakulteitsdag, Fakulteit Veeartsenykunde, Universiteit van Pretoria).

Uit die gevalle bespreek word gedemonstreer hoedat verskillende etiologieë 'n rol kan speel in die snellering van DIS sindroom[4].

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ARTIKEL ARTICLE

FOOT ABSCESS IN GOATS IN RELATION TO THE SEASONAL ABUNDANCE OF ADULT *ABLYOMMA HEBRAEUM* AND ADULT *RHIPICEPHALUS GLABROSCUTATUM* (ACARI: IXODIDAE)

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ABSTRACT: MacIvor, K.M. de F.; Horak, I.G. **Foot abscess in goats in relation to the seasonal abundance of adult *Amblyomma hebraeum* and adult *Rhipicephalus glabroscutatum* (Acari: Ixodidae).** *Journal of the South African Veterinary Association* (1987) **58** No. 3, 113-118 (En). Tick Research Unit, Rhodes University, 6140 Grahamstown, Republic of South Africa.

The seasonal prevalence of foot abscess affecting Angora and Boer goats in Valley Bushveld in the eastern Cape Province of South Africa was significantly related to the seasonal abundance of adult *Amblyomma hebraeum* and adult *Rhipicephalus glabroscutatum.* Angora goats harboured more ticks than Boer goats and also had a greater number of abscessed feet. Larger numbers of ticks and abscesses were recorded on hind feet than on fore feet. There was no significant correlation between rainfall and the occurrence of foot abscess.

The seasonal abundance of adult *A. hebraeum* and adult *R. glabroscutatum* on several ruminant species at various localities in the Cape Province is described.

Key words: Foot abscess, goats, *Amblyomma hebraeum, Rhipicephalus glabroscutatum*

INTRODUCTION

The geographic distribution of *Amblyomma hebraeum*[5] and more particularly that of *Rhipicephalus glabroscutatum*[8] overlap large portions of the regions in the eastern Cape Province in which goats are farmed for mohair and meat[11]. Theiler[10] states that *A. hebraeum* causes deep wounds which, if they become secondarily infected, may lead to abscess formation. Of *R. glabroscutatum* she says "It's habit of attaching to the feet between the claws often leads to extreme and painfull lameness in sheep and goats"[10]. The probable involvement of these ticks in the aetiology of foot abscess in goats has been mentioned by MacIvor & Horak[7]. The effects of lameness caused by foot abscesses are difficult to evaluate because affected goats do not normally die. Nevertheless the economic implication of parasitism of the feet by adult *A. hebraeum* and *R. glabroscutatum* in goat farming areas is considerable, because affected animals suffer loss of condition, presumably due to impaired feeding.

Foot abscess is characterised by the production of a thick, grey, sharpsmelling pus exuding from openings in the axial region (axial perioplic corium)[2] of the feet of sheep and goats[12]. The affected feet are hot and painfull and the animal often limps badly. The causative organism is *Fusobacterium necrophorum* and the condition may be aggravated by the presence of *Corynebacterium pyogenes*[12].

The seasonal abundance of *A. hebraeum* and of *R. glabroscutatum* has been the subject of a number of recent surveys conducted in the eastern Cape Province[4,6,7,9]. This paper establishes the association between the seasonal abundance of adult ticks of these species and the seasonal prevalence of foot abscess in Angora and Boer goats. We also describe the seasonal abundance of adult *A. hebraeum* and adult *R. glabroscutatum* on several host species at a number of localities in the Cape Province.

MATERIALS AND METHODS

All 4 feet of 10 Angora and 10 boer goats were examined at least once a month for the presence of foot abscess and of ticks for a period of 36 consecutive months from February 1981 to January 1984. These goats were not treated with an acaricide during the period of observation and ran in a heavily tick-infested camp in Valley Bushveld[1] on the farm "Brakhill" (33°33' S; 25°25' E) 30km north of Uitenhage in the eastern Cape Province. At each occasion ticks were collected from each foot and placed in separate vials containing 70% alcohol. These ticks were later counted and identified under a stereoscopic microscope. Total monthly rainfall was recorded in the camp.

In addition a mixed flock of Angora and Boer goats, also not treated with an acaricide, running in the same camp as the experimental goats, was observed for signs of lameness between September and December 1983. These were random observations made in the dense bush in the camp and continued until 100 goats of each breed had been seen. Because these goats were not individually identifiable, repeated observations of the same goats may have occurred. During October of the same year the feet of 300 Angora goats, regularly treated with an acaricide, and running in a comparatively tick-free camp on the same farm, were inspected for abscesses and for ticks.

Two Angora goats, 2 Boer goats, 1 kudu *(Tragelaphus strepsiceros)* and 1 grey duiker *(Sylvicapra grimmia)* were slaughtered on the farm at monthly intervals from February 1983 to January 1984. Cattle running in Valley Bushveld on the farm "Bucklands" (33° 05'S; 26° 41'E) and kudu on the adjoining Andries Vosloo Kudu Reserve (33° 07'S; 26° 43'E) north of Grahamstown; mountain reedbuck *(Redunca fulvorufula)* and eland *(Taurotragus oryx)* in Karroid *Merxmeullera*

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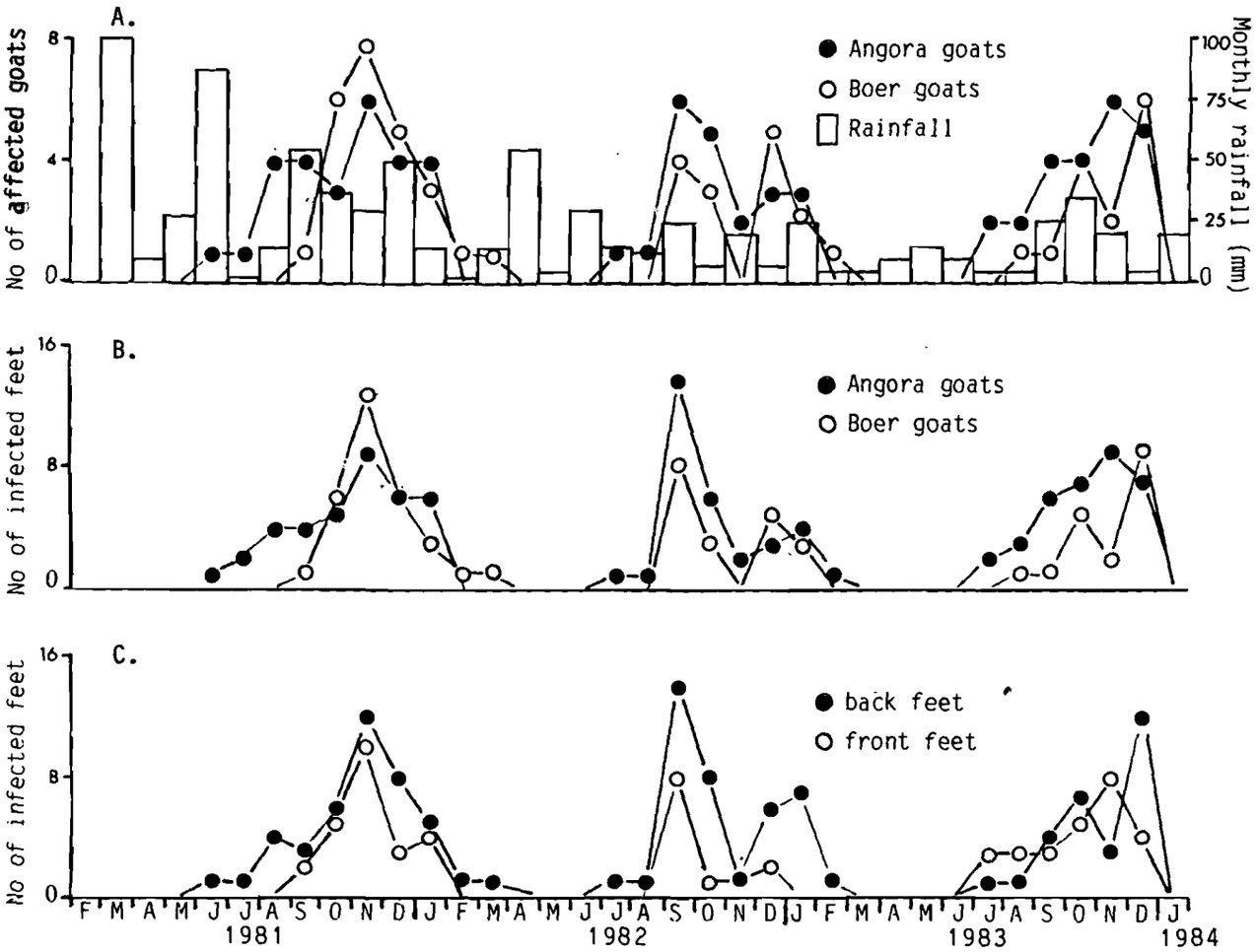

Fig. 1: A. Monthly rainfall and the numbers of goats with abscessed feet
B. The number of abscessed feet seen on 10 Angora goats and 10 Boer goats
C. The numbers of abscessed front and back feet

Mountain Veld replaced by Karoo[1] in the Mountain Zebra National Park (32° 15'S; 25° 41'E) south-west of Cradock; and bontebok *(Damaliscus dorcas dorcas)* running in False Macchia and Coastal Renosterbosveld[1] in the Bontebok National Park (34° 02'S; 20° 25'E) in the south-western Cape Province were also slaughtered at regular intervals. All these animals were carefully processed for the recovery of ticks[3]. The numbers of adult *A. hebraeum* and *R. glabroscutatum* recovered from the slaughtered goats, cattle and antelope are presented in this paper.

RESULTS

Monthly rainfall, the number of goats with foot abscesses and the number of abscessed feet recorded during 36 months are shown in Fig. 1.

Monthly rainfall was erratic, and total annual rainfall declined from 444mm during the first year of the survey to 220mm during the second year and 159mm during the third year. Despite the erratic monthly rainfall and decline in annual rainfall, the goats developed foot abscesses in very regular annual cycles. There was no significant correlation between rainfall and the number of abscessed feet ($r = 0,14$; $p > 0,05$). The greatest numbers of affected goats and abscessed feet were recorded from August to January in the first year, from September to January in the second year and from July to December in the third year.

The Angora goats tended to develop foot abscesses earlier than Boer goats. Paired T-test's indicated that significantly more Angora goats than Boer goats were affected ($t = 3,04$; $df = 23$; $pp < 0,01$) and they also had significantly more abscessed feet ($t = 2,92$; $df = 24$; $p < 0,01$). It was also common for a goat to have more than one abscessed foot at a time. The number of goats with foot abscesses and the number of abscessed feet followed nearly identical trends. There were significantly more abscessed back feet than front feet ($t = 2,98$; $df = 23$; $p < 0,01$) for both goat breeds.

Although there was considerable variation within each group, each of the 10 goats of both breeds developed foot abscesses during the 36 month period of observation. In the Angora goat group the total number of observations of foot abscess per goat ranged between 3 and 21 with a mean of 11,0 (s.d. ± 5,35), and a coefficient of variation of 49%. In the Boer goat group the number of observations per goat ranged between 1 and 16 with a mean of 8,7 (± 4,57) and a coefficient of variation of 52%. This indicated that the abscesses on some goats persisted for a number of months and were observed on more than one occasion.

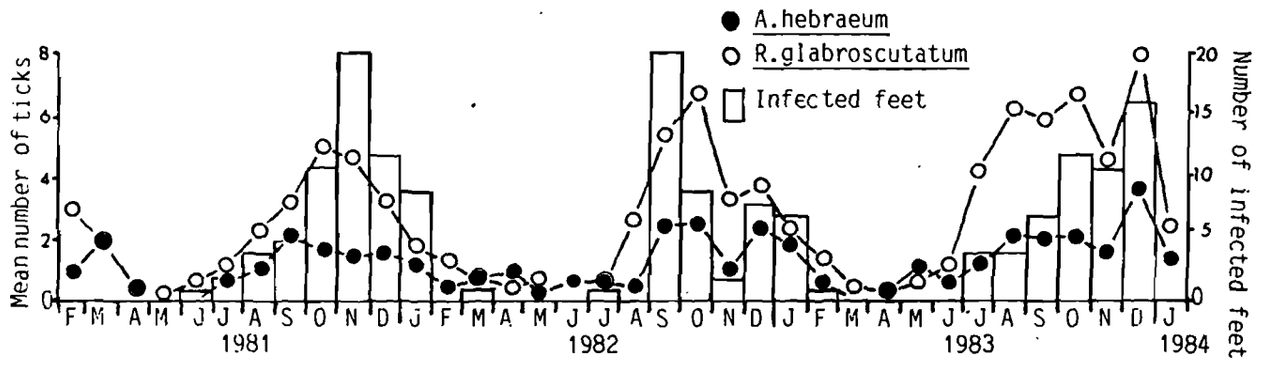

Fig. 2: The relationship between the number of abscessed feet and *the seasonal abundance of adult ticks on goats in Valley* Bushveld

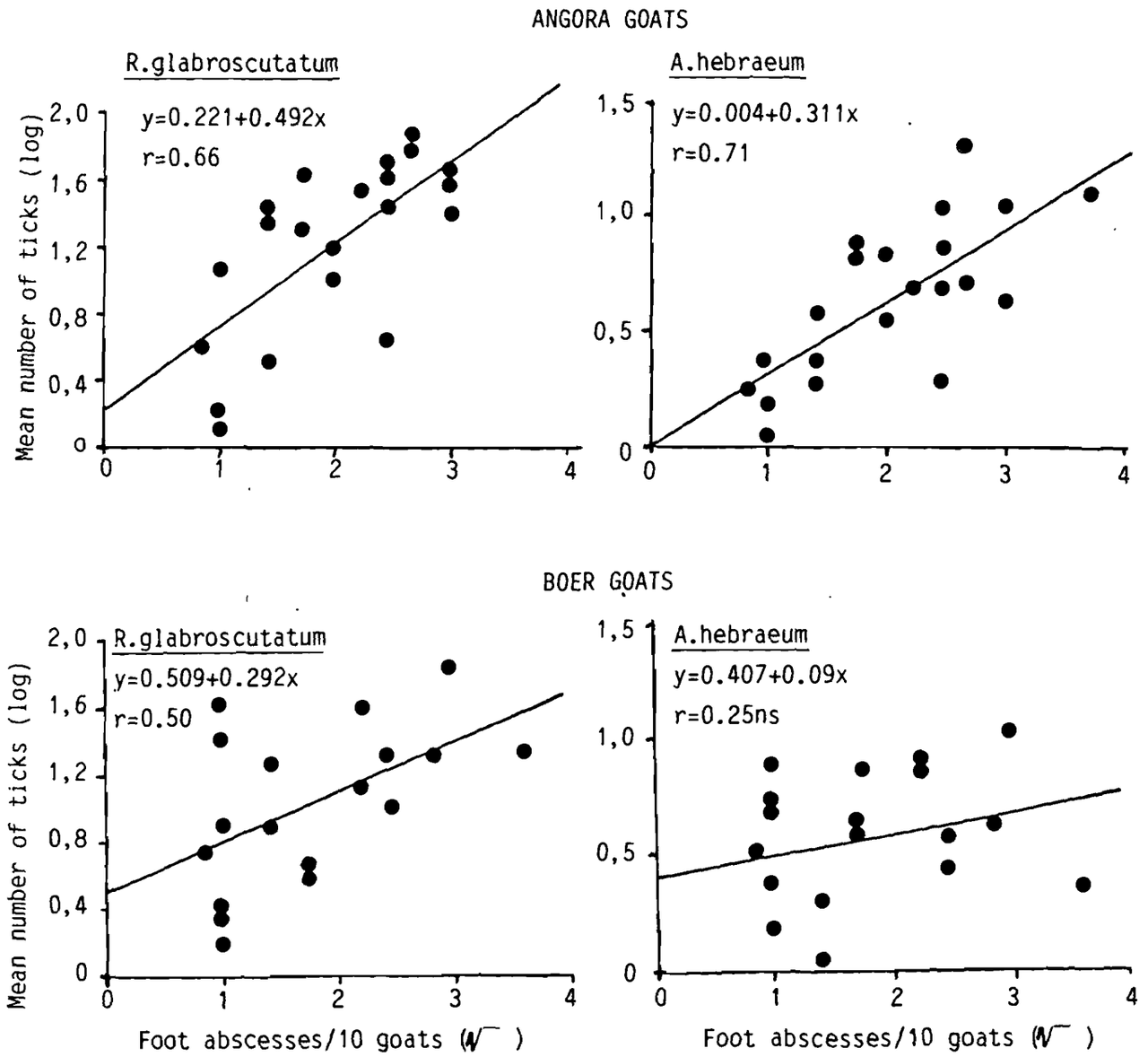

Fig. 3: The relationship between the numbers of abscessed feet on Angora and Boer goats and the numbers of adult *Amblyomma hebraeum* and adult *Rhipicephalus glabroscutatum*

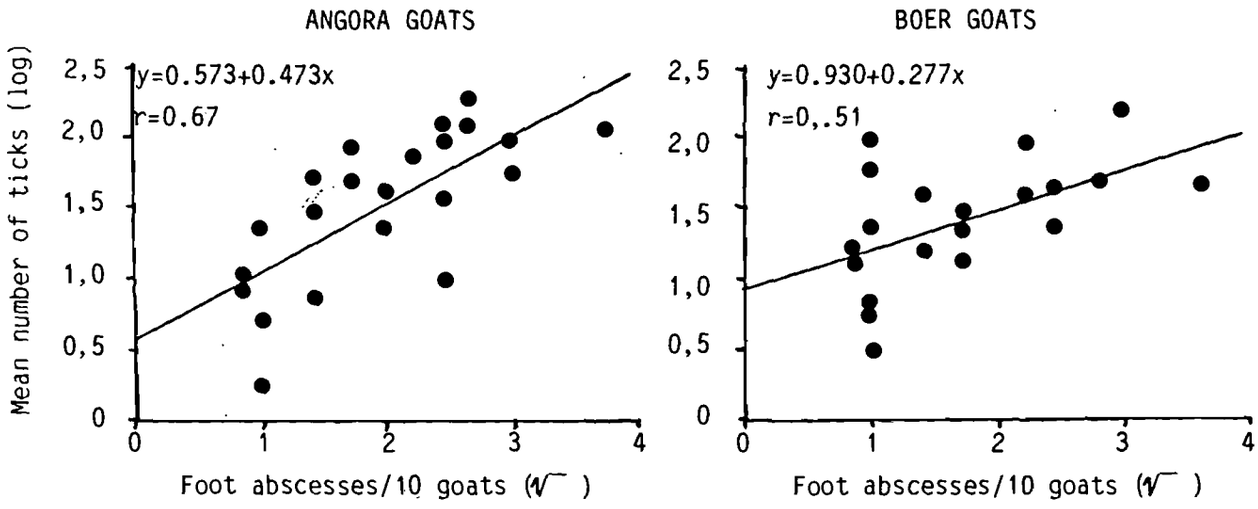

Fig. 4: The relationship between the numbers of abscessed feet on Angora and Boer goats and the total number of adult ticks

The number of abscessed feet in relation to the number of adult ticks is shown in Fig. 2.

A clear relationship between tick infestation and the number of abscessed feet was evident during the 3 years of observation. The numbers of abscessed feet followed a normal distribution in the first year, a bimodal distribution in the second year and a negatively skew distribution in the final year. The numbers of adult *A. hebraeum* and adult *R. glabroscutatum* collected from the feet of these goats exhibited distribution patterns similar to those of the abscessed feet during each of the 3 years. Total adult tick numbers reached peak levels from September to December during the first year, from September to January during the second year and from August to December during the third year. This corresponded closely to the periods during which the largest numbers of abscessed feet were observed.

The Angora goats harboured significantly more adult *A. hebraeum* ($F = 27{,}98$; $p < 0{,}001$) and *R. glabroscutatum* ($F = 133{,}33$; $p < 0{,}001$) than did the Boer goats. There were also significantly more adult *A. glabroscutatum* ($F = 98{,}82$; $p < 0{,}001$) and adult *R. glabroscutatum* ($F = 15.61$; $p < 0{,}001$) on the back feet than on the front feet of both goat breeds.

The relationship between the numbers of adult *A. hebraeum* and adult *R. glabroscutatum* separately and the numbers of abscessed feet on the two goat breeds is shown in Fig. 3. The relationship between the combined burdens of both tick species and foot abscesses on the two goat breeds is shown in Fig. 4.

There was a significant correlation between the numbers of both adult *A. hebraeum* and adult *R. glabroscutatum* and abscessed feet in Angora goats, while for the Boer goats only the numbers of adult *R. glabroscutatum* and abscessed feet were significantly correlated (Fig. 3). The relationship between adult tick burdens and the numbers of abscessed feet exhibits a steeper linear curve fit for the Angora goats than for the Boer goats. This implies that Angora goats are likely to develop foot abscesses at lower levels of tick infestation than Boer goats. The relationship between tick burden and numbers of abscessed feet was more variable for the Boer goats particularly at the lower levels of infestation.

The observations for lameness, as opposed to foot abscess, in the free-ranging goats revealed that 13% of Angora goats were seen to limp compared with 17% of Boer goats. These differences were not significant. Only 2% of the 300 Angora goats which were treated with an acaricide and ran in the comparatively tick-free camp, had foot abscesses and no ticks were seen on their feet.

The numbers of adult *A. hebraeum* and adult *R. glabroscutatum* recovered at slaughter from several host species at various localities are summarized in Tables 1 and 2.

Peak burdens of *A. hebraeum* on the goats at "Brakhill" were recorded from June or August to January or February. The Angora goats harboured considerably more adult *A. hebraeum* than did Boer goats. Both grey duiker and kudu were examined on the farm "Brakhill" and the kudu harboured a total of only 2 adult *A. hebraeum*, while the duiker were not infested with adult ticks of this species. Peak burdens on the cattle at "Bucklands" and kudu from the adjoining Kudu Reserve were present from September or October to April. No *A. hebraeum* were recovered from the animals examined in the Mountain Zebra National Park or Bontebok National Park.

The seasonal abundance of adult *R. glabroscutatum* on the various hosts in the different localities was fairly similar, maximum numbers were usually present from August to December or February. At "Brakhill" the Angora goats harboured slightly more adult *R. glabroscutatum* than did the Boer goats, while the kudu harboured considerably more than either of the goat breeds. The largest numbers of ticks were recovered from animals slaughtered at localities situated in Valley Bushveld and the smallest from the Bontebok grazing Renosterbosveld in the south-western Cape Province.

DISCUSSION

It has been suggested that wet or moist and muddy conditions are predisposing factors favouring the occurrence of foot abscess[11]. These conditions could develop during periods of high rainfall, or when stock are watered from leaking drinking-troughs or confined in poorly-drained, small camps or pens. In this study there appeared to be no relationship between rainfall and the occurrence of foot abscess. Abscesses were equally prevalent in years of high and low rainfall and followed a seasonal pattern unrelated to monthly rainfall.

Table 1: The numbers of adult *Amblyomma hebraeum* recovered from Angora goats, Boer goats, cattle and kudu examined at various localities

Month	Mean numbers of adult *A. hebraeum* recovered			
	"Brakhill"		"Bucklands"	Andries Vosloo Kudu Reserve
	Angora goats (24)	Boer goats (24)	Cattle (22)	Kudu (12)
January	6	0	69	18
February	1	43	52	39
March	0	0	18	48
April	0	0	38	100
May	0	0	1	8
June	13	2	0	34
July	27	0	2	0
August	62	9	1	2
September	12	12	2	73
October	28	9	32	0
November	16	15	27	22
December	32	3	65	304

( ) = Total numbers of animals slaughtered and examined for ticks

Table 2: The numbers of adult *Rhipicephalus glabroscutatum* recovered from Angora goats, Boer goats, cattle, grey duiker, kudu, mountain reedbuck, eland and bontebok examined at various localities

Month	Mean numbers of adult *R. glabroscutatum* recovered								
	"Brakhill"				"Bucklands"	Andries Vosloo Kudu Reserve	Mountain Zebra National Park		Bontebok Park
	Angora goats (24)	Boer goats (24)	Grey duiker (12)	Kudu (12)	Cattle (22)	Kudu (12)	Mountain reedbuck (18)	Eland (11)	Bontebok (16)
January	2	1	7	90	142	38	–	–	–
February	0	5	296	191	7	192	16	21	1
March	0	1	0	7	28	26	0	1	–
April	0	0	0	6	4	6	–	–	0
May	1	0	5	24	0	8	2	1	–
June	7	4	6	7	3	18	6	1	1
July	22	18	12	430	5	32	–	–	–
August	101	39	16	223	39	48	55	85	18
September	151	125	68	285	22	362	92	124	–
October	250	206	29	425	67	117	–	–	22
November	156	210	36	209	66	244	102	156	–
December	108	132	42	119	120	234	22	298	7

( ) = Total number of animals slaughtered

Van der Westhuysen et al[11] have suggested that usually only a single foot of affected goats is abscessed. Our results indicate that it is common for goats to have more than one abscessed foot. It could be argued that the regular removal of ticks from the feet of the experimental goats afforded sites suitable for bacterial entry, but the same could possibly apply if the ticks had been left on the feet and allowed to drop off naturally, or been removed by the goats during grooming.

This study shows that there is a significant relationship between the occurrence of foot abscess in goats and the presence of adult *A. hebraeum* and *R. glabroscutatum*. Support for this finding is provided by the similar distribution patterns of the numbers of abscessed feet and numbers of adult ticks during the 3 years of observation, as well as the greater numbers of both ticks and abscesses recorded on the Angora goats than on Boer goats and on back feet than on front feet. Furthermore the larger the number of adult ticks present the greater the incidence of foot abscesses. However, because Angora goats seem less prone to limp than Boer goats when they do have abscessed feet, it is essential that goats' feet be individually inspected to determine the incidence of abscessation.

It would appear that both *R. glabroscutatum* and *A. hebraeum* are implicated in the aetiology of foot abscess on Angora goats. There was a significant correlation between the numbers of adult ticks of each species and the incidence of foot abscesses on these goats. For Boer goats this correlation was only valid for adult *R. glabroscutatum*.

The mouthparts of *R. glabroscutatum* are relatively short and hence would not seem capable of causing extensive damage. However, the comparatively large numbers of these ticks that may be present would afford numerous foci for secondary bacterial invasion. *A. hebraeum* has very long mouthparts and is capable of causing considerable damage, but because the number of adult ticks of this species is usually not very large fewer potential sites for bacterial entry are present.

At "Brakhill" no adult ticks were found between the hooves of the wild antelope, while the goats frequently harboured large numbers. This difference between the antelope and domestic goats could possibly be attributed to the physical structure of the hooves. The axial corium of the goat hoof is concave resulting in a space in the interdigital region which can be exploited by ticks. In those antelope species which we examined the axial corium is straight, thus limiting the space between the hooves. No abscesses were encountered on the feet of any of the wild antelope examined.

Howell et al[5] have described the southern distribution of *A. hebraeum* in South Africa. They indicate that it extends westwards through the coastal regions of the eastern parts of the Cape Province, where it follows the valleys of the Kei, Fish and Sundays rivers inland, and terminates beyond Humansdorp. Further west an isolated focus is present in the Mossel Bay area. The farm "Brakhill" is fairly close to the western extremity of this distribution range while the farm "Bucklands" and the adjoining Kudu Reserve are situated more centrally within this southern distribution range. The seasonal abundance of adult *A. hebraeum* recorded at "Bucklands" and the Kudu Reserve in the present survey corresponds to that previously recorded at these localities[6,9].

The geographic distribution of *R. glabroscutatum* has been described by MacIvor[8]. He states that this tick is virtually confined to the drier regions of the eastern Cape Province and is absent in the coastal areas. The majority of collections he records come from the zone 31° 30'-33° 45'S and 24° 21' - 26° 43' E with isolated foci to the west of this region. The farm "Brakhill" lies at the southern extremity of the major distribution zone, "Bucklands" and the Kudu Reserve at the eastern extremity and the Mountain Zebra National Park in the northern portion of this zone. The Bontebok National Park is one of the isolated western foci. The seasonal abundance of adult *R. glabroscutatum* at each of these localities was reasonably similar and corresponds to that observed earlier on kudu from "Bucklands" and it's vicinity[6].

While we do not wish to imply that the ticks *A. hebraeum* and *R. glabroscutatum* are the only predisposing factors in the occurrence of foot abscess we feel that the present study clearly indicates their important role in the aetiology of this condition.

In order to control both these ticks and hence reduce the incidence of foot abscess we recommend regular foot-dipping from August to December. It must be borne in mind that all acaricides are at a disadvantage when applied to the feet. This is because the feet are in constant contact with soil, grass, dew and other contaminants all of which will serve to remove or dilute the acaricide.

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ARTIKEL ARTICLE

THE EFFECT OF POST-MILKING TEAT DIPPING ON TEAT CANAL INFECTIONS

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ABSTRACT: Du Preez J.H. **The effect of post-milking teat dipping on teat canal infections.** *Journal of the South African Veterinary Association* (1987) **58** No. 3, 119-123 (En) Department of Medicine, Faculty of Veterinary Science, University of Pretoria, P/Bag X04, 0110 Onderstepoort, Republic of South Africa.

Teat canal infection (TCI) or colonization, subclinical mastitis (SCM) cases and other forms of intramammary infections (IMI) may persist despite regular post-milking teat disinfection. Spontaneous healing of TCI can occur and this points to the dynamic state of the reactions of TCI etc. Disinfecting teat dipping does not necessarily prevent new TCI and IMI. Teat dipping may, however, be applied with good effect in the prevention of new udder and teat canal infections. Several factors predispose to TCI e.g. poor management, hygiene and animal husbandry.

Key words: Disinfecting, post-milking teat dipping, teat canal infection, teat canal swab sample.

INTRODUCTION

Teat dipping or spraying with a germicidal solution immediately after every milking is regarded as the single most effective practice for the prevention[22] and control[23 24 25] of new intramammary infection (IMI) in lactating dairy cows. However, not all types of infection are equally reduced by germicidal teat dips[10 20]. The use of a teat dip for the control of bovine mastitis was first reported by Moak[17] in 1916 when he controlled *Streptococcus agalactiae* infection by dipping the cow's teats in a weak solution of pine oil. However, the procedure did not receive serious attention until over 24 years ago, when Newbould & Barnum[21] reported on the effect of germicidal agents in reducing the number of staphylococci by teat dipping. Infections by pathogenic bacteria of the type that are spread primarily from quarter to quarter and from cow to cow during the milking process, are reduced markedly by germicidal dips; *S. agalactiae* and *Staphylococcus aureus* are the most common examples of such mastitogens. However, pathogens such as streptococci other than *S. agalactiae* and coliform bacteria, which are contracted frequently from the cow's environment, are controlled less effectively by teat dipping. Although some researchers have found teat dips efficacious for reducing such infections[29], others have found them relatively ineffective[4 14].

Philpot & Pankey[23] and Pankey[22] reported on the effectiveness of different generic groups of teat dips/sanitizers such as iodophors, quaternary ammonium compounds, chlorhexidines, sodium hypochlorites, acrylic latex (physical barriers), and dodecyl benzene sulfonic acid (DDBSA) for prevention of IMI in lactating cows.

Forbes & Herbert[12] proved that most *S. aureus* and *S. epidermidis* udder infections originate in the teat canal without any initial increase in somatic cell count of the milk. The majority of infections arising during lactation are preceded by teat canal infections (TCI). Adams, Rickard & Murphy[1] point out that in order for microorganisms, including *S. agalactiae,* to cause udder infection, they first have to establish themselves in the teat canal. Forbes[11] proved that in certain cases of new udder infections caused by *Micrococcaceae* and *Corynebacterium bovis* (as diagnosed by examining teat wall puncture milk samples), the bacteria are present in the teat canal for weeks before becoming established in the udder parenchyma. McDonald[16] pointed out that bacterial contamination of the teat canal is very common. All the common udder pathogens are capable of colonising the teat canal; *S. aureus* can persist there for up to 18 weeks without penetrating into the gland[3]. Mastitis is the most important and costly disease in dairy herds when adequate control procedures are not instituted[18 19]. Teat dipping can contribute to the diminishing of such losses.

The primary aim of this investigation was to establish whether post-milking disinfection by teat dipping eliminates already existing TCI and prevents new TCI and IMI and whether TCI can be spontaneously eliminated. A second objective was to determine the prevalence of TCI and other subclinical udder health conditions in herds receiving no post-milking teat dipping and to compare these herds with others in which teat dipping was practiced.

MATERIALS AND METHODS

Experimental animals

The investigation was performed on Friesian dairy cows in herds maintained on the Transvaal Highveld on a zero grazing system (Herd No. 1) and on a semi-intensive system (Herd No. 2). Herd No. 1 was milked three times a day by hand and Herd No. 2 twice a day by milking machine. The cows varied in age, lactation number, daily milkyield and stage of lactation (2-8 months). All the cows were clinically healthy and in good condition. The standard of management, animal husbandry and hygiene in Herd No. 1 was relatively poor and in Herd No. 2 relatively good.

Collection of parallel foremilk (FMS) and teat canal swab samples (TCSS)

Sampling routine: Foremilk (FMS) and teat canal swab samples (TCSS) were obtained immediately before milking and after thorough udder washing with clean run-

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ning water, drying with a disposable paper towel and disinfection of the teat tip 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[6,7]. Before implementing our sampling routine, we had established the efficacy of the disinfection process by culturing swab samples from 20 clinically healthy teat tips subsequent to disinfection; all were bacteriologically negative. All samples were transported on ice and analysis proceeded within 6 hours of sampling.

Somatic cell counts (SCC)

The SCC determination was done according to the method described by Van den Heever et al.[28] and the IDF bulletin[15].

Isolation and identification of bacteria

Facultative and microaerophylic bacteria were isolated as described by Du Preez[6] and Du Preez & Greeff[7] and the IDF Bulletin[15]. Species identification was done according to methods and criteria described by Cowan & Steel[5]. Specific attempts to culture anaerobic bacteria were excluded for practical reasons.

Statistical evaluation

The log-linear model was used for statistical analyses[9]

Health status of quarters and classification

The health status of quarters, determined according to data obtained by examination of FMS, was firstly classified according to the International Dairy Federation's (IDF) criteria[13,27] and secondly according to IDF criteria based on the results of cytological examinations of the FMS and bacteriological examination of the TCSS. The IDF criteria do not make provision for the classification of TCI, neither do they acknowledge its existence. Because all the corresponding TCSS of quarters diagnosed and classified according to the IDF criteria as latent udder infections (LUI) were bacteriologically positive and because it is assumed that the presence of bacteria in TCSS indicates TCI, the latter state must according to the existing IDF criteria be classified in the same category as LUI.

Post-milking disinfecting teat dipping

No post-milking teat dipping was practised for months in Herd No. 1 before commencing with this experiment, but in Herd No. 2 it was routinely applied. All the teats of the cows in the experimental groups (Herd No. 1 and Herd No. 2) were immersed in chlorhexidine gluconate 0,75% m/v ("Hibitane" G 1000, ICI (South Africa), Pharmaceuticals Ltd, Johannesburg) after milking. During the 3 months before teat dipping was introduced (Herd No. 1) as well as during the period of experimental teat dipping, a teat and milking ointment (Repvet (Pty) Ltd, Johannesburg) was also used before milking the cows.

RESULTS

From Table 1 it is apparent that the prevalence of the various udder health states differed according to the type of sample examined. Such differences become still more obvious from a comparison of the percentage values of udder quarters classified according to their health condition. There was a decrease of 1,2% and an increase of 9,9% of normal (N) quarters and a decrease of 6,2% and 17,3% of TCI cases diagnosed according to the FMS and TCSS before and after 3 weeks of teat dipping. Although there was a decrease in TCI after 3 weeks of teat dipping there was an increase of 7,5% and 7,4% of SCM cases diagnosed according to the FMS and TCSS after 3 weeks of teat dipping. Some normal teat canals (10%), as established according to the TCSS before teat dipping commenced, developed TCI during the teat dipping period; *S. aureus, S. agalactiae, C. bovis,* coagulase-negative staphylococci and *Escherichia coli* were cultured from these cases. The prevalence of TCI diagnosed according to the TCSS was 49,4% and 38,3% higher than diagnosed according to the FMS method in this herd before and after 3 weeks of teat dipping.

From Table 2, (control group) it is apparent that the health status of the teat canals as well as the udder parenchyma is dynamic. There was a spontaneous healing (self-cure) rate of 12,2% and 8,6% in TCI cases diagnosed according to the FMS and TCSS during the 3 weeks in which the experiment was conducted. The prevalence of N quarters, diagnosed according to both methods during the 3 weeks, remained approximately unchanged; this is at variance with SCM cases where there was an increase of 8,5% and 8,6% diagnosed according to the FMS and TCSS during the experimental period of 3 weeks.

From the results in Table 3 it is apparent that the prevalence in Herd No. 1 of cases of TCI, diagnosed on the basis of results of examination by the FMS and TCSS methods is much higher than in Herd No. 2. The same applies to cases of SCM and therefore to the prevalence of N quarters in Herd No. 2, diagnosed according to both methods. The prevalence of TCI, diagnosed according to the TCSS, is 22,5% and 11,3% higher in herd Nos. 1 and 2, respectively, than when diagnosed according to the FMS.

DISCUSSION

After 3 weeks of disinfecting teat dipping, the prevalence of TCI within Herd No. 1 decreased by 17,3% and 6,2% respectively, when assessed according to the TCSS and FMS diagnostic methods, although after 3 weeks of teat dipping, *S. aureus, S. agalactiae, C. bovis,* coagulase-negative staphylococci and *E. coli* were isolated by the author before teat dipping was instituted. Pankey[22] discussed the decreasing incidence of new IMI which is the consequence of post-milking teat sanitizers. Natzke[20] stated that, with the exception of teatcup pasteurization, the application of hygienic procedures in herds where teat dipping is practiced did not result in (the prevention of) the transfer of predominant mastitis pathogens during the milking routine. Many hygienic practices exert their effect by preventing spread of mastitis pathogens. The teat dip has several actions by which it aids in reducing new IMI[20]. Firstly, it kills organisms which have been transferred to the teat during the milking process or by direct environmental contact; secondly it prevents colonisation of the teat by organisms such as *S. aureus,* thus eliminating a major source of new infection, (this study indicates that this is not always the case) and thirdly it remains on the teat as

Table 1: Experimental group: The prevalence of normal quarters (N), teat canal infections (TCI), aseptic (ASM) and subclinical mastitis (SCM) cases in hand-milked lactating diary cows before and 3 weeks after instituting regular post-milking teat dipping with chlorhexidine gluconate 0,75% m/v (Herd No. 1, n = 162 quarters)

Status of quarter	Before commencing with teat dipping, Day 1		After 3 weeks of teat dipping	
	Sampling method		Sampling method	
	Foremilk sample (%) (FMS)	Teat canal swab sample (%) (TCSS)	Foremilk sample (%) (FMS)	Teat canal swab sample (%) (TCSS)
N	88 (54,3)	8 (4,9)	86 (53,1)	24 (14,8)
TCI/LUI	52 (32,1)	132 (81,5)	42 (25,9)	104 (64,2)
ASM	2 (1,2)	0 (0)	2 (1,2)	0 (0)
SCM	20 (12,3)	22 (13,5)	32 (19,8)	34 (20,9)
Total	162 (100)	162 (100)	162 (100)	162 (100)

Table 2: Control group: Prevalence of N, TCI, ASM and SCM in hand-milked lactating dairy cows at day 1 and after 3 weeks where no teat dipping was applied (Herd No. 1, n = 82 quarters)

Status of quarter	Day 1		After 3 weeks	
	Sampling method		Sampling method	
	(FMS) (%)	(TCSS) (%)	(FMS) (%)	(TCSS) (%)
N	45 (54,9)	5 (6,1)	48 (58,5)	5 (6)
TCI	26 (31,7)	66 (80,5)	16 (19,5)	59 (71,9)
ASM	1 (1,2)	0 (0)	1 (1,2)	0 (0)
SCM	10 (12,2)	11 (13,4)	17 (20,7)	18 (22)
Total	82 (100)	82 (100)	82 (100)	82 (100)

Table 3: Comparison of the prevalence of N, TCI, ASM and SCM cases in Herd No. 1* (hand-milked and no teat dipping) and Herd No. 2** (machine-milked and post-milking teat dipping with chlorhexidine gluconate 0,75% m/v)

Status of quarter	No teat dipping		Post-milking teat dipping	
	Herd No. 1, handmilked		Herd No. 2, machine-milked	
	Milk sampling method			
	Foremilk sample (FMS) (%)	Teat canal swab sample TCSS) (%)	FMS (%)	TCSS (%)
N	849 (59,4)	528 (36,9)	210 (62,8)	172 (51,5)
TCI/LUI	241 (16,8)	562 (39,3)	19 (5,7)	57 (17,0)
ASM	172 (12,0)	118 (8,3)	87 (26,0)	83 (24,9)
SCM	168 (11,75)	222 (15,52)	18 (5,4)	22 (6,6)
Total	1 430 (100)	1 430 (100)	334 (100)	334 (100)

* Standard of management, animal husbandry and hygiene in Herd No. 1 was relatively poor and in Herd No. 2** was relatively good.

a residue, which reduces the number of organisms present at the next milking[20]. Eberhart et al.[8] pointed out that teat dipping significantly reduced new IMI caused by *S. aureus,* streptococci (other than *S. agalactiae*), coagulase-negative staphylococci and *C. bovis* and also reduced clinical mastitic cases caused by infections established during the trial. However, the dip did not reduce new IMI or clinical mastitis caused by coliform bacteria. Sheldrake & Hoare[25] also stated that teat dipping is effective for controlling bovine IMI. The effectiveness of a teat dip with a 1,94% v/v solution of DDBSA for the prevention of IMI was determined in cows experimentally challenged with *S. agalactiae* and *S.* aureus[2]. DDBSA was effective in resisting the experimental challenge.

It would appear from the results of the present study that teat dipping brings about a slight (not statistically significant) decrease in the prevalence of TCI (Table 1) but allowance should also be made for spontaneous healing as showed in the control group (Herd No. 1, Table 2). Steward & Philpot[26] showed that IMI caused by *Micrococcus* species which have low virulence are often transitory and most infections were eliminated spontaneously. It appears that teat dipping does, in fact, prevent to a great extent new cases of TCI in that there was no increase in the prevalence of TCI after 3

weeks of teat dipping although new TCI can occur as already discussed. Since hand-milking with teat salve had already been applied for 3 months before teat dipping was introduced, the probability that the milking salve could eliminate or prevent TCI is slight. Despite teat dipping and hand-milking with a milking salve, new TCI occurred during the teat dipping period in the teat canals of individual cows which were free of TCI before teat dipping commenced (Table 1). Several factors predispose to TCI/udder infections such as poor management. Hygiene and animal husbandry could have given rise to this condition as well as to the increase of SCM cases in the control group of Herd No. 1 (Table 2).

The prevalence of TCI and SCM cases in the hand-milked herd (No. 1) was higher than in the herd (No. 2) in which machine milking and post-milking teat dipping were practiced (Table 3), possibly because the standard of management, animal husbandry and hygiene in Herd No. 1 was relatively poor in comparison with Herd No. 2.

Various TCI and IMI existed despite regular disinfecting teat dipping (Table 3, Herd No. 2) for months before TCSS and FMS sampling and bacterial culturing. Many quarters in this study, diagnosed according to the FMS and classified as normal according to the IDF criteria, were proved to be true cases of TCI by means of teat canal swab sampling. An explanation for this phenomenon could be that the bacteria colonise in the teat canal so densely, or the attachment of the colonising bacteria to the teat canal tissue were so good that with foremilk sampling no contamination of milk takes place from the teat canal. With teat canal swab sampling the bacteria in the teat canal are removed together with the keratin and epithelial cells, by the swab for the purpose of culturing. The greater mechanical friction caused by teat canal swab sampling allows greater diagnostic efficiency than the flushing action during foremilk sampling. The same principle is applicable to the data in Tables 1 & 2. With the use of TCSS instead of FMS for diagnosing udder infection, fewer quarters are indicated as being normal (Tables 1-3); in other words, the teat canal swab sampling is a more sensitive and accurate diagnostic method for determining the health status of quarters. With the use of the FMS to establish the status of the quarter, false negative results were obtained in more than 20% of the cases (Table 3, Herd No. 1), when compared with the results of the TCSS.

Statistical conclusions

There is no statistically significant difference in the prevalence of N quarters, cases of TCI, ASM and SCM, diagnosed according to the FMS and TCSS before and three weeks after teat dipping (Table 1). This applies equally to the control group (Table 2). Teat dipping alone therefore does not contribute significantly towards eliminating the various forms of udder infection and TCI in a herd in which factors many predispose to mastitis.

CONCLUSIONS

1. TCI and forms of IMI may persist despite regular disinfecting teat dipping.
2. Disinfecting teat dipping does not necessarily prevent new TCI and/or IMI. Teat dipping may, however, be applied with good effect in the prevention of new udder and teat canal infections.
3. The prevalence of cases of TCI and SCM in a dairy herd does not statistically decrease when regular disinfecting post-milking teat dipping is practised.
4. The prevalence of cases of TCI and SCM diminished in dairy herds where the standard of management, animal husbandry and hygiene is relatively good.
5. TCI can only be diagnosed accurately by employing TCSS.
6. In the routine examination of FMS, many cases of TCI (between 10-20% or more) are not detected.
7. The health status of the teat canal and udder parenchyma is dynamic and spontaneous healing (self-cure) of TCI and SCM cases can occur.
8. By using only the bacterio-cytological results of FMS, the IDF criteria for the classification of various forms of SCM do not facilitate an accurate classification of the health status of the udder.

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ARTICLE ARTIKEL

EFFECTS OF CERTAIN ANAESTHETICS ON PLASMA METABOLITE CONCENTRATIONS IN THE BABOON *(PAPIO URSINUS)*

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ABSTRACT: Van der Merwe J.N.; Du Bruyn D.B.; Van der Walt W.H.; Sly M.R. **Effects of certain anaesthetics on plasma metabolite concentrations in the baboon *(Papio ursinus)*.** *Journal of the South African Veterinary Association* (1987) **58** No. 3, 125-128 (En) National Food Research Institute, Council for Scientific and Industrial Research, P.O. Box 395, 0001 Pretoria, Republic of South Africa.

Four groups of 12 baboons each were sedated for a period of 3 hours by means of one of 4 anaesthetic treatments. The treatments involved repeated administration of either ketamine or phencyclidine, or initial sedation with one of these followed by pentobarbitone. A number of plasma metabolites were measured at 30 min intervals. With one exception, there were no significant differences between the 4 individual anaesthetic treatments; there was a small, but significant, decrease in plasma calcium (3,4%) in the group receiving phencyclidine followed by pentobarbitone. There were, however, significant changes with time. Plasma inorganic phosphorus and magnesium concentrations in all the groups rose similarly, reaching mean respective increases of 21,8% and 7,7% after 3 hours. In a number of cases divergent time trends were observed between the pair of groups given pentobarbitone and the pair receiving only ketamine or phencyclidine. Pooling the results from each of these pairs of treatment groups doubled the overall group sizes to 24 animals each and enabled significant differences between the divergent time trends to be detected. In the former pair plasma zinc increased (by a maximum of 16,1%) and protein decreased (by a maximum of 4,0%), while in the latter pair alkaline phosphatase and cholesterol levels both increased (up to maximums of 7,8% and 5,2%, respectively). No significant changes with time were found to be attributable to diurnal variation. Some of the changes in plasma metabolite concentrations following anaesthesia are such that due care should be exercised in their determination and interpretation in anaesthetised baboons.

Key words: Anaesthesia, plasma metabolites, baboon.

INTRODUCTION

When blood samples have to be obtained from large and potentially dangerous animals such as baboons, it is necessary to minimize the risk to the handler and stress to the animals. The use of chair restraints[5] or blood sampling by means of a tether system[1] or by remote conrol[7] is often impractical and chemical restraint by anaesthesia is usually the method of choice. Anaesthetics, however, are known to elicit effects on various blood constituents. Steyn has recorded some of the changes in blood chemistry and haematology that take place in the baboon over a one hour period of phencyclidine anaesthesia[15]. He found that the administration of the anaesthetic and the concomitant stress caused a considerable number of changes, though most were of short duration. Steyn has further suggested that the values for various blood constituents found for a particular group of baboons kept under particular dietary and environmental conditions should not be directly applied to other sets of baboons[16].

The present study was undertaken as a prerequisite to monitoring the appearance in the bloodstream of various nutrients administered to baboons by stomach tube. To establish the baseline levels and changes thereof that were particular to the colony and pertinent to the proposed study, consecutive plasma samples were taken from baboons anaesthetized for a period of 3 hours. Four different anaesthetic treatments were investigated, in order that the one eliciting the least changes could then be used on a routine basis.

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MATERIALS AND METHODS

Animals and Experimental Design

Twenty-four chacma baboons *(Papio ursinus;* 12 males and 12 females), originally caught in the wild, and of estimated ages between 2 and 3 years (weightspread 10-15 kg for females and 15-30 kg for males), were used in the study. Twelve animals (6 males and 6 females) were allocated at random to each of four anaesthetic treatments recommended for use in baboons[4]. The availability of 24 baboons meant that two treatments could be run in one session. A period of four weeks was allowed to elapse from the end of the first session before the animals were re-used for the second session of two treatments. In order to ensure that each treatment was conducted over the same time period each day, only four baboons were processed per day. They were anaesthetised at 09h00 following an overnight fast from approximately 16h00.

In the first session, the two groups were initially sedated with 5 mg kg^{-1} body weight intramuscular ketamine hydrochloride (Ketalar, Parke-Davis) and in the second session with 1 mg kg^{-1} body weight intramuscular phencyclidine hydrochloride (Syclan, Centaur Labs). For each session, anaesthesia was maintained either with repeated doses of the initial anaesthetic, or by the intravenous injection of 6 mg kg^{-1}body weight sodium-pentobarbitone (Sagatal, Maybaker (SA)) given 40 min after the initial anaesthetic was administered. It was necessary to repeat ketamine, phencyclidine and pentobarbitone administrations at intervals of approximately 25, 50 and 30 minutes, respectively. The zero timepoint blood sample (10 ml) was drawn from the femoral vein into Venoject tubes (Terumo) containing lithium heparin as soon as sedation became effective,

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Table 1: Anaesthetic treatment groups

	Group	No. of baboons	Initial sedation	Prolonged anaesthesia
Session 1	1	12	Ketamine.HCl	Ketamine.HCl
	2	12	Ketamine.HCl	Na-Pentobarbitone
Session 2	3	12	Phencyclidine.HCl	Phencyclidine.HCl
	4	12	Phencyclidine.HCl	Na-Pentobarbitone

which was usually after about 10 min. Further blood samples were drawn at half hour intervals for a total of 3 hours, and plasma fractions stored frozen.

Four weeks after the second treatment session was completed, the 24 baboons were again divided randomly into two equal groups in order to ascertain the changes in plasma metabolite levels that might be attributable to that part of the diurnal variation occurring between 09h00 and 12h00. The animals from the first group were given phencyclidine hydrochloride at 09h00 (administered in rapid succession to six animals per day for 2 days), and the second group the same anaesthetic at 12h00. A single 10 ml blood sample was drawn from each baboon as soon as sedation was achieved, and plasma prepared.

Analytical Methods

Plasma inorganic phosphorus concentrations were determined by a phosphomolybdate method using ferrous sulphate as reducing agent[17], and concentrations of zinc, calcium and magnesium by atomic absorption spectrophotometry. Diagnostic kits from Boehringer-Mannheim were used to measure plasma total cholesterol (CHOD-PAP Method) and total protein concentrations, and alkaline phosphatase activities.

Statistical Methods

In order to reduce between-baboon variance, the percentage change in concentration of each plasma metabolite has been plotted relative to time zero. For each metabolite the significance of differences with time and the significance of differences between individual treatments was examined.

Plasma variables were related to the time over which anaesthesia was maintained by means of random coefficient growth models (third degree polynomial plots) for repeated measurements[6]. If for any treatment the response changed significantly with time at a nominal 5% level, then the expected response at each time point was compared with that of time zero using the Bonferroni method[10] to ensure a simultaneous significance level of at most 5%. The response-time curves for the different anaesthetic treatments were compared by means of a Wilk's multivariate analysis of variance procedure[10].

The statistical representation of the data shown in Fig. 1(a) and 1(d)-(i) is based on the Studentized extreme deviation[2,11] and may be explained as follows: if a group mean curve lies outside the 95% critical range for differences about the grand mean response, then it is different at a simultaneous 5% significance level from one or more of the other group means at that given time. Conversely, if all curves lie within this critical range (bordered by the hatch marks), then there is no statistical difference between any two of the groups.

RESULTS

There were no significant ($p > 0.5$) group differences in plasma inorganic phosphorus concentrations because the plot for each group lies within the 95% critical range hatch marks (Fig. 1a). However, plasma inorganic phosphorus in all groups showed a highly significant ($p < 0,001$) increase with time. On pooling the results (Fig. 1b), mean inorganic phosphorus concentrations were seen to eventually reach levels 21,8% above initial values. There were also no group differences in plasma magnesium concentrations, levels of which rose ($p < 0,001$) similarly by a mean maximum of 7,7% after 3 hours (Fig. 1c).

Although there were no significant differences between the 4 individual anaesthetic groups in respect of plasma total cholesterol concentrations (Fig. 1d) (all plots fell within the 95% critical range hatch marks), levels in the groups receiving repeated injections of either ketamine or phencyclidine showed a significant ($p < 0,01$) increase with time, while those receiving pentobarbitone showed no change. Confirmation of a divergent time trend was obtained by pooling the results from those pairs of groups which behaved similarly; the larger ($n = 24$) group sizes obtained allowed a maximum significant difference ($p < 0,001$) of 5,2% to be detected after 3 hours (Fig. 1e). Significant divergent trends between the pooled results of the pair of groups given pentobarbitone and the pooled results of the pair given repeated injections of initial anaesthetic were also observed for plasma zinc, alkaline phosphatase and total protein levels ($p < 0,001$ for each metabolite), though in each case there were no individual group differences.

Only in the groups receiving pentobarbitone did the plasma zinc concentrations change significantly with time; by 3 hours levels in the combined groups had increased to a maximum of 16,1% above initial values (Fig. 1f). In the case of alkaline phosphatase, only levels in the groups receiving repeated administrations of ketamine or phencyclidine changed. They rose significantly above starting values towards the end of the period of anaesthesia, but the increase amounted to a maximum of only 7,8% at 3 hours (Fig. 1g). Plasma total protein concentrations in those groups receiving pentobarbitone decreased, though the maximum drop only amounted to a fall 4,0% below initial values (Fig. 1h). Plasma total protein in the groups receiving only ketamine or phencyclidine showed no changes.

Plasma calcium concentrations in the group receiving phencyclidine followed by pentobarbitone decreased, reaching a maximum fall of 3.4% after 3 hours. Although small, this fall was sufficient to produce significant ($p = 0,001$) differences from the other three groups (Fig. 1.1).

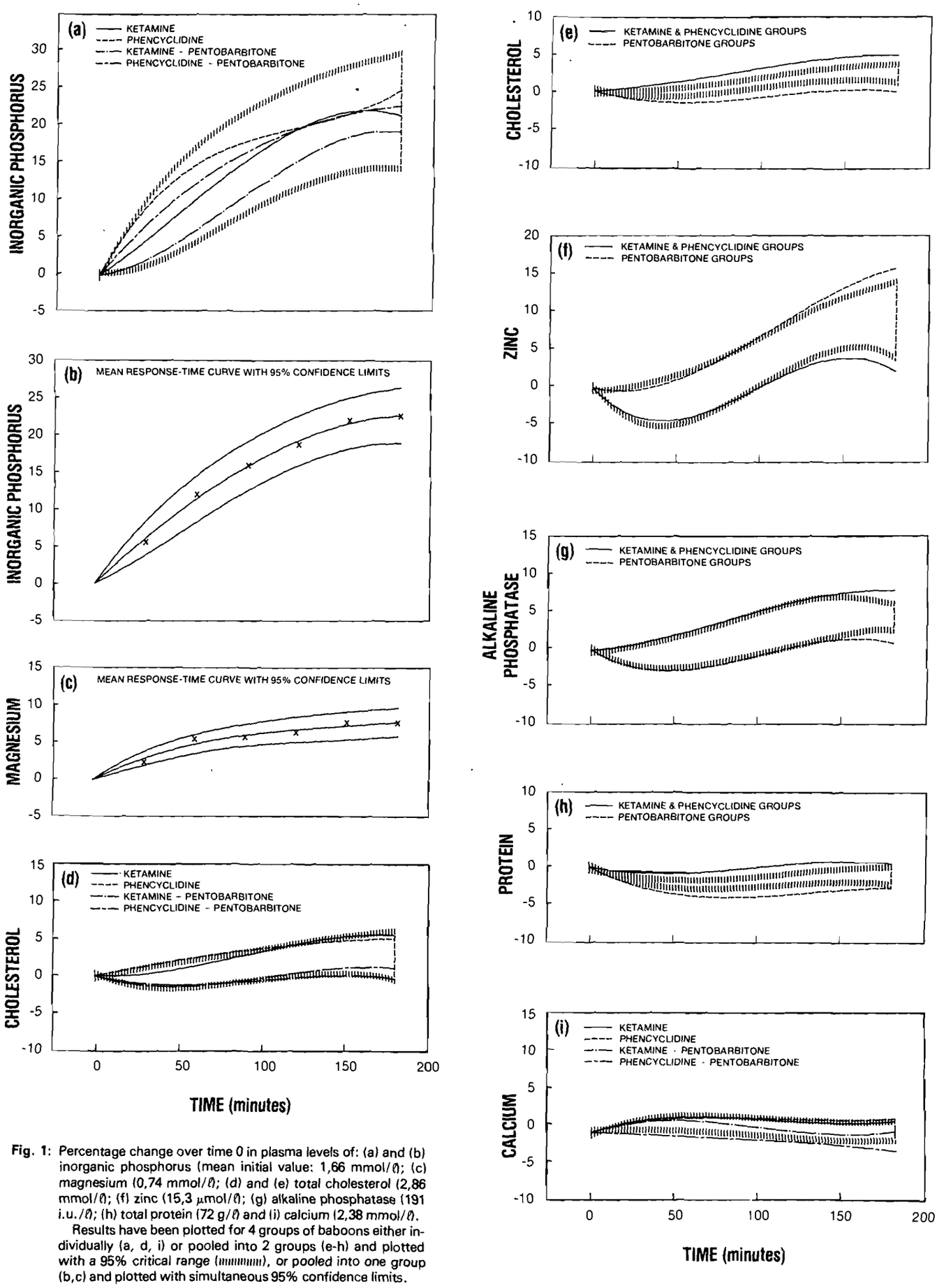

Fig. 1: Percentage change over time 0 in plasma levels of: (a) and (b) inorganic phosphorus (mean initial value: 1,66 mmol/ℓ); (c) magnesium (0,74 mmol/ℓ); (d) and (e) total cholesterol (2,86 mmol/ℓ); (f) zinc (15,3 μmol/ℓ); (g) alkaline phosphatase (191 i.u./ℓ); (h) total protein (72 g/ℓ) and (i) calcium (2,38 mmol/ℓ).
Results have been plotted for 4 groups of baboons either individually (a, d, i) or pooled into 2 groups (e-h) and plotted with a 95% critical range (▮▮▮▮▮▮▮▮▮), or pooled into one group (b,c) and plotted with simultaneous 95% confidence limits.

Mean (± SE) plasma metabolite concentrations, measured at 09h00 and 12h00, respectively, in order to assess the possible contribution of the diurnal variation to the apparent effects of the anaesthetics, were: 1,72±0,08 and 1,54±0,05 mmol inorganic phosphorus/ℓ; 2,83±0,09 and 2,67±0,13 mmol cholesterol/ℓ; 0,786±0,014 and 0,735±0,020 mmol magnesium/ℓ; 15,9±0,8 and 15,5±1,2 μmol zinc/ℓ; 194±26 and 209±49 i.u. alkaline phosphatase/ℓ; and 2,59±0,03 and 2,68±0,04 mmol calcium/ℓ. Only in the case of calcium was there a significant difference (Mann Whitney U test[14]; 5% level).

DISCUSSION

The results of the present study show that ketamine and phencyclidine, alone or in combination with pentobarbitone, can cause significant changes in the concentrations of a number of plasma metabolites and that there were tendencies for certain anaesthetics to behave differently. None of these changes can be attributed to that part of diurnal variation which occurred between 09h00 and 12h00, save possibly in the case of calcium, where a 3% increase in concentration over the time course of the anaesthetic study may have helped to mask falls.

Of the metabolites measured, inorganic phosphorus increased to the largest extent (21,8%). It is thought that there are increases in membrane permeability subsequent to anaesthesia[18], and it is not inconceivable that such changes could be accompanied by the release of intracellular phosphorus; large amounts of phosphorus can apparently be mobilized from this source[8]. Alternative means by which increases in plasma inorganic phosphorus may be brought about include a breakdown of phosphocreatine following muscle injury[9], stress[13] or decreased excretion of inorganic phosphorus. Increases in plasma inorganic phosphorus have also been found in rats anaesthetised with urethane[12], ethanol or pentobarbital[9], and in impala given xylazine[3]. Steyn[15], however, found no significant changes in serum inorganic phosphorus concentrations in baboons over a one hour period of phencyclidine anaesthesia. After one hour the average rise in the present study was 8%.

Though most of the other anaesthetic-induced changes in metabolite concentrations were not of the same magnitude as the changes in plasma inorganic phosphorus, they may still be of relevance because any such change warrants due consideration as to the possible consequences it might have on the interpretation of the results of an experiment. The changes found by Steyn[15] were not always in the same direction or of the same duration as those found in the present study. For example, he found that calcium increased and protein decreased with time, while cholesterol and alkaline phosphatase levels remained unaltered. These differences in findings emphasise the importance of characterising the changes particular to any given baboon colony with its own set of colony conditions (e.g. caging, diet, degree of stress involved in anaesthetising the animals, dose of anaesthetic, etc.).

The divergent time trends in changes of cholesterol, zinc, alkaline phosphatase and protein levels between groups given intramuscular ketamine or phencyclidine alone and those given intravenous pentobarbitone, may be attributable to the route of administration or differences in the mechanism of action of the anaesthetics. Halothane or other gaseous products may be more acceptable for long-term anaesthesia because they would obviate the need for repeated administration of shorter-acting anaesthetics. However, their use would be impractical when large numbers of animals are involved.

The finding that various plasma metabolites do change when baboons are subjected to anaesthesia demonstrates the need for caution when these metabolites are the object of interest in any study. In some cases it may be possible to choose an anaesthetic which gives rise to the least change in the variable of interest, and the examination of a wider range of anaesthetics may extend the number of options available. In order to limit variation in the values it would be advisable to follow a strict schedule in collecting specimens by always allowing the same interval between the administration of the anaesthetic agent and the collection of blood, as suggested by Steyn[15]. Gentle handling, the use of the same dosage level and the elimination of all unnecessary stress-provoking procedures should be part of the routine followed whenever animals are handled for the sampling of blood.

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GEVALVERSLAG CASE REPORT

CHRONIC SPLENITIS AS A POSSIBLE COMPLICATING FACTOR IN CALVES INOCULATED WITH AN ATTENUATED REDWATER VACCINE

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ABSTRACT: Ehret W.J.; Potgieter F.T.; De Waal D.T.; Van der Lugt J.J.; Van der Vyver F.H. **Chronic splenitis as a possible complicating factor in calves inoculated with an attenuated redwater vaccine.** *Journal of the South African Veterinary Association* (1987) **58** No. 3, 131-134 (En) Johannesburg City Health Department, P.O. Box 1477, 2000 Johannesburg, Republic of South Africa.

In two consecutive years a total of 3810 calves were inoculated with the Onderstepoort redwater live-blood vaccine, containing attenuated strains of *Babesia bigemina* and *Babesia bovis*. Twenty calves died 8-13 days later due to *B. bigemina*-infection. Necropsies were performed on 19 of these animals. In 11 calves the spleen was distinctly smaller than normal and its morphology was distorted, and showed changes of chronic splenitis. Histological lesions in the spleens of 6 of the 11 calves are described. The potentiating effect of the splenic lesions to reduce the ability to contain the vaccine reaction, is discussed.

Key words: Redwater attenuated vaccine, calves, chronic splenitis.

INTRODUCTION

Babesiosis or redwater, caused by *Babesia bigemina* and *Babesia bovis*, is regarded as one of the most economically important diseases of cattle in the Republic of South Africa[2]. Vaccination of cattle against both forms of the disease is widely practised in this country using the Onderstepoort live-blood vaccine containing both *B. bigemina* and *B. bovis*. Before 1982 severe vaccine reactions were occasionally reported with the "old" vaccine[4], but with the inclusion of attenuated strains of *B. bovis* and *B. bigemina* into the vaccine, this problem has largely been overcome. Both *Babesia* spp. are of low virulence and give solid protection against local strains[4 5].

The observation by Smith & Kilborne[12] that young animals have an innate or non-specific resistance to *Babesia* infections, was later confirmed by other workers[10 15]. The infection of young calves in an enzootic area is thus generally considered to be symptomless[10]. It is therefore recommended that the vaccine be given to young animals less than 9 months of age, since severe vaccine reactions are sometimes seen in older cattle.

This paper deals with deaths due to severe vaccine reactions in predominantly *Bos taurus*-crossbred beef calves 4-6 months of age, the majority of which suffered from a chronic splenitis.

HERD HISTORY

The herd under review is the Johannesburg City Council's crossbred beef herd which is run on two farms. Farm 1 is 40 km north and Farm 2 is 25 km south of Johannesburg. The average cattle population on the two farms over the last 15 years has been 6 800 animals. Foundation breeds used in the herd consisted primarily of various *Bos taurus* breeds, in particular Simmentaler, Hereford and Charolais. Limited infusion of various *Bos indicus* breeds has also taken place. The breed composition of individual animals does, however, vary markedly. The cattle graze irrigated, predominantly rye grass, pastures for most of the year. During the latter half of summer and autumn strategic use is made of natural veld, which is generally adjacent to the farm boundaries.

Tick control, via spray race application of acaricides with different residual effects, has been regularly practised on both farms over the years, with the frequency varying from weekly to three weekly depending on whether the cattle were on natural veld or artificial pastures.

Until 1970 the Onderstepoort "old" redwater live-blood vaccine had been used regularly on both farms. Vaccination was again introduced in 1976 on Farm 1 and in 1978 on Farm 2. Since 1982 the vaccine containing the attenuated strains has been used. During the period 1967 to 1986 sporadic fatal cases of naturally acquired redwater have occurred on the two farms, and *B. bigemina* was incriminated on each occasion where positive identification was made.

Since 1976 a total of 19 634 calves, less than 6½ months of age, have received redwater vaccine. Three deaths occurred in 1976 on Farm 1, 9-10 days following administration of the vaccine. These calves were 56-90 days old and in each calf *B. bigemina* was incriminated as the cause of death. Twenty calves out of a total of 3810 vaccinated, died 8-13 days after vaccination during the period 1985-86. These calves were 127-191 days old.

RESULTS

Pathology: In each of the 20 calves that died 8-13 days after being inoculated with the redwater vaccine, thin smears of peripheral blood stained with 10% Giemsa revealed parasitaemias of *B. bigemina* varying between 1% to about 10%.

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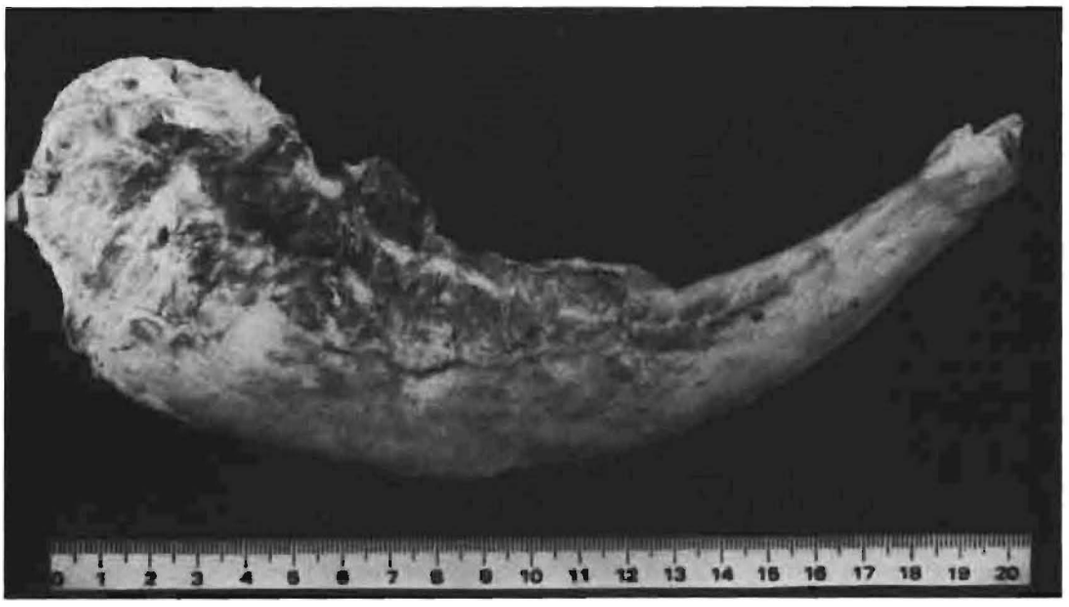

Fig. 1: Irregular nodular surface, marked distortion of morphology and reduced size of spleen. Scale in centimetres.

Necropsies were performed on all but one of the calves. Varying degrees of anaemia, icterus, haemoglobinuria, hepatomegaly and pulmonary oedema were present in the majority of cases.

In 11 animals the spleen was distinctly smaller than normal and its morphology was distorted by areas of fibrosis and nodules of varying size (Fig. 1). Extensive fibrous adhesions were evident between the capsule of the spleen and the diaphragm and the ruminal wall. On cut surface the capsule was moderately thickened and the parenchyma had a firm consistency.

Specimens of spleen of 9 of these 11 calves were fixed in 10% buffered formalin, routinely processed and stained with haematoxylin and eosin (HE). Selected sections of spleen were stained with periodic acid-Schiff (PAS), Gomori's methenamine silver (GMS) and Gram's stain. In 6 calves the macroscopical splenic lesions were similar, and autolytic changes were minimal. The capsule and trabeculae were moderately thickened by fibrous connective tissue, and infiltrated by mild to moderate numbers of lymphocytes and macrophages, a few plasma cells and an occasional giant cell (Fig. 2 & 3). Some of these phagocytes contained variable amounts of haemosiderin. In several areas fibrous connective tissue extended from the capsule and trabeculae, replacing adjacent parenchyma (Fig. 2). Mineralization of numerous elastic and connective tissue fibres and smaller blood vessel walls, as well as occasional thrombosis of lymphatics was evident (Fig. 2). Multiple randomly distributed, varying sized foci of necrosis of the red pulp were characterised by fibrin exudation and neutrophil infiltrations (Fig. 3). The red pulp was congested and the white pulp somewhat depleted of lymphocytes. No bacteria or fungal hyphae were seen with the PAS, GMS or Gram's stains. Capsular and trabecular thickening of the spleen was confirmed histologically in the other 3 calves, but detailed interpretation of lesions was not possible due to advanced autolytic changes.

In the remaining 8 calves, the spleen was moderately swollen, the parenchyma was soft and dark-red but did not show any changes of a chronic splenitis.

Bacteriology: Specimens of brain, liver, lung and kidney from two calves were submitted for routine aerobic bacterial isolations. *Pasteurella haemolytica* was isolated from the lung of one calf.

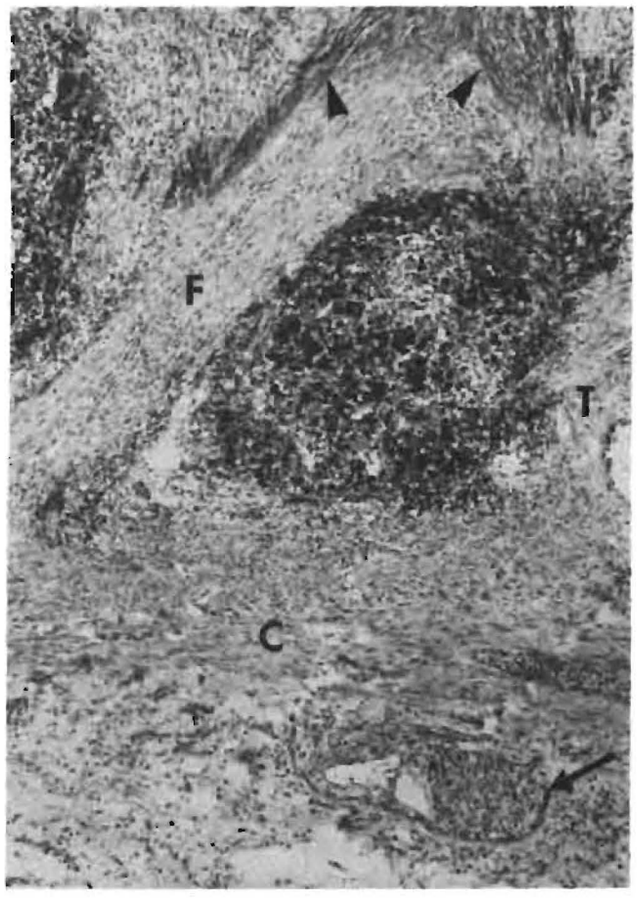

Fig. 2: Splenic capsule (C) and subcapsular trabecula (T) thickened by fibroplasia; fibrous connective tissue band (F) intersecting parenchyma. Note thrombosis of lymphatic (arrow) and mineralisation of connective tissue (arrowheads). HE X 200

Fig. 3: Thickened trabecula (T) infiltrated by inflammatory cells. Focus of necrosis (arrow) in red pulp. HE X 200

DISCUSSION

The mortality rate in calves, directly associated with redwater vaccinations on these particular farms from 1976 to 1984, was only 0,02% (3 out of 15 824 calves). The extremely high increase in losses during 1985/86 (10 out of 1 999 calves (0,5%) in 1985; 10 out of 1 811 calves (0,55%) in 1986) is a cause for concern.

The important role of the spleen in controlling *Babesia* infections has been studied in various rodent models[9, 11]. Protection against these parasites was attributed to the lymphoid tissue in the spleen, which acts as a source of phagocytes, antibody-forming cells and cellular immunity[13]. This is clearly illustrated in the more severe and often fatal disease that develops when a splenectomized animal is infected with *Babesia* parasites.

The spleen is apparently solely responsible for the important initial erythrophagocytosis of infected cells[14], with the liver and bone-marrow also performing this function at a later stage[7]. The rapidity of the splenic antibody response appears to be critical for the intitial development of acquired immunity[7]. The bone-marrow is only involved to a lesser degree in antibody formation, while the liver is not an antibody forming organ[13]. Todorovic et al.[14] observed that the onset of demonstrable antibodies in splenectomised rats was delayed when compared to intact animals. Non-splenic sources eventually take over the function of the spleen to maintain antibody formation over long periods[1, 13]. The spleen, however, still plays an essential part in the defence mechanism of the recovered host in dealing with antigenic variants of the parasite as they appear[9]. The complex interplay of the factors involved in the natural and acquired immunity to babesiosis has produced varying degrees of reliance on the spleen in the host-parasite association. It is our experience with the redwater vaccine that the average prepatent period for *B. bigemina* is 5,69 ± 3,26 days, while that for *B. bovis* is 10,0 ± 2,83 days. For this reason *B. bigemina* was associated with the death of these calves, while *B. bovis* parasites were probably present below detectable levels and difficult to identify in such mixed infections.

It is speculated that the chronic splenitis in 11 of the 20 calves, may have affected their ability to contain the infection during the crucial early phase of the reaction. During the preparation of this manuscript, 5 young cows (four 4-year-old and one 3-year-old) died on Farm 2 of *B. bigemina,* two of which showed macroscopical splenic lesions similar to those reported here. The 5 cows received redwater vaccine as calves, and they may have acquired the splenic lesions subsequent to vaccination. In the absence of adequate natural challenges, antibodies in a vaccinated herd will drop, and animals will become serologically negative[3]. Protective immunity after a single vaccination in the case of *B. bovis* appears to last for several years[8]. The duration of immunity to *B. bigemina* however, is unknown, and appears to break down in time and such animals could succumb when challenged[6].

During 13 years of experience in the Section of Pathology, VRI, chronic splenitis as described in this paper, has not been seen in calves originating from all over the Republic of South Africa. The specific cause of the splenitis is unknown, but the inflammatory changes indicated that it most probably had an infectious origin. The chronic nature of the lesions indicated that the splenic lesions developed earlier in life, possibly by local extension from a traumatic or inflammatory lesion in the rumen or reticulum. However, no lesions in the forestomachs or other chronic peritoneal lesions were associated with the splenitis in these animals. Examination of the pedigree of the affected calves also failed to reveal a familial association.

Further study on similar cases is necessary to determine the aetiology of the splenitis.

ACKNOWLEDGEMENTS

The Section of Photography, VRI, is acknowledged for the illustrations.

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SHORT COMMUNICATION **KORT MEDEDELING**

BLOOD CHEMICAL AND ELECTROLYTE CONCENTRATIONS IN THE MOUNTAIN REEDBUCK *REDUNCA FULVORUFULA*

J. DAUTH, R.H. KEFFEN+, J. VAN HEERDEN**, M.J. DREYER*, and C. LOURENS*

Abstract: Dauth J.; Keffen R.H.; Van Heerden J.; Dreyer M.J. and Lourens C. **Blood chemical and electrolyte concentrations in the mountain reedbuck *Redunca fulvorufula*.** *Journal of the South African Veterinary Association* (1987) **58** No. 3, 135-136 (En) Department of Chemical Pathology, Faculty of Medicine, Medical University of Southern Africa, 0204, Medunsa, Republic of South Africa.

Concentrations of sodium, potassium, chloride, inorganic phosphorus, total magnesium, total calcium, iron, urea, creatinine, total protein, albumin, total bilirubin, alkaline phosphatase (ALP), alanine transaminase (ALT), lactate dehydrogenase (LD), creatine kinase (CK), gamma-glutamyltransferase (GGT) and aspartate transaminase (AST) were determined in serum specimens collected from 53 free-ranging mountain reedbuck *(Redunca fulvorufula)* during live capture using nets. Considerable variations in the concentrations of the enzymes ALP, LDH, CK, GGT and AST were found as well as in the concentrations of creatinine, bilirubin and iron. This wide variation in results seriously questions the usefulness of similar blood investigations on heterogenous groups of mechanically restrained animals.

Key words: Mountain reedbuck, *Redunca fulvorufula,* serum analyses, electrolytes, biochemical constituents, serum enzyme activities

INTRODUCTION

It has become accepted practice in veterinary science to determine the concentrations of a wide variety of blood biochemical components to aid in the diagnosis of various metabolic, nutritional and organ disorders. Reference values for the various biochemical constituents are available for most domesticated animals but not for all species of wild animals. The object of this paper is to present values of some blood constituents in mountain reedbuck *Redunca fulvorufula* captured in nets.

MATERIALS AND METHODS

Venous blood specimens were collected in evacuated tubes (Vac-u-test, Radem Laboratory Equipment) from 53 mountain reedbuck of different ages and sex, on four occasions within five minutes after being captured alive in nets. The animals were chased and herded into the nets by a helicopter for periods ranging between five to ten minutes.

Blood specimens were obtained by venipuncture of the jugular vein. The animals were free-ranging within the confines of the Pilanesberg National Park, Bophuthatswana. The clotted specimens were centrifuged, the serum delivered into plastic tubes and stored in a cool bag until delivered at the laboratory two hours later where they were frozen (−20°C) and analyzed in a batch. The specimens which were stored for periods ranging from two to six months, were analyzed for sodium (Na), potassium (K), chloride (Cl), inorganic phosphorus (In PO_4), total magnesium (tMg), total calcium (tCa), iron (Fe), urea, creatinine, total protein (TP), albumin (Alb), total bilirubin, alkaline phosphatase (ALP), alanine transaminase (ALT), lactate dehydrogenase (LDH), creatine kinase (CK), gamma-glutamyltransferase (GGT) and aspartate transaminase (AST) by described methods[2].

The mean ($\bar{x}$), range standard error of the mean (SE) coefficient of variation % (CV%), variance (Var) and standard deviation (SD) were calculated for the analysed constituents.

RESULTS

Table 1 demonstrates the values of the different constituents which were statistically analysed. Considerable variations were reported in the activities of the enzymes ALP, LDH, CK, GGT and AST as well as in the concentrations of creatinine, bilirubin and iron.

DISCUSSION

The presented values for the different blood constituents give an indication of values that may be obtained from mountain reedbuck, chased and captured in nets, in the Pilanesberg National Park. Although specific parameters for stress were not monitored, these animals were obviously under stress due to the helicopter drive, capturing in the net and handling by human beings at the time of collection of blood specimens.

The concentrations of most of the constituents apart from enzyme activities, are in agreement with ranges reported for most other ruminants[3]. The tremendous range in the concentrations of LDH, ALP, CK, GGT, AST, creatinine, iron and bilirubin would limit their use as diagnostic aids in the mountain reedbuck. The wide variation in the reported figures could, however, be due to one or more of the following factors: (a) specimens were obtained only from an apparently healthy population which consisted of individuals of both sexes and of different age groups. It is a well known fact, for exam-

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Table 1: The mean ($\bar{x}$), standard error of the mean (SE), coefficient of variation % (CV%), variance (Var) and standard deviation (SD) for the different constituents evaluated in serum of mountain reedbuck.

	n	$\bar{x}$	range	SE	CV%	VAR	SD
Na mmol ℓ^{-1}	53	151,56	141,00-170,00	0,74	3,57	19,28	5,41
K mmol ℓ^{-1}	53	5,40	3,80-7,50	0,10	13,98	0,57	0,75
Cl mmol ℓ^{-1}	53	105,75	97,00-118,00	0,65	3,62	22,68	4,76
Phosphorus mmol ℓ^{-1}	52	1,92	1,03-3,20	0,07	26,72	0,26	0,51
tMg mmol ℓ^{-1}	53	1,28	0,96-1,60	0,01	10,23	0,01	0,13
tCa mmol ℓ^{-1}	53	2,78	2,30-3,19	0,02	6,68	0,03	0,18
Fe μmol ℓ^{-1}	52	33,06	16,20-55,50	1,42	31,09	105,74	10,28
ALP U ℓ^{-1}	52	169,95	25,00-506,00	8,93	34,85	3 509,57	59,24
ALT U ℓ^{-1}	52	10,03	2,00-16,00	0,32	31,56	10,3	3,16
LDH U ℓ^{-1}	52	685,94	10,00-1 303,00	1,90	19,68	18 226,59	135,00
CK U ℓ^{-1}	52	113,15	1,00-1 586,00	0,21	56,38	4 070,23	63,79
GGT U ℓ^{-1}	52	73,73	2,00-134,00	2,52	24,74	332,74	18,24
AST U ℓ^{-1}	52	78,03	2,00-247,00	6,04	55,90	1 903,01	43,62
Urea mmol ℓ^{-1}	48	5,82	1,50-11,80	0,35	43,82	6,51	2,55
Creatinine μmol ℓ^{-1}	53	178,00	108,00-248,00	4,03	16,52	864,80	29,40
TP g ℓ^{-1}	53	70,67	60,00-90,00	0,97	10,08	50,78	7,12
Alb g ℓ^{-1}	53	29,83	25,00-35,00	0,30	7,43	4,91	2,21
Bilirubin μmol ℓ^{-1}	52	45,03	23,00-71,00	1,71	27,47	153,13	12,37

ple, that the serum activity of ALP is influenced by the age of the animal; (b) the difference in periods of storage of different serum specimens; (c) different perception of stress by different animals. The stress of live capture of wild animals is invariably associated with strenuous muscular excercise which may have influenced for example, concentrations of potassium, CK, LDH and AST. Positive skewness, although not a diagnostic feature of stress, is frequently found in stress data[1]. Individual responses of animals to a given stressful situation are considered to be the basis of skew-distributed data; (d) the difference in time from start of chase to the collection of blood specimens between different animals.

The wide variation in concentrations of constituents as listed above, found in this investigation seriously questions the usefulness of similar blood chemical investigations on heterogenous groups of mechanically restrained animals. The application of Gaussian descriptive parameters in statistical analyses of data under such circumstances is, therefore, also not fully applicable. It should, however, be borne in mind that this investigation was a byproduct of the capture and handling of mountain reedbuck for purposes other than merely an analysis of constituents in blood.

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SHORT COMMUNICATION **KORT MEDEDELING**

BLOOD CHEMICAL PARAMETERS IN THE WARTHOG *PHACOCHOERUS AETHIOPICUS*

R.H. KEFFEN***, J. VAN HEERDEN*+, J. DAUTH** and M.J. DREYER**

ABSTRACT: Keffen R.H.; Van Heerden J.; Dauth J.; Dreyer M.J. **Blood chemical parameters in the warthog *Phacochoerus aethiopicus*.** *Journal of the South African Veterinary Association* (1987) **58** No. 3, 137-142 (En) P.O. Box 1201, Pilanesberg National Park, Mogwase, Bophuthatswana.

Concentrations of sodium, potassium, chloride, inorganic phosphorus, total calcium, total magnesium, albumin, total protein, cholesterol, urea, creatinine, cortisol as well as the activities of alkaline phosphatase, alanine transaminase, aspartate transaminase, gamma-glutamyltransferase, creatine kinase and lactate dehydrogenase were determined in serum specimens collected from 100 free-ranging warthogs *Phacochoerus aethiopicus* within five minutes after they were killed with a shotgun.

Average concentrations for the following chemical constituents were found: sodium (145 mmol ℓ^{-1}), potassium (8,6 mmol ℓ^{-1}), chloride (102,5 mmol l^{-1}), phosphorus (2,31 mmol l^{-1}), calcium (2,93 mmol l^{-1}), magnesium (1,23 mmol l^{-1}), albumin (26,4 g l^{-1}), serum proteins (62,2 g l^{-1}), cholesterol (1,82 mmol l^{-1}) and urea (8,74 mmol l^{-1}). The cortisol concentrations ranged from 55-340 nmol ℓ^{-1} (n = 30). Wide variations were recorded in the concentration of creatinine as well as in the activities of the various enzymes.

Key words: Warthog, *Phacochoerus aethiopicus,* serum biochemistry, serum enzymes.

INTRODUCTION

Blood chemical parameters have been reported for other domestic and captive Suidae. The object of this investigation was to determine the concentrations of blood constituents found in serum specimens of free-ranging warthog *Phacochoerus aethiopicus* obtained shortly after death.

MATERIALS AND METHODS

Venous blood specimens were collected in evacuated tubes (Vac-u-test, Radem Laboratory Equipment) from 100 free-ranging warthogs in the Pilanesberg National Park, of undetermined age and sex within five minutes after they were shot during a culling programme over a period of eight months. Specimens were collected from an ear vein. Most animals were killed almost instantaneously by headshots. Where this was not achieved, struggling preceded death.

The clotted blood specimens were centrifuged, whereafter the serum was transferred into plastic tubes and stored in a cool bag until transported to the laboratory within two hours, where they were frozen. Specimens were stored for periods ranging from 4-8 months and then analysed in a batch.

Serum specimens were analysed for sodium (Na), potassium (K), chloride (Cl), inorganic phosphorus (P), total calcium (tCa), total magnesium (tMg), albumin (Alb), total protein (TP), urea, creatinine, alkaline phosphatase (ALP), alanine transaminase (ALT), aspartate transaminase (AST), gammaglytamyltransferase (GGT), creatine kinase (CK), lactate dehydrogenase (LD) and cortisol by described methods[4,5]. Serum cholesterol concentrations were determined with a Flexigem centrifugal analyser (Electro-Nucleonics Inc., Fairfield, New Jersey, U.S.A.) utilizing the Gemii™ cholesterol CHOD-PAP reagent kit (Electro-Nucleonics).

The mean ($\bar{x}$), range, coefficient of variation % (CV%), variance (Var) and standard deviation (SD) were calculated for constituents with analysed values within a relatively narrow range.

RESULTS

Figures 1-17 demonstrate the concentrations or activities of the different constituents analysed.

Cortisol concentrations ranged between 55-340 nmol/ℓ (n = 30). The average potassium and urea concentrations were higher than the accepted normal ranges for most mammals[1,2,6]. Although haemolysis, as evidenced by the pink to reddish discolouration of serum, was observed in a number of specimens, the concentration of potassium in these specimens in general conformed to the average concentration of potassium in the unhaemolysed specimens. Wide variations were recorded in the concentrations of cortisol and creatinine as well as in the activities of the various enzymes.

Assays to determine the concentrations of cortisol, magnesium, calcium and cholesterol could only be conducted on a limited number of specimens due to inadequate sample volumes.

DISCUSSION

Serum specimens analysed in this investigation were collected from warthogs that were shot for managemental reasons. The result give some indication of biochemical values to be expected in a heterogenous group of shot warthogs.

The method of killing probably had a profound effect on the concentrations of parameters like potassium and

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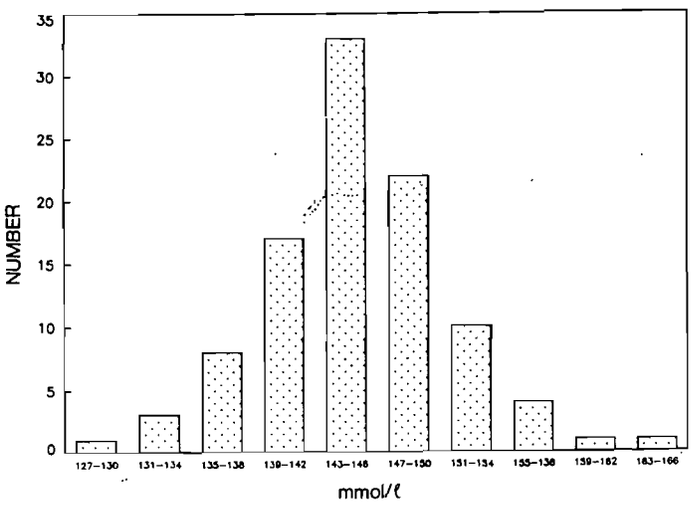

Fig. 1: The frequency distribution of serum sodium (mmolℓ^{-1})
n = 100
$\bar{x}$ = 145,04
CV% = 4,17
VAR = 36,52
SD = 6,04

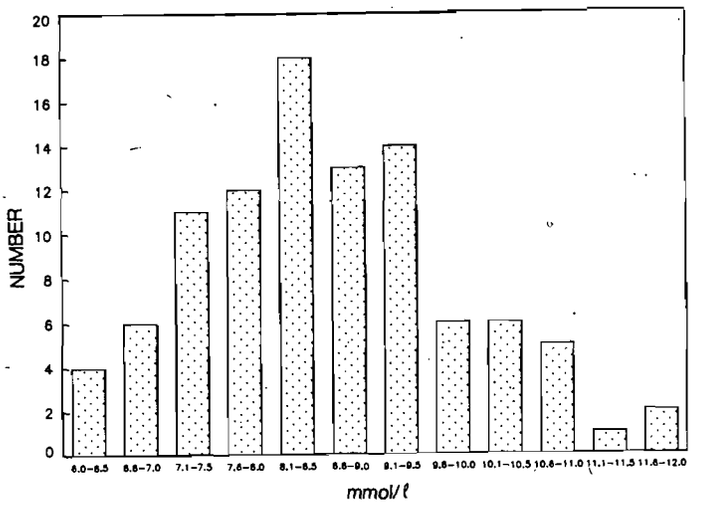

Fig. 2: The frequency distribution of serum potassium (mmolℓ^{-1})
n = 100
$\bar{x}$ = 8,60
CV% = 14,66
VAR = 1,59
SD = 1,26

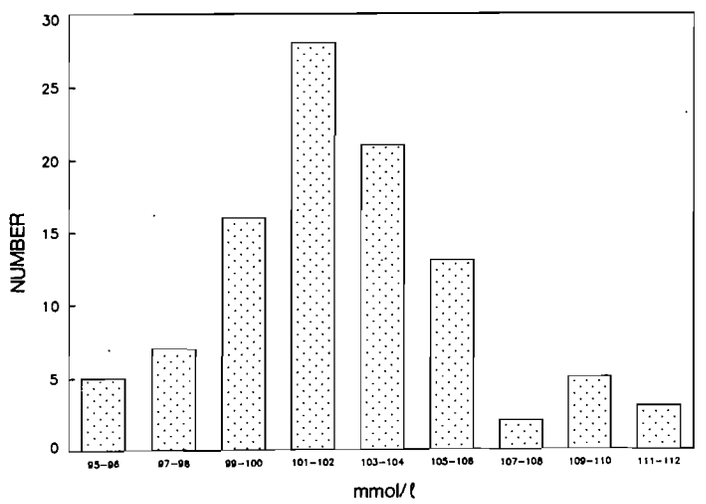

Fig. 3: The frequency distribution of serum chloride (mmolℓ^{-1})
n = 100
$\bar{x}$ = 102,45
CV% = 3,57
VAR = 13,34
SD = 3,65

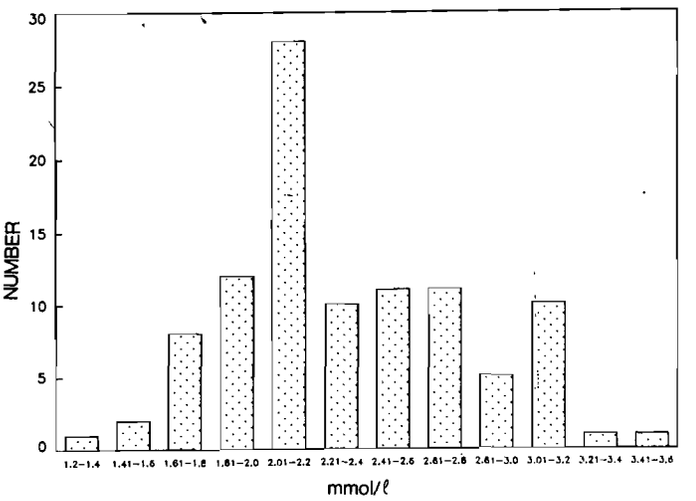

Fig. 4: The frequency distribution of serum inorganic phosphorus (mmolℓ^{-1})
n = 100
$\bar{x}$ = 2,31
CV% = 19,90
VAR = 0,21
SD = 0,45

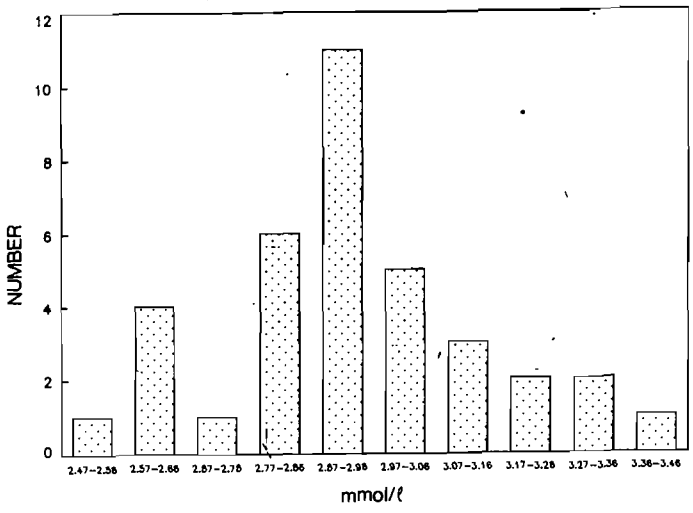

Fig. 5: The frequency distribution of total serum calcium (mmolℓ^{-1})
n = 36
$\bar{x}$ = 2,93
CV% = 7,33
VAR = 0,05
SD = 0,21

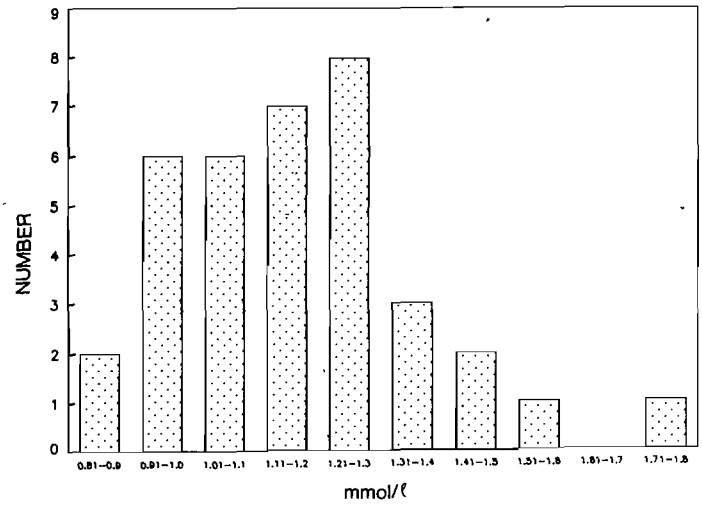

Fig. 6: The frequency distribution of total serum magnesium (mmolℓ^{-1})
n = 36
$\bar{x}$ = 1,23
CV% = 16,52
VAR = 0,04
SD = 0,19

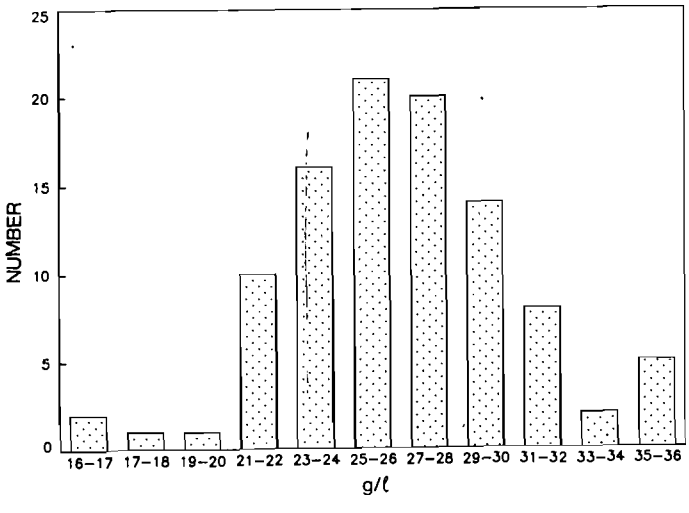

Fig. 7: The frequency distribution of serum albumin (gℓ^{-1})
n = 100
$\bar{x}$ = 26,38
CV% = 14,11
VAR = 13,58
SD = 3,72

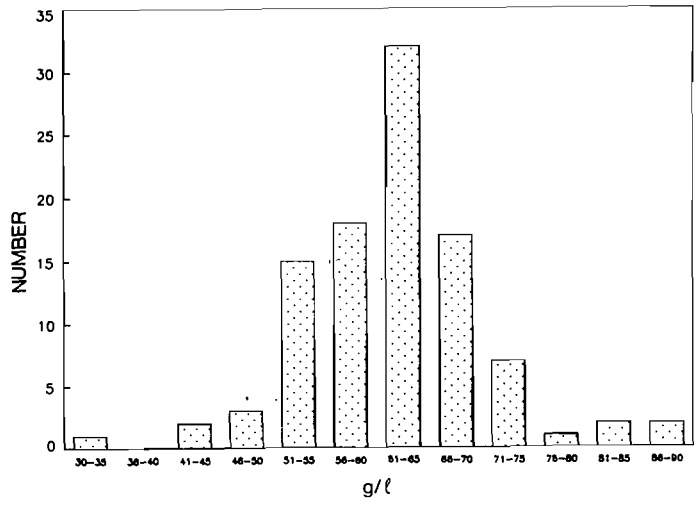

Fig. 8: The frequency distribution of total serum proteins (gℓ^{-1})
n = 100
$\bar{x}$ = 62,20
CV% = 14,02
VAR = 76,00
SD = 8,71

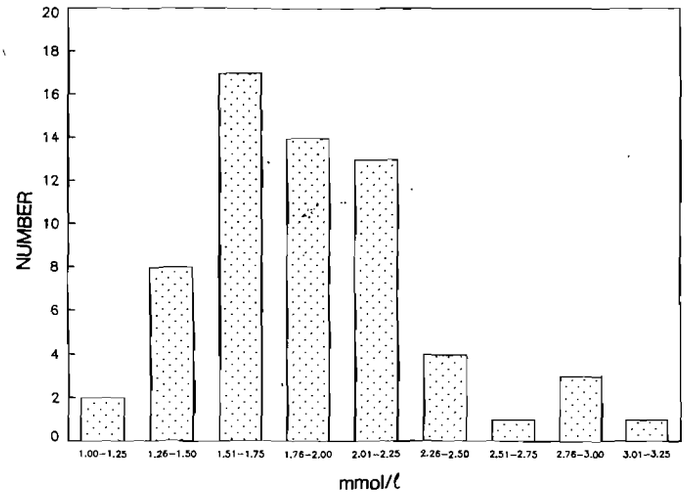

Fig. 9: The frequency distribution of serum cholesterol (mmolℓ^{-1})
n = 63
$\bar{x}$ = 1,82
CV% = 20,11
VAR = 0,13
SD = 0,37

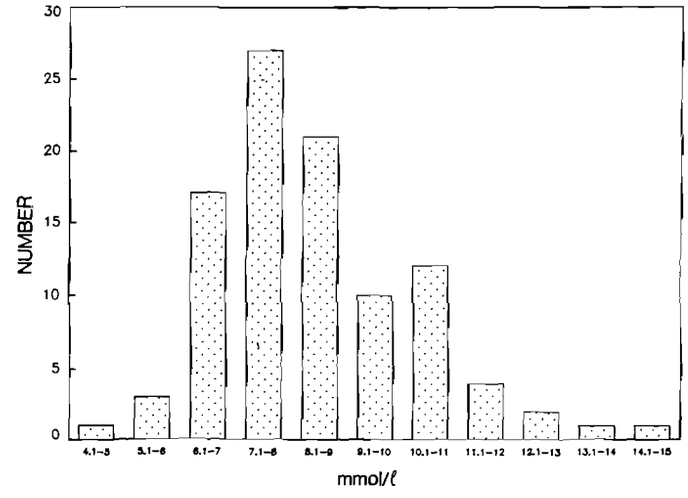

Fig. 10: The frequency distribution of serum urea (mmolℓ^{-1})
n = 100
$\bar{x}$ = 8,74
CV% = 42,05
VAR = 13,51
SD = 3,68

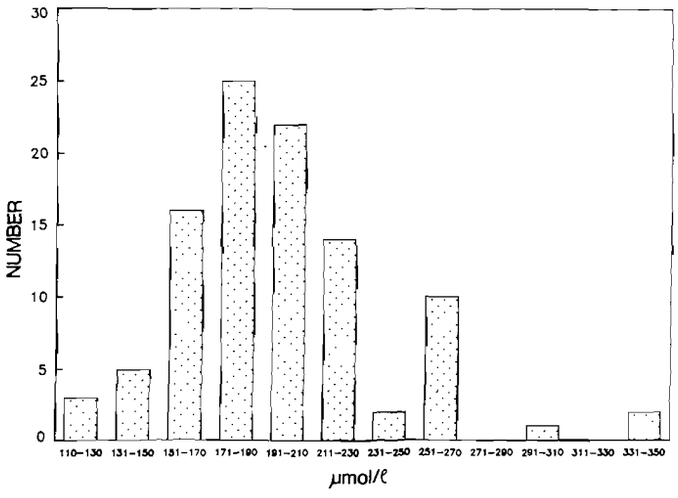

Fig. 11: The frequency distribution of serum creatinine (μmolℓ^{-1})
n = 100
range = 113-343

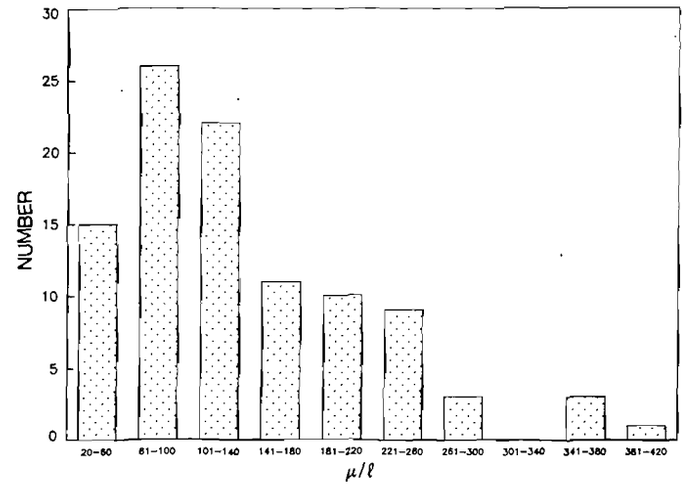

Fig. 12: The frequency distribution of serum alkaline phosphatase (u ℓ^{-1})
n = 100
range = 35-405

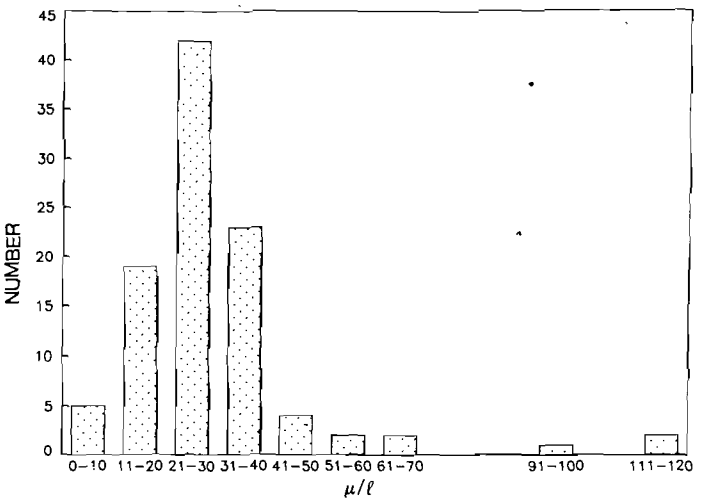

Fig. 13: The frequency distribution of serum alanine transaminase (u ℓ^{-1})
n = 100
range = 6-120

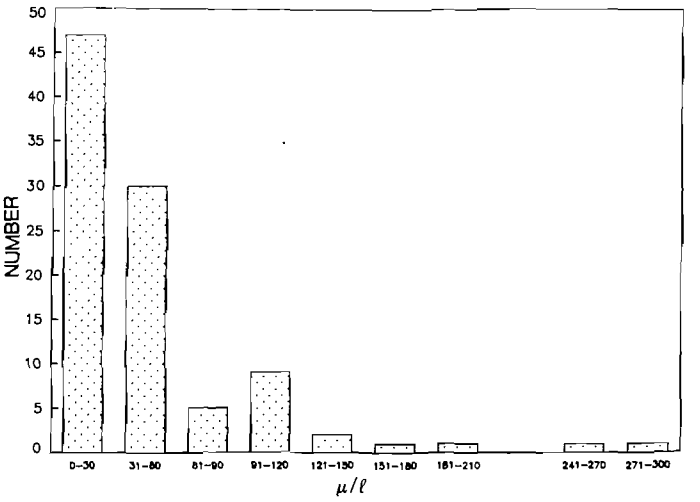

Fig. 14: The frequency distribution of serum aspartate transaminase (u ℓ^{-1})
n = 98
range = 4-267

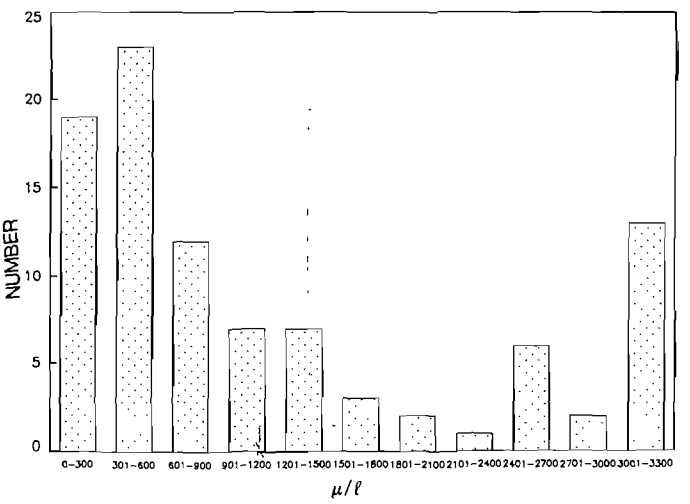

Fig. 15: The frequency distribution of serum creatine kinase (u ℓ^{-1})
n = 95
range = 15-3 300

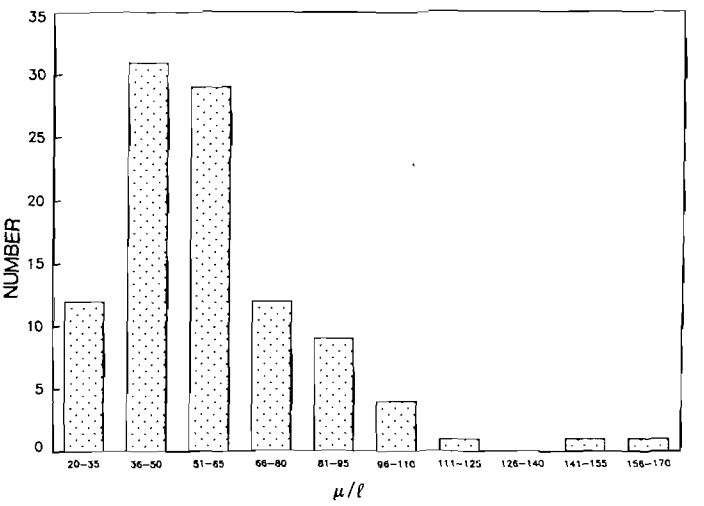

Fig. 16: The frequency distribution of serum gamma-glutamyltransferase (u ℓ^{-1})
n = 100
range = 20-163

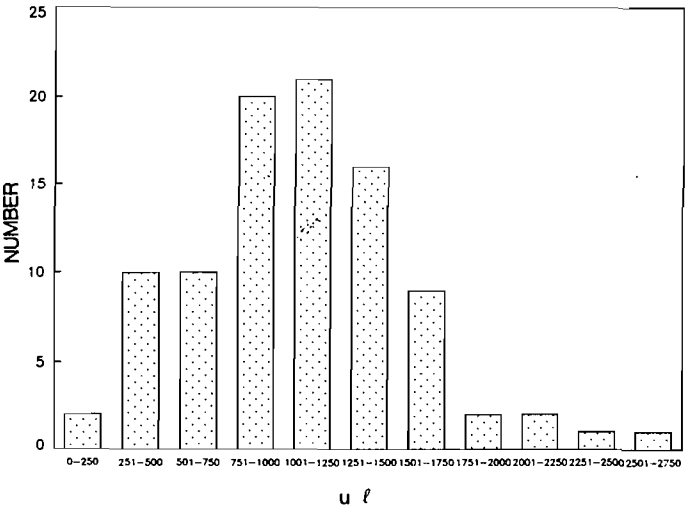

Fig. 17: The frequency distribution of serum lactate dehydrogenase (u ℓ^{-1})
n = 94
range = 189-2 630

urea. The collection of blood in shot animals is, for several reasons, not ideal. Such killing usually results in, amongst other, a greater or lesser degree of terminal hypoxia, stress and muscular contractions; all of which may, for example, influence the concentration of potassium[3]. The elevated potassium concentrations in this investigation were thus possibly partly due to terminal hypoxia in the animal as well as early post-mortem changes. Relatively high serum potassium concentrations were also recorded in serum collected from shot mountain zebra *Equus zebra zebra*[4].

The concentrations of sodium, chloride, inorganic phosphorus, calcium, magnesium, albumin, total proteins and cholesterol are, in general, in agreement with values accepted as normal for other Suidae[6].

The wide fluctuation in the concentration of creatinine as well as in the activities of the different enzymes may be due to one or more of the following factors: (a) Storage of serum specimens. It has, for example, been shown that storage of ovine serum at −20°C resulted in a decrease in the concentration of LDH[2]. It is not known what the influence of storage on enzyme activities in serum of warthogs is. (b) Specimens were collected from apparently healthy animals only. It is possible that subclinical disease may have existed in one or more animals. (c) Different levels of stress. The method of killing did not always result in immediate death. The high concentration of cortisol measured in the serum of certain animals is ample evidence of this. Agonal struggling (muscular contractions) may have contributed to very high activities of enzymes like creatine kinase and lactate dehydrogenase in certain animals. (d) Ante-mortal activities of animals. In an investigation like this on free-living animals, such ante-mortal activities are usually not known and could have differed widely amongst animals. (e) Age. In young growing animals, for example, ALP levels are considerably higher than in mature animals.

The wide variation in concentrations or activities of blood constituents as reported in this paper, questions the usefullness of blood chemical investigations on a heterogenous group of shot animals.

ACKNOWLEDGEMENTS

We are indebted to Mr C Lourens, Mrs L R Boyes, Mrs B L Mentz and Mr F F Kühn for their assistance.

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SHORT COMMUNICATION **KORT MEDEDELING**

THE USE OF KETAMINE HYDROCHLORIDE IN THE IMMOBILISATION OF THE CAPE VULTURE *GYPS COPROTHERES*

J. VAN HEERDEN*, J. KOMEN+ and E. MYER+

ABSTRACT: Van Heerden J.; Komen J.; Myer E. **The use of ketamine hydrochloride in the immobilisation of the Cape vulture *Gyps coprotheres*.** *Journal of the South African Veterinary Association* (1987) **58** No. 3, 143-144 (En) Department of Medicine, Faculty of Veterinary Science, Medical University of Southern Africa, 0204 Medunsa, Republic of South Africa.

Ketamine hydrochloride was successfully used at a dosage rate ranging from 7,5-28,8 mg kg^{-1} to immobilise Cape vultures *Gyps coprotheres* (n = 7)

Key words: Cape vulture, *Gyps coprotheres,* ketamine hydrochloride.

INTRODUCTION

Ketamine hydrochloride is used extensively as an immobilising and/or anaesthetic agent in birds. Dosages of up to 40 mg kg^{-1} of ketamine hydrochloride are recommended for the induction of anaesthesia in birds of prey[1]. No information could, however, be found in the literature on the use of ketamine hydrochloride in Cape vultures. The fact that the vulture can inflict painful and potentially serious bites inspired us to investigate the use of ketamine hydrochloride as an immobilising agent.

MATERIALS AND METHODS

Seven immature birds (less than one year of age) were injected with ketamine hydrochloride into the pectoral muscle, at dosage rates varying from 2,9-28,8 mg kg^{-1} (Table 1). The behaviour of these birds were subsequently studied and recorded intermittently for periods up to 492 minutes after injection.

Three immobilised birds suffering from lameness and who were unable to walk, were subjected to radiographic examination.

Full autopsies were conducted on two birds that died.

RESULTS AND DISCUSSION

The body mass of birds and dosage of ketamine hydrochloride are given in Table 1. Birds were immobilised within two to 13 minutes. A dosage of 2,9 mg kg^{-1} in one bird was found to be adequate in facilitating handling of the bird which remained in the upright condition but sitting on its haunches. A copious flow of mucoid fluid dribbling from the beak was observed in three birds. Nystagmus was seen in another three vultures. Only one bird showed bouts of jumping and somersaulting 45 minutes after injecting the drug.

It was very difficult to judge the time when full recovery from the effects of the drug was experienced by the birds, but with the exception of two birds they were fully conscious of their surroundings within 8 hours. One bird which received a dosage of only 2,9 mg/kg, was fully awake and biting 114 minutes after the injection of ketamine hydrochloride. Another bird could never walk again after it was immobilised, despite the fact that its general habitus and appetite remained good. Sixteen days after immobilisation, this bird was killed. The most outstanding lesion found at autopsy, was pressure on the spinal cord in the thoracolumbar area due to prolapse of intervertebral disc material.

One bird which was in a very poor condition, never regained full consciousness from the immobilising agent and died. On autopsy the bird was found to be in an extremely poor physical condition; massive ulceration of the crop was also observed.

Radiographic examination of all three birds revealed multiple fractures of leg and wing bones. Bone cortices were generally clearly identifiable.

All vultures were effectively immobilised with ketamine hydrochloride. The lower dosages were in general associated with much shorter recovery periods — an obvious advantage of an immobilising agent. The salivation noticed with the high dosage rates was not seen in Bird No 12 which received a low dosage (Table 1). This low dosage resulted in complete immobilisation and adequate sedation of the bird which remained in the upright position (sitting on its haunches). It is thus tentatively recommended that ketamine hydrochloride be used for the immobilisation of Cape vultures at dosage rates not exceeding 4 mg kg^{-1}. Chemical immobilisation of birds facilitate handling, especially when procedures like venipuncture and the administration of fluids intravenously need to be performed.

The only bird that did not recover from the effects of ketamine hydrochloride immobilisation was No 11 which had high serum urea, plasma glucose and serum creatinine levels (Van Heerden, unpublished observations) before the administration of the drug. The autopsy confirmed the poor physical ante-mortal condition of the bird.

Ulceration of the crop has also been observed in a number of caught nestlings where it was associated with sharp penetrating sticks in the crop (J Komen, unpublished observations). The presence of sticks in the crop has previously been reported by Van Heerden[2].

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Table 1: Dosages of ketamine hydrochloride and body mass of vultures

Bird No.	Body mass (kg)	Dosage of ketamine (mg kg^{-1})	Min after injection to full immobilisation	Remarks
6	7,0	28,8	2	flaccid paralysis, eye reflexes intact, copious mucus dribbling from oral cavity
7	7,1	21,1	2	nystagmus present
8	4,9	10,2	2	
9	6,6	7,5	3,5	fluid dribbling from oral cavity, somersaulting 45 min after administration of the drug nystagmus
10	7,5	9,3	2,5	
11	5,6	8,9	4	
12	6,1	2,9	13	remained sitting on its haunches; nystagmus seen

The radiographic features of adequate mineralisation and fractures are suggestive of fractures secondary to osteoporosis. Analysis of serum proteins in a small number of birds with bone fractures (Van Heerden, unpublished observations) revealed serum albumin concentrations that may be indicative of malnutrition. The possibility of malnutrition in vulture chicks resulting in osteoporosis with pathological fractures does, however, require further investigation.

ACKNOWLEDGEMENT

The authors would like to express their gratitude towards Mr E Marais and Miss A van Dyk for their assistance.

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SHORT COMMUNICATION **KORT MEDEDELING**

SERUM BIOCHEMICAL AND HAEMATOLOGICAL PARAMETERS IN THE CAPE VULTURE *GYPS COPROTHERES*

J. VAN HEERDEN*, J. DAUTH**, J. KOMEN+ and E MYER+

ABSTRACT: Van Heerden J.; Dauth J.; Komen J.; Myer E. **Serum biochemical and haematological parameters in the Cape Vulture *Gyps coprotheres*.** *Journal of the South African Veterinary Association* (1987) **58** No. 3, 145-146 (En) Department of Medicine, Faculty of Veterinary Science, Medical University of Southern Africa, 0204 Medunsa, Republic of South Africa.

Serum concentrations of total proteins, albumin, glucose, alkaline phosphatase, alanine transaminase, aspartate transaminase, gamma-glutamyltransferase, lactate dehydrogenase, creatine kinase, urea, creatinine, total calcium, ionised calcium, total magnesium, sodium chloride, potassium, phosphorus, cortisol, parathormone, 25-hydroxy-VitD3 and insulin as well as the results of haematological investigations in Cape vultures (n = 10) are presented.

Key words: Cape vulture, *Gyps coprotheres,* cortisol, parathormone, 25-hydroxy-VitD3, insulin, serum biochemistry, haematology.

INTRODUCTION

The decline in the Cape vulture *Gyps coprotheres* population in southern Africa is considered to be endangering the survival of the species[1]. While osteodystrophy has been implicated as a possible cause of disease and death of vulture chicks[4], the cause of this decline appears to be multifactorial. Clinical and radiological features of osteodystrophy have been described by Evans & Piper[3]. We could not find any clinical pathological data in the literature on the Cape vulture in order to assess a suspected metabolic bone abnormality or any other disease condition. The aim of this preliminary investigation was thus to establish some baseline biochemical and haematological parameters in Cape vultures.

MATERIALS AND METHODS

Study animals consisted of mature vultures (N = 10) (more than one year of age) in an apparently healthy physical state. These birds were held in an enclosure in the open at a research farm of the National Zoological Gardens, Pretoria at De Wildt. All birds were physically restrained and bled from a wing vein. Blood was slowly aspirated with a 10 ml syringe and subsequently transferred to evacuated tubes (Vac-u-test, Radem Laboratory Equipment) without any coagulant. For blood glucose analysis, tubes containing oxalate and sodium fluoride were used. Haematological investigations were performed on blood collected in EDTA-containing tubes. Ionised calcium determinations were done on blood collected in heparin.

Serum specimens were analysed for sodium (Na), chloride (Cl), potassium (K), total proteins (TP), albumin (Alb), glucose, urea, creatinine, total calcium (t-Ca), total ionised calcium (t-Ca), total magnesium (t-Mg), phosphorus, alkaline phosphatase (ALP), creatine kinase (CK), alanine transaminase (ALT), aspartate transaminase (AST), gamma-glutamyltransferase (GGT), and lactate dehydrogenase following standard methods[2,5]. The concentrations of the following serum constituents were determined by radioimmunoassays: cortisol (Gammacoat TM) (125I) Cortisol Radioimmunassay Kit, Clinical Assays); carboxyterminal parathyroid hormone (C-terminal PTH) which detects the 65-84 aminoacid sequence of human PTH (C-terminal PTH, Immuno Nuclear Corporation); midmolecule parathyroid hormone (PTH-MM) which contains the 44-68 aminoacid sequence of human PTH (PTH-MM, Immuno Nuclear Corporation), 25-hydroxy-Vitamin D3 (Euro-Diagnostics BV) and insulin (Phadaseph insulin RIA, Pharmacia Diagnostics). The total red and white cell counts were determined manually in a counting chamber. Haemoglobin concentrations were determined with a haemoglobinometer (Counter Electronics).

RESULTS AND DISCUSSION

The results of biochemical and hormone analyses are given in Table 1.

The total red cell count varied from 2,32-3,30 x $10^{12}\ell^{-1}$ (n = 9). The packed cell volume (n = 9) varied from 0,43-0,49 and the total white cell count ranged from 29,000-60,000 x $10^{9}\ell^{-1}$. Considerable variations were found in the concentrations of CK and LDH.

At present it is impossible to draw meaningful conclusions from the biochemical, hormonal and haematological data obtained from the small number of birds investigated in this study. It is nevertheless presented in the hope that it may stimulate a detailed investigation into the blood chemistry of the Cape Vulture, especially with regards to the possible role of malnutrition in the development of bone fractures in vulture chickens.

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The authors would like to express their gratitude towards Mr E Marais and Miss A van Dyk for their assistance.

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Table 2: Blood chemical parameters of vultures in an apparently healthy state

Serum constituent	n	Mean	Standard deviation
25-hydroxy-Vit D3 ng $m\ell^{-1}$	9	56,47	9,85
Cortisol nmol ℓ^{-1}	9	7,46	3,91
Parathormone (carboxyl-terminal) pmol ℓ^{-1}	9	94,09	18,10
Parathormone (midmolecule) pmol ℓ^{-1}	9	29,39	12,10
Na mmol ℓ^{-1}	9	157,22	1,71
K mmol ℓ^{-1}	8	1,20	0,21
C1 mol ℓ^{-1}	9	117,11	1,61
TP g ℓ^{-1}	9	37,77	2,68
Alb g ℓ^{-1}	9	19,22	2,68
Glucose mmol ℓ^{-1}	10	13,11	0,81
ALP U ℓ^{-1}	8	137,87	10,88
CK U ℓ^{-1}	8	208,37	44,91
ALT U ℓ^{-1}	8	25,62	5,99
AST U ℓ^{-1}	8	110	16,95
GGT U ℓ^{-1}	8	1,37	0,51
LDH U ℓ^{-1}	8	476,25	97,34
S-urea mmol ℓ^{-1}	8	0,77	0,26
Creatinine μmol ℓ^{-1}	9	44,66	8,60
t-Ca mmol ℓ^{-1}	10	2,57	0,07
t-Ca mmol ℓ^{-1}	11	1,24	0,05
t-Mg mmol ℓ^{-1}	10	0,82	0,04
Phosphorus mmol ℓ^{-1}	10	0,79	0,28
Insulin mU ℓ^{-1}	8	6,35	4,44

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REVIEW **OORSIG**

CANINE TRANSMISSIBLE VENEREAL TUMOUR (TVT): A REVIEW

M.I. VERMOOTEN*

ABSTRACT: Vermooten M.I. **Canine transmissible venereal tumour: A review.** *Journal of the South African Veterinary Association* (1987) **58** No. 3, 147-150 (En) Department of Medicine, Faculty of Veterinary Science, Medical University of Southern Africa, 0204 Medunsa, Republic of South Africa.

The occurrence, transmmission, clinical appearance, histological findings, chromosome studies, immunity, different methods of treatment and the prevention of canine transmissible venereal tumour are reviewed.

Key words: Canine, canine transmissible venereal tumour, TVT.

INTRODUCTION

Canine transmissible venereal tumour (TVT) is usually a benign neoplasm found predominantly on the external genitalia of both male and female dogs. It is also called canine condyloma[9 31 32], venereal granuloma[32], infectious or transmissible lymphosarcoma[9 14 21 25 31-33], infectious sarcoma[24], venereal lymphoma[22] and Sticker's tumour or granuloma[9 24 32].

The TVT was first described by M.A. Novinsky, a Russian veterinarian[9 24 31 32 35] who demonstrated that this tumour can be transplanted from one host to another by means of viable whole cells. In nature TVT is propagated by means of coitus[11 24 31 32] as well as by social licking of external genitalia[9 11 31 32]. Experimentally TVT can be transmitted very easily by transplanting viable cells subcutaneously[8 11 16 35 47]. Prier & Johnson[47] injected cell suspensions intravenously with the resultant formation of disseminated tumours in most internal organs. Other species that were experimentally "infected" are coyotes *(Canis latrans)*[15], a fennec *(Fennecus zerda)*[25], nude mice[23 33 42], hamsters[35] and a fox[34 35]. All these species showed varying degrees of tumour growth subsequent to subcutaneous injection of viable whole tumour cells. Cockrill & Beasley[15] attempted to transmit TVT to suckling normal mice, rats, hamsters, kittens and opossums, but failed to do so.

Geographically TVT is found amongst dogs world wide[9 31 32]. There seems to be a high incidence in the more temperate areas of the tropics and sub-tropical regions[32], with reports of a high incidence of naturally occurring TVT in Kenya[34 38 40 49], the Bahamas[24], Nigeria[2 29 43] and India[37 44].

Extragenital tissues can be invaded by TVT and it has been described in the subcutis[18 24 26 51 53], skin[18 24 51 53], lymph nodes (especially the inguinal lymph nodes)[24 28 36 53], spleen[24 53], liver[24 53], kidneys[40 53], eyelids[7 24 53], soft palate[10 24] and the brain[3 49 53]. It is often also found in the ovaries, Fallopian tubes and uterus[51]. In immature and immunosuppressed dogs the tumours tend to lose their benign form and metastasise[3 16 24 36 41 54]. Metastases have been described to occur more often in strays and dogs in poor health[24].

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Spontaneous regression has been documented in experimentally induced cases[8 21 22 33 37], but no accurate data are as yet available on the frequency of spontaneous regression in natural occurring cases of TVT.

TRANSMISSION

In nature TVT is transmitted by deposition of intact tumour cells on epithelial surfaces that have been damaged[12 24 47]. Genital lesions are more common due to the vigorous method of mating and the extent of the genital lesions depend on the extent of damage to the mucous membrane. Biting, scratching and licking predispose to cutaneous inoculation of the tumour cells[12].

Experimental subcutaneous implantation[8 11 16 35 47], intraperitoneal implantation[35] and intravenous injection[24] of viable cells have resulted in the formation of tumourous masses.

Although dogs of any age are susceptible to TVT growth when inoculated subcutaneously or intravenously, the typical genital lesions are seen predominantly in young but sexually mature dogs, ie. in the age group where sexual activity is the greatest[24]. Subcutaneous injection of tumour cells in newborn puppies resulted in malignant tumours that grew rapidly, ulcerated and metastisised to other organs[54].

CLINICAL FINDINGS

In females, TVT is characterised by vaginal tumour masses that cause a foetid haemorrhagic vaginal discharge[24 31 32 35]. The tumour may be solitary or consists of multiple nodules and develops into pedunculated, multi-lobulated or cauliflower-like masses[12]. Expansive growth occurs in the submucosa. The overlying epithelium becomes attenuated and ulceration of the epithelium is common[31 32]. After ulceration has occurred, irregular friable masses are present and may project into the vestibule or ulcerate out to the vulva and perineum[12 24 31 32]. The friable tumour mass is usually bathed in a mucopurulent discharge[24 32 34].

In males, TVT usually involves the glans penis[12 24 32 43 39], the bulbus glandis[23 32 39], or the penile sheath[24 32 39]. It is, however, possible to find lesions in all three locations at once[24 32 39]. The bulbus glandis is most often involved[12] and this could be ascribed to the occurrence of the so-

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called copulatory tie during which the bulbus glandis swells and is held tightly in place in the vagina by constriction of the vulvar muscles of the bitch. Mild superficial mucosal damage of the bulbus glandis at this stage of copulation, is usually seen. Paraphymosis is common if the bulbus is involved due to the extensive growth preventing the penis from returning to the sheath[39].

TVT in the male is characterised by greyish-red nodular growths that are friable and bleed when handled[32 39]. It is possible to have single or multiple masses that may be firm, sessile, pedunculated, nodular or papillary[31 32]. Ulceration of the sheath is sometimes seen[24 49]. A mucohaemorrhagic discharge from the sheath may be present[32 39].

Involvement of the inguinal lymph nodes is common, especially in males with extensive tumour growth[31 32 39]. Autotransplantation is sometimes seen, especially when the TVT is licked and skin injuries are then licked[12 24].

HISTOLOGICAL FINDINGS

The canine transmissible venereal tumour is a round cell tumour. The cells are strikingly uniform in size and appearance and may be large, round, polyhedral or slightly oval. The nuclei are large, vesicular and contain a prominent eccentrically situated nucleolus. Mitotic figures are numerous[31 32 37].

In experimentally induced cases in which spontaneous regression has occurred, the following were observed: the numbers of infiltrating leukocytes, spindle shaped cells and collagen are correlated with the growth status of the TVT[33 37]. TVT's were divided into three categories, ie. progressive, steady and regressive states[21 31]. It was found that as the TVT progressed from the progressive to the regressive state, the number of round cells decreased[25 37]. In the steady state, more cells in apparent transition were seen, ie. the round and fibroblastic cells were sharing morphologic characteristics and it is thought that there may be differentiation from round cells to fibroblastic cells in structure as well as in function[25].

Classification of the cell type present in the TVT has always been controversial[31]. On histological evidence it has been described as endothelioma, lymphosarcoma, reticulo-sarcoma, neuroblastoma, sympathogonioma, histiocytoma and mature end -cell of reticulo-endothelial origin[9 31].

CHROMOSOME STUDIES

The normal canine cell contains 78 chromosomes: 76 are acrocentric, while the X and possibly the Y chromosomes are metacentric in nature. The total number of chromosomes of the cells of TVT is 59 with 17 metacentric and 42 acrocentric[27 28 32 35 43 52 53].

The chromosome count of TVT has universally a constant; this has led to the assumption that TVT is infectious via some unknown viral agent[5 22 35 44]. Originally it was believed that whole cells were needed for transmission of TVT, but in 1960 Ajello et al.[4] transmitted the tumour by means of a supernatent fluid obtained by the centrifugation of a paper filtrate. Since then, electron microscopical examinations have been performed and characteristic icosahedral electron-dense particles were found in abundance within the cytoplasm of the TVT cells[5 22 25 33 43].

Adams, Carter & Sapp[1] reported a type C viral particle in TVT tissue culture. Anekwe[6] found in an in vitro system of L-fibroblasts that deoxyribonucleic acid (DNA) extracted from TVT was capable of changing normal cells to morphologically abnormal cells. This effect was proportional to the concentration of nucleic acid extract used. Purified RNA did not contribute to the observed activity. A viral origin of TVT is therefore quite feasible, but further studies will have to be performed.

IMMUNITY

In experimental cases, spontaneous regression is often seen. These dogs, especially if they are in good condition and good health, are refractive to further TVT transplantation[46]. Young & Jones[54] found that when dogs were randomly selected for TVT implantation, a certain percentage would not develop any tumours. They also found that newborn puppies from these dams, or from dams that developed TVT and recovered spontaneously, were not all susceptible to tumour growth (36 of 43 developed tumours). In the 36 that did develop tumours, the latent period for development was in some cases up to 6 times that of other experimental dogs and spontaneous regression occurred in 20% of these animals.

Palker & Yang[45] identified and physicochemically characterised a specific tumour-associated antigen from TVT. Cohen[17] described circulating antibodies to TVT that could be detected at about 40 days or more after transplantation.

Chandler & Yang[14] did B-, T- and null-lymphocyte counts in draining lymph nodes and the tumour itself during the three stages of tumour growth. It seemed that more T-lymphocytes were present during the regression stage than at any of the other stages.

Beschorner et al.[8] found that TVT is highly antigenic and consistently stimulated the production of both inhibitory and blocking factors. Both these factors were demonstrated to be present in the IgG 2a subclass of immunoglobulins. In cases where regression takes place, it is proposed that the inhibiting IgG as well as cellular immunity plays a role in tumour suppression. Where tumours grow progressively in animals, it has been shown that serum of these cases block cellular immunity, probably due to the blocking factor.

The role of cellular immunity against TVT is presently still under investigation[8 21].

TREATMENT

1. Surgery: Varying success has been achieved with surgical excision; regrowth is common[2 29]. Cryosurgery seems to be more successful in preventing regrowth of the TVT[48].
2. Radiotheraphy: A high success rate has been obtained with radiotherapy. Results with orthovoltage radiotherapy indicate that TVT can be cured by a single dose of 10 Gy in most dogs[50].
3. Chemotherapy:

(a) Cyclophosphamide: This is an alkylating agent that interferes with DNA synthesis of fast dividing cells. Side effects seen while using this drug are vomition, depression and bone marrow suppression. Cyclophosphamide has been used very successfully on its own at a dosage

rate of 5mg kg^{-1} body weight daily per os for 10 days[22]. Females were more resistant to treatment, presumably because oestrogens stabilise lysosomal membranes and the lysosomal enzymes are no longer capable of destroying defective or abnormal cells[22]. Another dosage regimen is cyclophosphamide at 3 mg kg^{-1} body weight and prednisolone daily for 5 days, followed by rest for 5 days. This schedule is repeated 3 times. Haematinics should be added. The TVT will disappear in about 60 days[12].

(b) Vincristine: Vincristine (mitotic inhibitor) causes the dissolution of tubulin, which is a key protein in microtubules, and is very active in fast growing TVT's. The dosages is 0,025 mg kg^{-1} body weight intravenously with a maximum of 1,0 mg weekly for 2-7 weeks[13]. At this dosage results are very good and side effects are minimal.

(c) Vinblastin: This is also a vinca alkaloid and works in a similar way to vincristine. The proposed dosage is 0,1 mg kg^{-1} body weight intravenously weekly for 4-6 weeks.

(d) Combinations:

(i) Busulfan and cyclophosphamide: Busulfan is a member of the alkylsulfonates. There are 2 regimens of treatment. The first is a high dose regimen of 3mg kg^{-1} body weight busulfan for 2 days plus 50 mg kg^{-1} body weight cyclophosphamide on Day 3 which invariably causes bone marrow suppression. The second is a low dose regimen of 1mg kg^{-1} body weight busulfan for 2 days and 20 mg kg^{-1} cyclophosphamide on Day 3. This regimen produces tolerable toxicity. Both regimens will cause regression of TVT[19].

(ii) Cyclophosphamide, vincristine, methotrexate. An additive anti-tumour effect was attempted by using an alkylating agent (cyclophosphamide — interferes with DNA synthesis), an antimetabolite (methotrexate — inhibits biosynthesis of nucleic acid) and a mitotic inhibitor (vincristine — arrests cell division) Two regimens were used:

(a) vincristine at 0,0125 mg kg^{-1} body weight intravenously weekly, cyclophosphamide at 1 mg kg^{-1} body weight per os daily and methotrexate at 0,3-0,5 mg kg^{-1} body weight intravenously weekly

(b) vincristine at 0,025 mg kg^{-1} body weight intravenously weekly, cyclophosphamide at 50 mg m^{2-1} body surface per os on even days and methotrexate at 2,5 mg m^{2-1} of body surface per os on odd days.

Signs of toxicity were transient on both regimens[11]

(iii) Cyclophosphamide and vincristine. With the use of cyclophosphamide at 0,1 mg kg^{-1} body weight per os daily and vincristine at 0,025 mg kg^{-1} body weight intravenously every 96 hours, 93% cure was achieved[30].

4. Autogenous formalised vaccine: The vaccine is produced from tumours that were surgically removed. Five millilitres of the suspension are injected subcutaneously four times at weekly intervals. In 50% of the cases there was no recurrence of the tumour[44].

5. Bacillus Calmete Guerin (BCG) immunotherapy:

(a) Intralesional treatment is effective in causing regression of injected lesions within 63 days[23].

(b) Intravenous BCG inoculation was also used to increase TVT cell death after radiotherapy[26].

The action of BCG vaccine is presumably due to the T-lymphocyte (cellular immunity) stimulatory effect.

PREVENTION

The spread of TVT amongst the dog population can be limited by reducing the number of strays and dogs that roam about freely. Because sexual contact is the prime method of transmission, sterilisation of pets as well as affected dogs will limit spread. Treatment at this stage is very expensive and therefore prevention is better than cure.

CONCLUSION

Canine transmissible venereal tumour is still the most common neoplasm of the genital system amongst dogs in South Africa. The current cost of treatment is prohibitive, especially in the lower income areas where the disease is rife. More knowledge of the disease as well as the prevention thereof could lead to the control of TVT.

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LETTER TO THE EDITOR — BRIEF AAN DIE REDAKSIE

BURSITIS CALCAREA

Die gevalverslag van sogenaamde *bursitis calcarea* deur kollegas Johannes Odendaal en Llew Evans (Tydskrif van die SAVV (1987) 58 No 1, 31-32) is wel die eerste in Suid-Afrika. Veels geluk!

Die toestand is miskien nie so 'n absolute rariteit as wat algemeen aanvaar word nie. In die Afdeling Radiologie van die Fakulteit Veeartsenykunde op Onderstepoort is na skatting seker omtrent 15 ander gevalle oor die afgelope agt jaar waargeneem. In al die gevalle was die letsels terloops waargeneem in honde wat vir roetine heupdisplasie ondersoek aangemeld was. Almal was asimptomaties en heg ek persoonlik dus nie veel kliniese waarde aan die teenwoordigheid daarvan nie. Meeste van die gevalle is waargeneem in Rifrûe, Rottweilers en Duitse Herders.

My indrukke van die letsel is dat dit eerder 'n geringe avulsie is by die punt van inplanting van 'n pees op een van die apofieses van die proksimale femur. Op goeie kwaliteit X-foto's is daar in meeste gevalle sprake van 'n geringe defek in die belyning van die apofiese met 'n sone van verdigting in die beensubstans daaromheen.

'n Soortgelyke toestand word redelik dikwels in die gastrocnemius pees net bo die calcaneus waargeneem en daar speel trauma en avulsie beslis 'n rol.

PROF C J ROOS
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BLOEDNIER IN DIE SUIPLAM

Ek het die verslag van Joubert, Vermeulen en Kellerman (Tydskrif SAVV Maart 1987, p.43) in verband met bloednier in suiplammers met belangstelling gelees. Soortgelyke gevalle is in 1978 deur Dr A.P. Schutte gerapporteer en dit het aanleiding tot 'n studie gegee wat daarop gemik was om 'n optimale immuniseringskedule te ontwikkel ten einde suiplammers doeltreffend teen bloednier te beskerm. Antitoksien bepalings op die sera van die ooie en die lammers sou kon aantoon of hulle wel volgens voorskrif geënt was al dan nie.

Daar is 'n vermoede dat bokke nie so goed soos skape op immunisering teen bloednier (en moontlik ander siektes) reageer nie. Sover ek weet is hierdie aspek nog nie deeglik bestudeer nie en bly dit 'n leemte in ons kennis.

C.M. CAMERON
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VERWYSING

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BOOK REVIEW BOEKRESENSIE

PAREYS STUDIENTEXTE 51: GRUNDZÜGE DER STRAHLENKUNDE FÜR NATURWISSENSCHAFTLER UND VETERINÄRMEDIZINER

H. EDER, J. KIEFER, J. LUGGEN-HÖLSCHER and S. RASE Verlag Paul Parey, Berlin u. Hamburg, Germany 1986 pp.167, illustrations 89, tables 22, price not stated (ISBN 3-489-57116-9)

This soft-covered book entitled "Basic radiation science for natural scientists and veterinary medicals" is one of a well known series and intended particularly for students of veterinary medicine, the various natural sciences and all who are interested in this subject material.

It is described by the publishers as an elementary review of the biological-medical application of radiation, its action and the basis of radiation protection. Commencing with basic physical considerations of radiation and its measurement, the authors proceed to deal with the various types of radiation and their sources and the generation of Röntgen ("X") rays. This is followed by an extensive exposition of physiological and cellular responses in order to elucidate an understanding of the effect of radiation on tissues, organs and the organism as a whole. The themes of the following chapters are diagnostic applications, X-ray diagnostics and nuclear medicine as well as food radiology as a particular example of radiation application.

To conclude the authors go into the matter of legal provisions to ensure effective protection against radiation and discuss a matter which is of concern to the general population i.e. the sources, effects and relevant levels of environmental radiation. Finally, the answers to many questions may be found in a compilation of the more significant statutary prescriptions and provisions and a comprehensive list of references for further reading.

The book is a mint of information on a subject of great interest and concern and it is indeed a pity that such relatively inexpensive books are not available in the English language. To those who can read German it is highly recommended.

L W VAN DEN HEEVER

SURGERY OF THE REPRODUCTIVE TRACT OF LARGE ANIMALS

JOHN E. COX

Third Edn. Liverpool University Press, Liverpool L69 3BX. 1987 pp XIV and 194, numerous line drawings. Price not stated. (ISBN 0-85323-375-6)

According to the preface of the second edition, the book grew out of brief duplicated notes for final year veterinary students and the author hopes that the book will also provide material of interest and practical value to veterinary surgeons in the field. The third edition is a revision with a few chapters rewritten. With his target readership in mind, the author has achieved his objective as evidenced by a third edition.

The reader should note that the contents are heavily loaded in favour of the equine and bovine animal. As indicated by the author, where expedient, conditions outside the strict confines of surgery are also discussed e.g. equine coital exanthema, foetotomy, parturient medical therapy and restraint and anaesthesia, the latter in appendices I to IV.

It is clear that the author did not intend presenting a volume that is too detailed or too deep in scope, but a work of ready reference in day to day practice. In this easily readable book he has succeeded and as such it is recommended.

C F B HOFMEYER

0038-2809/87/03/0153-0155

PRINCIPLES OF VETERINARY RADIOGRAPHY

S W DOUGLAS, M E HERRTAGE & H D WILLIAMSON

4th Edn. W B Saunders 1987 pp X + 366, 174 radiographs and numerous sketches and photographs. Price £29-95 (ISBN 0-7 020-1176-2)

In this new edition, the authors have succeeded admirably in their objectives of providing a theoretical and practical guide in the safe and efficient use of radiography in veterinary practice. Throughout the book greater stress has been placed on radiation protection resulting in many of the positional sketches having to be redrawn. A welcome change is the new standardised terminology for radiologists based on the revised anatomical nomenclature. The older more familiar terms have still been included to make the transition easier.

The general page layout is easy to read with a positional sketch adjacent to the radiograph of that particular region. The radiographic reproductions are of a high standard and many have been enlarged in this edition. The more complex anatomical structures have topographical explanatory diagrams. At the bottom of each page space has been allowed for the radiographer to include the exposure factors of his or her machine.

Part I consists of 134 pages and 8 chapters on theory and equipment. The authors have managed to do this in a concise and easy to follow manner.

Much valuable information is given for those wishing to start up or improve their own X-ray facilities.

Part II consists of an atlas of positioning and devotes 145 pages to the canine, 8 pages to the feline, 47 pages to the equine and 19 pages to farm and laboratory animals and exotic species. Several additional radiographic positions are illustrated in this edition. The chapter on contrast techniques has been updated and tables on the various contrast agents have been included. Unfortunately no mention is made of epidurography. The section on laboratory animals has been extended to include long term radiographic monitoring of experimental animals.

References are given after some of the chapters as well as an extensive list of suggested reading in Appendix I. Appendix II lists radiographic exposure charts for 4 different X-ray machines in order to assist the radiographer to select the correct exposure factors for their apparatus.

A regretable omission is that of ultrasonography. Although not radiography, ultrasound techniques should be familiar to the radiographer and a chapter on this would have been a welcome addition.

This book is highly recommended for the student and practitioner and will assist them in improving the quality of their radiographs, resulting in better diagnostic capabilities.

R M KIRBERGER

WILDPLAASBESTUUR

J DU P BOTHMA (Redakteur)

J.L. van Schaik, Pretoria. 606 pp talle tabelle, illustrasies en foto's (ISBN 0627 014 70 4)

Wildplaasbestuur onder redaksie van Professor J du P Bothma bevat 'n magdom van inligting oor feitlik alle aspekte van wildboerdery: vanaf die moontlike redes vir die ontwikkeling van 'n wildplaas tot oornagfasiliteite vir jagters en die maak van wildswors. Die boek wat 'n gekoordineerde poging van 'n aantal deskundiges is om bestaande kennis oor wildplaasbestuur aan die leser beskikbaar te stel is waarskynlik van kardinale belang vir die voornemende wildplaaseienaar en wildplaasbestuurder. Een kernboodskap word baie duidelik wanneer die teks bestudeer word: Wildboerdery, 'n bedryf waarin steeds meer miljoene rande belê word, behels veel meer as bloot die omheining van 'n stuk grond met wild daarop.

In die eerste gedeelte van die boek word die voornemende wildplaasbestuurder vertroud gemaak met basiese ekologiese begrippe soos energievloei, basiese doelwitte vir wildplaasbestuur en veldtipes.

Die beplanning van 'n wildplaas word voorts in besonderhede behandel: die faktore wat 'n rol speel by die aankoop van grond vir voornemende wildboerdery; die omskepping van 'n vee- tot wildplaas; 'n kontrole-lys vir die vestiging en finansiering van 'n ekonomiese wildplaas; gedetaileerde inligting oor die omheining van 'n wildplaas; watervoorsiening op 'n wildplaas (selfs die bepaling van verlangde enjinsterkte); aanleg en bou van paaie en vliegvelde; beplanning van kamp- en slaggeriewe en die bou van wildhokke en uitkyktorings.

Die gedeelte oor wild dek habitatskenmerke, voedselbehoeftes, waterafhanklikheid, reproduksie en lewenskenmerke van die verskillende diersoorte. Die basiese beginsels van die bevolkingsdinamika van wild word toegelig met besprekings oor geboortes en vrektes, ouderdomstruktuur en bevolkingsgroei. Meer besonderhede ten opsigte van kenmerke, liggaamsontwikkeling en gedrag word ook vir sekere diersoorte verskaf. Inligting oor kondisiebepaling van wild word opgevolg met kort gedeeltes oor die meer algemene siektetoestande en parasiete van wild. 'n Baie nuttige gedeelte oor die versameling van monsters is ook in die boek ingesluit. Kort gedeeltes handel oor volstruise, jagvoëls en roofdiere.

Die verskillende oesmetodes, wildtellings en verskillende metodes daarvoor, jaggewere, akkommodasie van die trofee-jagter en die verwerking van trofeë is maar enkele aspekte onder die benutting van wild wat in besonderhede hanteer word.

Die teks word voltooi met hoofstukke oor mineraaltekorte en veldbeutting in verskillende veldtipes.

Een aspek van wildboerdery waaraan daar miskien meer aandag gegee kon word, is meer inligting oor ekonomiese aspekte. (Dit is heel waarskynlik weggelaat omdat die tipe van inligting besonder gou verouder en meesal slegs 'n aanduiding kan wees van 'n praktyksituasie!). Die boek is dus van min waarde as 'n voornemende boer 'n idee wil kry van wat byvoorbeeld grond of wild hom gaan kos.

Soos verwag kan word van 'n publikasie van hierdie formaat is die inhoud waarskynlik nie altyd een honderd persent feitelik korrek nie: Die gifstof van die vermeersiektebossie (bladsy 192) word aangegee as onbekend; daar word aanbeveel dat natriumbikarbonaat intraveneus toegedien word (bladsy 196) wanneer tekens van vangspiersiekte waargeneem word terwyl die tipe van behandeling waarskynlik slegs as voorkomende maatreël en in akute gevalle van hulp mag wees; die behandeling van renosters vir diareë is, om die minste te sê verdag; die behandeling van 'n renoster met byvoorbeeld penisillin teen 'n dosis van "20 ml" per dag is 'n totaal onwetenskaplike benadering.

Wildplaasbestuur word uitgegee in 'n aantreklike volkleur hardeband uitgawe. Ek het dit egter moeilik gevind om tussen afdelings en onderwerpe te onderskei omdat die begin van nuwe hoofstukke nie pertinent aangedui word nie. Ek sou ook graag 'n ietwat meer rojale spasiëring in die aanbieding van tabelle wou sien. Die belangrikste kritiek teen die uitleg van aanbieding van die boek in die geheel is miskien die feit dat nog die register nog die inhoudsopgawe reg laat geskied aan die magdom inligting wat in die boek vervat word. Die woorde "krokodil" en "luiperd" verskyn byvoorbeeld nie in die register nie, terwyl die boek wel nuttige inligting omtrent hierdie diersoorte bevat. As die leser dalk onthou om onder "roofdiere" te kyk, verwys dit slegs na 'n deel van die inligting vervat in die boek oor die luiperd. "Reptiele" is nie gelys nie.

Wildplaas-bestuur is in vele opsigte 'n monumentale werk in Afrikaans. Dit sal ongetwyfeld enige voornemende en bestaande wildplaas-eienaar en -bestuurder baat om hierdie boek aan te skaf en te gebruik. Die boek word ook sterk aanbeveel vir veeartse met 'n belangstelling in die wildbedryf.

J VAN HEERDEN

Proceedings of the Buffalo symposium arranged by the Wildlife Group of the South African Veterinary Association at the National Zoological Gardens, Pretoria on 15 November 1985

THE STATUS AND DISTRIBUTION OF THE BUFFALO *(SYNCERUS CAFFER)* IN SOUTH AFRICA

V DE VOS: National Parks Board, Private Bag X402, 1350 Skukuza, Republic of South Africa

After briefly giving a taxonomic description, the past distribution of the African buffalo *(Syncerus caffer)* is discussed. It is concluded that in historic times the African buffalo has been one of the most widely distributed of the larger mammals, their original range comprising practically all of the northern savanna zone southward, except the more arid areas such as the Kalahari, Karoo, the Namib desert and the treeless plains of the Orange Free State and much of the Transvaal which are in the southern savanna zone.

History also shows that the African buffalo was probably one of the species of wildlife that suffered most from the presence of man and a foreign disease such as rinderpest. This resulted in most of the buffalo over southern Africa being killed and only relict populations remained in areas such as the Kruger National Park, Hluhluwe and Umfolozi Nature Reserves and the Addo Elephant National Park shortly after the turn of the century. Here the animals again thrived under good conservation practices in conservation areas such as the National Parks of South Africa. With foot-and-mouth disease and Corridor disease prohibiting the spread of buffalo from the Kruger National Park and Hluhluwe-Umfolozi Game Reserves, the Addo population represented the only known foot-and-mouth and Corridor-disease-free free-living population of buffalo in existence. From Addo excess animals were later systematically distributed throughout southern Africa to areas where they existed previously. Distribution maps of the earlier and present distribution patterns are provided. Lately the buffalo numbers have been augmented further by importations from European zoological gardens.

In general it was found that buffalo from Addo that were sold to the private sector did not increase appreciably, whilst excellent growth was recorded throughout for buffalo that were in the hands of conservation authorities. Several reasons for this phenomenon are hypothesised; ie. minimal viable numbers as a breeding unit, unfavourable habitat, a fast turnover which is brought about by excessively high prices, hunting pressure, etc.

It is concluded that although the position of the African buffalo in South Africa is considered very favourable with large numbers residing in National Parks, the demand in the private sector for breeding purposes and trophy hunting at this stage still grossly exceeds the availability; this creates a scarcity factor and grossly inflated prices.

REMOTE CHEMICAL IMMOBILIZATION OF AFRICAN BUFFALO *(SYNCERUS CAFFER)*

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A short review and evaluation of the available information on the chemical immobilization of the African buffalo *(Syncerus caffer)* is given. It is followed up by experience which the author has acquired over the past 21 years in the National Parks of South Africa. The drug compounds and equipment which have been found to be most effective are described briefly. This is followed up by a more in-depth description of the actual immobilisation procedure. Recommended dosage rates and a field guide to the use of immobilisation are also provided. Average recommended dosage rates of the primary immobilisation agents for buffalo under free-ranging conditions are given as 15 $\mu g\ kg^{-1}$ for etorphine hydrochloride, 12 $\mu g\ kg^{-1}$ for carfentanil and 120 $\mu g\ kg^{-1}$ for fentanyl. Azaparone and xylazine hydrochloride at dosage rates of 50-150 $\mu g\ kg^{-1}$ and 50-100 $\mu g\ kg^{-1}$, respectively, were found to be the best additives. It is also pointed out that buffalo, adapted to captivity, require dosages as low as half to one third of the abovementioned dosage rates. It was also found that buffalo are hardy animals and low capture myopathy risks.

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PRACTICAL ASPECTS IN THE HANDLING OF AFRICAN BUFFALO *(SYNCERUS CAFFER)*

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There are an estimated 60 000 African buffalo *(Syncerus caffer)* in Zimbabwe distributed mainly in three areas: the northwest Hwange National Park and Matetse Safari Hunting Area, the Zambezi Valley and the southeastern Lowveld, most confined to the Gonare Zhou National Park.

The Zambezi population has for a long time been isolated from contact with cattle. Most buffalo occur well within the tsetse fly areas where there are few if any cattle. Because they share the same water points and graze the same pastures as cattle, I believe the south-eastern Lowveld buffalo populations have initiated the majority of foot-and-mouth disease (FMD) outbreaks. In 1968 SAT type 1 FMD virus was isolated from buffalo in the south-eastern Lowveld. This type had not been seen in cattle for 14 years prior to this date.

The Minister of Agriculture was subsequently urged to take action against the buffalo. The public in general, however, were not fully informed of all facts of the matter and when the Minister issued the "Destruction of Buffalo Order", many complaints were lodged.

Publicising the Veterinary Department's efforts to produce foot-and-mouth disease-free buffalo, did not placate the public, and continued opposition to the Veterinary Department's policy to eradicate all the buffalo within a corridor around the National Parks was experienced. "All the buffalo" may have sounded a lot but may only have been as few as 80 animals at the time. A total of 4 000 buffalo have been eradicated for FMD control.

Over the last decade I have gained a lot of experience in capturing calves from wild herds. Wild herds are driven into plastic bomas and left undisturbed until nightfall. The team then enters the boma with Land Rovers and spotlights. Large adult bulls are darted, crated and dispatched to safari areas. Calves are then caught by hand. On one night for example, 32 calves were caught in two and a half hours. Some calves were actually pulled from between their mother's legs, and although they bellowed, there was no reaction from the cows. On this particular operation the remainder of the herd was loaded into large cattle trucks. A long race and funnel was constructed with poles and using cattle prodders and a Land Rover with a push board, approximately 40 head were loaded at night and transported elsewhere.

CALF REARING

Day-old calves can be reared without difficulty on pure cows' milk. The older the calves were when caught, the more difficult it was to get them to start feeding. Eight-week-old calves can be reared without milk, but they are very reluctant to eat meal. The presence of a tame buffalo is of tremendous help in teaching calves and getting them started on meal.

Heifers progressed noticeably better than bull calves. During the first and second winters in captivity and in spite of feeding with lucerne and silage, males lost a lot of condition.

Weaner captive buffalos have been performance tested against cattle, and were found to lack the ability to take advantage of ad lib. feeding.

Bulls, which are very tame in captivity, start becoming aggressive at about four years of age. One bull in a group tends to become dominant while others of the same age are submissive and non-aggressive.

Cows usually calve for the first time at the age of five years. Well-fed heifers have been known to calve at four years. One of our cows produced two calves in one year, one on 16 January and a second on 20 December.

PROBLEMS ENCOUNTERED WITH CALF REARING

A peracute outbreak of coccidiosis occurred and animals died before diarrhoea set in. If a large number of calves are being reared, it is advisable to be very strict on hygiene.

E. coli infections, buffalo pox and fungal pneumonia have been diagnosed.

Helminth infestations have never been a problem and can easily be controlled by the routine use of Fenbendazole.

Lice are also easily controlable by dipping regularly or by spraying.

Scrotal sucking can be controlled by confining calves individually for about an hour following bottle feeding.

CASTRATION

Castrated males do not put on as much weight as cows, even with excessive feeding.

DEHORNING

Perfect dehorning cannot be achieved because the boss continues to grow. If animals are to be handled in crushes or stables, it is desirable to dehorn. Bosses of castrated animals are less horny than those of bulls.

TRAINING TO BE LED AND TO THE YOKE

The process is no different to that used for cattle except that training should start at a younger age and close contact with the animals must be maintained constantly. Young animals are irresponsible and become more reliable and manageable as they mature. The best pair in the yoke are a cow and an ox both nine years of age.

HERD MANAGEMENT

Tame herds cannot be driven from one place to another. They are easily led however, and a second stockman is used for chasing the lazy stragglers.

The herd usually lies down in a tight group to ruminate, unlike cattle.

There seems to be a more definite, or more noticeable dominance hierarchy in buffalo than in cattle. When one or two animals have been removed from the herd for observation, and then returned, this is frequently followed by considerable social upheaval for a couple of hours.

Near Harare two groups of 5 animals each are kept. The groups are housed on farms and are kept for long-term theileriasis studies. Both groups consist of bulls and cows. On several occasions these animals have for no known reason decided to go "walkabout" and gone off in a straight line for up to 24 km before being discovered. The move is not motivated by food, water sex, season or weather. Is there such a thing as a minimum stable social unit? Or is the explanation perhaps something simple such as a biting fly?

On a few occasions individuals in the National Parks herd of 60 head have become separated from the herd. They panicked and moved off in a straight direction over long distances.

There is a tremendous variation in conformation, horn type and temperament. We have also noticed while working with wild herds, that some are easier to work with and less aggressive than others.

The domesticated buffalo cows are remarkably non-aggressive and on many occasions we have moved new-born calves and carried them to a waiting vehicle with the mothers following. We have learned that tame buffalo must never be hit on the head. Reflex aggression is never far under the surface.

DIPPING

Buffalo enjoy water and routine dipping for tick control with a spray race presents no difficulties. The aggressive and very dangerous bull is enticed into a pen with some feed and the rest of the herd is led to the spray race about 500m away. The bull is hand-sprayed in his pen. The routine was used as soon as the bull started showing aggressiveness. He now accepts this process, but initially when the herd was taken away from his control, he would get very upset.

EMBRIO-OORPLASINGS BY BUFFELS

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Moeilik hanteerbare buffelkoeie wat met dragtige-merrie-serum behandel is het follikels ontwikkel maar geen ovulasie getoon of *corpora lutea* ontwikkel nie. Geen ova of embrio's is gekollekteer nie. Hantering van die koeie vir sinchronisasie, superovulasie, kunsmatige inseminasie en spoeling het algemene verdowing met etorfienhidrochloried genoodsaak.

Buffelkoeie wat meer geredelik hanteer kon word is slegs met xilasien kalmeer. Van die koeie het superovulasie getoon met follikelstimulerende hormoon.

Sintetiese prostaglandiene is effektief bevind vir sinchronisasie van koeie. Bevrugting is verkry met kunsmatige inseminasie. Vroeë dragtigheid is bevestig in twee gevalle waarna óf resorbsie óf aborsie plaasgevind het.

Semen is baie moeilik vanaf buffelbulle verkry. Enkele bulle is dood na verdowing met etorfienhidrochloried en asaperoon en daaropvolgende elektroejakulering. Die kwaliteit van die semen wat verkry is was swak en dit het nie goed bevries nie.

Beter resultate met embrio-oorplasings sal waarskynlik verkry word indien dit nie nodig sou wees om koeie te verdoof nie.

FOOT-AND-MOUTH DISEASE AND THE AFRICAN BUFFALO: A REVIEW

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SUMMARY

Historically, clinical foot-and-mouth disease has been seen in cattle in southern Africa since 1795. More recently it has been observed that most outbreaks in cattle occurred where wild ungulate contact existed. With improved techniques for virus isolation and antibody determination, surveys in southern Africa showed that out of the many wild cloven-hooved species tested, only the African buffalo *Syncerus caffer* acted as a long-term carrier and reservoir of the SAT viruses.

Subsequent epidemiological research has shown that foot-and-mouth disease (FMD) cycles in an infected buffalo population and that buffalo calves are colostrally protected in early life but later become susceptible, infected and thereafter develop "carrier" status. Transmission of FMD from carrier buffalo to susceptible cattle has been shown to be an inefficient and rare event. Actively (usually asymptomatic) infected buffalo, however, following primary exposure to a specific SAT virus type, shed substantial quantities of virus in their saliva, nasal secretions and respiratory aerosols. During this period (lasting up to 4 weeks post-infection) they are quite capable of infecting cattle in close contact. Cyclical epidemics in impala *Aepyceros melampus* probably also arise from buffalo contact, but is experimentally difficult to duplicate. Future research aimed at the role of other cloven-hooved species as virus amplifiers and the possible role of arthropod vectors, are required to unravel the epidemiological puzzle posed by endemic FMD.

HISTORY

The first description of possible foot-and-mouth disease (FMD) in southern Africa was recorded by Le Vaillant in 1795[17]. Thereafter in 1850, Gordon Cumming[8] described what was certainly FMD along the Vaal River and in 1858 a similar condition in cattle was reported by General Kruger[14]. In 1892 a serious epizootic of FMD swept down from Mashonaland (Southern Rhodesia) through Bechuanaland and as far south as the Cape colony. FMD was thus present in the two Boer Republics, the Cape and Natal colonies, Rhodesia and Bechuanaland at the time when the rinderpest panzootic hit southern Africa in 1896. After the rinderpest devastation only isolated pockets of cattle and wildlife survived and as a result FMD disappeared from southern Africa until 1931/32 when a large outbreak occurred in cattle in south-eastern Rhodesia. This outbreak spread across into south-eastern Bechuanaland in 1932 and crossed the South African border into the Koedoesrand area of the Potgietersrus district. It was eradicated from the Transvaal in 1934.

In 1937 FMD appeared in Portuguese East Africa and spread across into the adjoining portion of the Barberton district in the eastern Transvaal. From 1938-1983, 28 outbreaks of FMD occurred in South Africa of which 25 outbreaks were in the area surrounding the Kruger National Park (KNP) and the other three outbreaks occurred in the western Transvaal and northern Cape (1957, 1958 and 1961). In Botswana (1948-1970) there have been eight outbreaks of FMD and in Zimbabwe (1931-1978) there have been 62 outbreaks. Initially it was thought that FMD was predominantly a disease of cattle which spilled over into the surrounding wild ungulate populations[1 4]. Heightened surveillance, observations and reports by field staff quickly dispelled this theory as more and more focal outbreaks occurred in the absence of cattle movement and where the presence of wild ungulates was a common factor[13 15 16].

Game shot in the area of, and at the onset of an outbreak of FMD in cattle, often showed healing or older lesions than the domestic stock. Rossiter & Albertyn[19] reported on findings in game animals bordering on the KNP in the 1944 FMD outbreak and speculate that FMD had in fact become endemic in game animals in the eastern Transvaal.

In 1948 the southern African strains of FMD were first typed and found to be different from the European strains. They were appropriately called Southern African Territories types 1, 2 and 3 — now abbreviated to SAT 1, 2 and 3.

The technological advancements which made isolation of the virus feasible, opened new avenues for research, such as the screening of sera for antibodies and typing of virus strains.

The first large serological surveys of wildlife was started in Rhodesia during the 1958-1962 Operation Noah activities on Lake Kariba. This was augmented (1962-64) by sera obtained from animals killed during tsetse control operations and other wildlife culling operations[3 14 16]. Subsequently, serological surveys were also carried out in Botswana and the Republic of South Africa. These serological surveys showed that all the southern African buffalo populations with the exception of the Zululand and Addo buffalo, had high antibody titres to one or more of the SAT type FMD viruses. A few individuals of other species, eg. kudu, impala and warthog did show antibodies to one virus type, but typically these titres were of a much lower order. Live virus was isolated from a clinical case in an impala in the Kruger National Park by Meeser[18].

In the mid 1960's it became possible, because of the development of pharyngeal scraping or probang test, to sample epithelium and secretions from the oesophagopharyngeal area of asymptomatic animals for virus isolation and typing. This technique was perfected in Botswana and Rhodesia[11] and was also later used in South Africa and east Africa.

Hedger[11] reported virus isolation from between 50 and 65% of wild buffalo sampled. All three SAT virus types were present in each buffalo population. In the Kruger National Park, Gradwell (Pers. Comm.) reported isolation of FMD virus from 70% of buffalo sampled (age 1-3 years). Oesophagopharyngeal scrapings from the Zululand and Addo buffalo sub-populations were negative. Anderson et al.[2] reported from East Africa

that virus was isolated from 13,6% of 135 buffalo samples. Pharyngeal scrapings from all other asymptomatic wild ungulates were negative for virus. Hedger et al.[12] further reported failure of development of long-term carrier status following artificial infection of impala, warthog and bushpig. Kudu, however, did carry virus for up to 3 months.

Thus, after extensive research work in southern and east Africa in the 1960's and 70's it was found that only buffalo remained long-term reservoirs of virus after FMD infection. Following infection, the virus may persist in the pharynx of buffalo for at least 5 years[10 12] and from the observation that SAT 1 and 2 could be maintained in a small isolated herd of buffalo (less than 100 animals) for at least 24 years[6] it must be concluded that carriers may play a role in maintaining FMD virus in buffalo populations.

Despite the apparent ease with which the SAT types of FMD virus are transmitted between carrier and susceptible buffalo, attempts at showing that the same is possible between carrier buffalo and susceptible cattle have generally been unsuccessful[2 5]. More recently however, transmission of SAT 3 from carrier buffalo to cattle after 2 years of close contact between the two species, have been reported[6].

Despite these contradictory findings a strong belief exists among veterinarians in southern Africa that buffalo in general are the ultimate source of SAT viruses in FMD epidemics of domestic stock.

This belief is based on the following strong circumstantial evidence:

(a) Among all African wild ungulates, only buffalo have been shown to be long-term reservoirs of FMD virus and infection rates are high in most buffalo populations.
(b) Looking at buffalo distribution maps of South Africa, Zimbabwe and Botswana, one finds that most FMD outbreaks in cattle over the past 40 years have arisen in areas of contact between proven FMD reservoir buffalo populations and domestic cattle[7 10].
(c) In areas where domestic stock are in contact with other ungulate species eg. wildebeest, blesbok, kudu, etc. but no buffalo are present, no FMD occurs even through contact is often close enough to result in transmission of other diseases such as bovine malignant catarrh (BMC), gedoelstiasis, African swine fever, etc.
(d) When buffalo sub-populations which are serologically negative and which also yielded negative results by the examination of pharyngeal scrapings (eg. Zululand and Addo populations) come into contact with domestic stock, no transmission of FMD occurs.

These facts have led to several veterinary control measures, eg.:

Elimination of buffalo from large cattle farming areas of Zimbabwe (Rhodesia Government Notice no. 251, the Animal Health (Destruction of buffalo) order — 7th April, 1978)

Barrier fences to prevent contact between cattle and buffalo eg. fencing of the southern and western boundary of the Kruger National park and the erection of the so called "buffalo fence" across the southern part of the Okavango delta in Botswana.

Cattle vaccination belts in the proximity of infected buffalo populations.

4. Permit control for movement of buffalo and buffalo products.

It must be realised that this problem has important implications for both access of southern African countries to international meat markets and for wildlife conservation and utilisation.

PRESENT RESEARCH

The present research undertaken in the Kruger National Park and the FMD Research Laboratory has been aimed at several epidemiological factors:

If the transmission of FMD from carrier buffalo to domestic stock is basically an inefficient and rare event, are there other factors operating to initiate or facilitate transmission? This first project examined the possible role of the buffalo calf during primary infection as a source of adequate virus to infect contact cattle. Buffalo calves lose their passive (colostral) immunity at between three and seven months of age. They should then theoretically be susceptible to close contact infection from other associated carrier buffalo. This project was done as follows:

Ten pregnant buffalo cows, six of which were subsequently shown to be carriers of FMD (all 3 SAT virus types present), were captured in the Kruger National Park (KNP) and allowed to calve in captivity. The buffalo cows and calves were separated by a fence from six FMD susceptible cattle, but the buffalo and cattle were obliged to use common drinking troughs and hayracks. Over a period of 15 months, during which the buffalo calves lost their maternally derived immunity, neither the buffalo calves nor the susceptible cattle became infected with FMD virus. By the end of the observation period, however, only one buffalo cow still had detectable virus in her oesophaegeal/pharyngeal specimen.

That a group of 6 buffalo cows, shown to be carriers of FMD virus, failed to transmit the infection to their calves negated to a large extent the primary objective of this investigation, which was to ascertain whether buffalo calves, in the acute stages of infection, are likely to provide a source of infection for cattle in the immediate vicinity.

A new approach was thus indicated:

2/6 FMD susceptible yearling buffaloes were infected by needle innoculation with SAT 1. The other four contact buffalo became naturally infected but failed to infect Bonsmara cattle in the same pen. The experiment was repeated using SAT 2 and young Drakensberger cattle which associated closely with the buffalo. FMD spread rapidly to these cattle during the acute phase of infection. Three more susceptible cattle which were introduced into the buffalo pen 47 days after primary buffalo infection, failed to become infected, even following dexamethasone injection of the carrier buffalo (20 mg/day for 5 days) which has done to artificially simulate a stress situation.

Since cyclical endemics of FMD do occur in impala in the KNP[20], it was deciced to repeat this experiment using impala in place of cattle. Contact took place through a fence into which communal hayracks and water troughs were built. The buffalo used originated from the Hluhluwe/Umfolozi complex in Zululand. Three concomitant studies were run parallel in this project, namely:

1. To obtain final proof that the Zululand buffalo, which had tested serologically negative[9], were indeed susceptible to FMD
2. To see whether acutely infected buffalo could transmit FMD to impala in an adjoining pen.
3. To quantitate virus excretion rates and routes in buffalo during acute infection.

Again 2/6 buffalo were needle infected and FMD spread rapidly to the 4 contact buffalo. Monitoring and regular sampling revealed that:

(a) FMD was at no stage transmitted to the impala.
(b) Buffalo, as with cattle, shed virus in saliva, nasal secretions, prepu tial secretions and vaginal secretions
(c) These Zululand buffalo were totally susceptible to SAT 2 FMD virus

This experiment was again repeated several months later using SAT 1 virus. Once again no transmission occurred to impala. The Zululand buffalo were found to be totally susceptible to SAT 1 and virus was again shed in saliva, nasal secretions and preputial/vaginal secretions.

Other findings were that regularity of virus detection as well as quantity of virus in buffalo specimens, was generally lower than for cattle infected with viruses of the same type. Conversely, virus was detected in nasal secretions and saliva of three buffalo up to four weeks after infection, a situation which has not been encountered in cattle. It was also found that most buffalo develop small interdigital hoof lesions, but no mouth lesions with these virus strains.

FUTURE RESEARCH

The following are important aspects of FMD epidemiology requiring future study:

(a) Since impala are involved in cyclic epidemics of FMD in the KNP and a serological survey of this specie is being presently undertaken, it is important to infect a herd of impala in captivity and monitor the following aspects:
  (i) Percentage of impala showing clinical signs of infection
  (ii) Virus excretion rates and routes in impala
  (iii) The magnitude, time course and duration of the antibody response in impala
(b) Fingerprinting or T. mapping of virus strains obtained from cattle, buffalo and impala to determine association of infection.
(c) Mathematical models to study epidemiological variables and possibilities.
(d) The possible role of other species eg. kudu in FMD epidemiology.
(e) Looking at possible vectors of FMD eg. biting flies, ticks and oxpeckers.

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WETLIKE ASPEKTE BETROKKE BY DIE VERVOER EN VERSPREIDING VAN BUFFELS

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Veeartsenydiens, wat 'n Direktoraat onder die Hoofdirekteur, Regulatoriese Diens, in die Departement Landbou-ekonomie en -bemarking is, word deur die Wet op Dieresiektes en -parasiete, Wet 13 van 1956 en die Vaste Regulasies (G K no R 1531 van 4-10-63) daarkragtens uitgevaardig, gemagtig om die vervoer van buffels te beheer.

Voordat daartoe oorgegaan kan word om die wetlike aspekte betrokke by die vervoer van buffels te bespreek is dit wenslik om die woordomskrywingbepalings van woorde en begrippe wat gebruik word in hierdie bespreking te definieer en aan te dui waar hulle verband gevind kan word in die Wet en Vaste Regulasies of in Regulasies wat later uitgevaardig of gewysig is.

WOORDOMSKRYWING EN WOORDBEPALINGS

Besmetlike ding:
Artikel I (II): 'n ander ding as 'n dier, vervoermiddel of parasiet, wat die Minister by kennisgewing in die Staatskoerant verklaar waarskynlik 'n siekte of parasiet die Unie sal inbring of versprei. G.K. no R. 1924 van 25 Oktober 1974. Deel 1(a) (II): Vir die doeleindes van Deel V van hierdie regulasie (Bek-en-klouseer Beperkings): die hare, borselhare, wol, horings, hoewe, bene, saad, bloed, mis, huide, velle, rou melk en rou melkprodukte van diere, die ongekookte vleis, organe en ingewande van spleethoewige diere...

Dier: (G K no R 1931 van 4 Oktober 1963): alle soogdiere, uitgesonderd die mens en die wat tot die orde Cetacea en Sirenia behoort; alle voëls insluitende pluimvee; alle amfibië; alle reptiele

Permit: (Artikel 1 XIII): 'n Skriftelike permit wat kragtens hierdie Wet uitgereik is.

Siekte (G K R 428 van 26 Maart 1965): Siekte beteken enige siekte, besmetting of ander ongesonde of ongunstige toestand waarvoor diere vatbaar is.

Wilde diere: (GK no R 1924 van 25 Oktober 1974 Deel 1 1(g): Alle diere wat nie honde, katte, beeste, perde, muile, donkies, skape, bokke, varke, amfibieë, reptiele en voëls is nie, maar sluit nie ook diere, ten opsigte waarvan die eienaar die houer is van 'n lisensie uitgereik kragtens die Beskerming van Gedresseerde Diere Wet, 1935 (Wet 24 van 1935) in nie.

SAKE WAT VERBAND HOU MET DIE UITREIKING VAN 'N PERMIT

Aangesien permitte so 'n belangrike rol speel in die beheer oor bewegings en vervoer van diere (dit wil sê ook buffels), besmetlike dinge en produkte is dit wenslik om op die volgende aangaande permitte te let:

Uitreiking en toon van permitte: (Artikel 21 (1)): Behoudens die bepalings van sub-artikel (2) van hierdie artikel en artikel 22 kan 'n persoon wat ingevolge die regulasies gemagtig is om permitte uit te reik, na goeddunke 'n permit uitreik of weier om 'n permit uit te reik, en kan die direkteur na goeddunke 'n permit kanselleer of 'n beampte gelas om dit te doen.

'n Permit mag nie uitgereik word nie ten opsigte van 'n dier of ding ten opsigte waarvan iemand by die Staat vir enige bedrag in die skuld staan alvorens sodanige bedrag betaal is.

Iemand wat toesig oor 'n dier of 'n ding het wat binne die Unie verwyder word op gesag van 'n permit kragtens hierdie Wet uitgereik, moet daardie permit op aanvraag deur 'n beampte of 'n polisiebeampte vir inspeksie toon.

Appèlle (Artikel 22): Wanneer iemand wat ingevolge die regulasies gemagtig is om permitte uit te reik, weier om 'n permit uit te reik, of wanneer die direkteur 'n permit kanselleer of laat kanselleer is daar 'n appèl teen sodanige weiering of kansellering na die Minister, wie se beslissing finaal is.

Misdrywe en strawwe vir oortredings met betrekking tot permitte (Artikel 28. Misdrywe en strawwe): Iemand wat 'n Permit deur middel van valse voorwendsels verkry; 'n permit, in enige opsig verander of vervals; met die opset om die bepalings van hierdie Wet te ontduik 'n veranderde of vervalste permit of sertifikaat gebruik; nadat hy 'n permit verkry het, 'n handeling strydig met 'n voorwaarde daarin opgelê, verrig, is aan 'n misdryf skuldig en by skuldigbevinding strafbaar —
1ste skuldigbevinding R1 000 of 6 maande
2de skuldigbevinding R2 000 of 12 maande
3de skuldigbevinding R5 000 of 24 maande ens.

INVOER VAN BUFFELS

Die belangstelling om buffels veral uit dieretuine na Suid-Afrika in te voer neem toe en daarom moet ook na die aspek gekyk word soos vervat in G K no R 1924 van 25 Oktober 1974 by: (Deel II Invoer van diere in die Republiek): Aansoek om 'n permit vir die invoer van 'n dier moet, in die vorm in Aanhangsel A vervat, gedoen word by die direkteur of by 'n persoon deur hom benoem.

Benewens die besonderhede in genoemde aansoekvorm verstrek, moet 'n applikant vir 'n permit soos in subregulasie (1) genoem, indien die Direkteur of die benoemde persoon dit van hom vereis, die verdere besonderhede en/of inligting wat gevra word betreffende sodanige dier en die voorgenome invoer daarvan, op die verlangde wyse verstrek.

Die Direkteur of die persoon deur hom benoem, kan so 'n permit skriftelik uitreik op die voorwaardes wat hy nodig mag ag en wat in die permit of in 'n aanhangsel daarby gemeld moet word, ten einde die inbring of verspreiding van siektes of parasiete te voorkom.

In die geval van buffelinvoere word onder andere vereis dat die gebied vir minstens 12 maande vry van bek-en-klouseer moet wees en dat die buffels vir vier

geslagte of meer in die betrokke dieretuin aangehou moes gewees het.

R 1531 AANHANGSEL A: AANSOEK OM PERMIT OM DIERE IN DIE REPUBLIEK IN TE VOER

1. Volle naam en adres van applikant in hoofletters

.....

2. Die getal, spesie en klas diere waarvan die invoer aangevra word

.....

3. Die land, en die deel van die land, vanwaar dit die voorneme is om hulle in te voer, en die hawe, lughawe of plek waar hulle op die vervoermiddel gelaai sal word wat hulle na die Republiek sal vervoer

.....

4. Die aard van die vervoermiddel waarmee dit die voorneme is om hulle na die Republiek te vervoer

.....

5. Die hawe, lughawe of plek in die Republiek waardeur dit die voorneme is om hulle na die Republiek te vervoer

.....

6. Die doel waarvoor dit die voorneme is om hulle in die Republiek in te bring

.....

7. Hul onmiddellike bestemming in die Republiek na vertrek van die hawe, lughawe of ander binnekomplek in die Republiek

.....

Datum.....

.....
Handtekening van applikant

Invoer van Besmetlike Dinge in die Republiek van Suid-Afrika: 'n Permit (soos hierbo) word eweneens vereis. Dit sal geld wanneer aasoeke vir semen- of embrio-invoere, of enige produkte van buffels ontvang word.

VERPLIGTE AANMELDBARE SIEKTES WAARVOOR BUFFELS OOK VATBAAR IS EN DIE TOEPASLIKE REGULASIES

Alhoewel die beheermaatreëls meer betrekking het op plaasdiere is sommige daarvan ook van belang wat die beleid oor die beweging van buffels betref. (G K no R 1471 Aanhangsel D 81/7/17): Siektes waarop die bepalings van die volgende regulasies van Deel VI van toepassing is en die diere wat vir sodanige siektes vatbaar is. Deel VI

1. Verpligte aanmelding van besmetting of vermoede van besmetting.
2. Afsondering.
3. (2) Ontsmetting
3. (3) Toegang van vatbare diere tot besmetting verhinder
14. Beskikking oor besmette karkas — voorskrifte.
15. Opgrawe van besmette karkasse — permit nodig
16.(2)(d) Inperking van vatbare diere op 'n omskrewe deel van die grond
28. (1)Register hou van vatbare diere.

Buffels is betrokke by die volgende siektes in Aanhangsel D vermeld waarop bogemelde regulasies van toepassing is:

Siekte	*Vatbare diere*
Bek-en-klouseer	Beeste, skape, bokke, varke, ander diere met gesplete hoewe en olifante
Korridor- of buffel siekte	Beeste en buffels
Miltsiekte	Beeste, skape, bokke, varke, perde, muile, donkies, wild, volstruise
Ooskuskoors	Beeste, buffels
Runderpes	Beeste, ander diere met gesplete hoewe

Siektes waarop die bepalings van die volgende regulasies van Deel VI van toepassing is (R1471 Aanhangsel E 81/7/17):

2.(1) Eienaar moet toegang tot besmette diere belet uitgesonderd 'n beampte of iemand wat 'n diagnose moet gaan maak, behandeling toepas of die dier versorg.
11. 'n Besmetlike ding mag nie, sonder 'n permit, van die besmette eiendom verwyder word nie.
13. Niemand behalwe 'n beampte of 'n veearts in diens van 'n plaaslike owerheid mag 'n besmette karkas oopsny nie.
16(1) Toegang tot 'n besmette plek moet verhinder word tot na skoonmaak en ontsmetting, soos die Staatsveearts bepaal, afgehandel is.

Buffels is by die volgende siektes en regulasies hierbo in Aanhangsel E vermeld, betrokke:
Bek-en-klouseer
Korridor- of buffelsiekte
Miltsiekte
Ooskuskoors
Runderpes

Siektes waarop die bepalings van die volgende regulasies van Deel VI
10. Niemand mag diere na of deur die grond waarop diere wat met onvermelde siektes besmet of vermoedelik besmet is, beweeg, sonder 'n permit nie.
12. Niemand mag 'n dier op die grond waarop daar besmette diere is, slag sonder 'n permit nie.

Siektes in Aanhangsel F genoem wat betrekking het op buffels en die regulasies hierbo vermeld is die volgende:
Bek-en-klouseer
Korridor- of buffelsiekte
Miltsiekte
Ooskuskoors
Runderpes

BEHEER OOR SEKERE AANMELDBARE SIEKTES WAARVOOR BUFFELS VATBAAR IS

Behalwe die algemene regulasies van Deel VI van die Vaste Regulasies wat by elk van die drie Aanhangsels hierbo bespreek is, bestaan daar nog spesifieke Goewermentskennisgewings wat betrekking het op sommige van die siektes soos hierna aangedui: (G K R 1924 van 25 Oktober 1974, Deel V 7):

Bek-en-klouseer — beperkings in sekere gebiede (Sien Aanhangsel A):

Niemand mag 'n gespleethoewige dier of besmetlike ding in die gebied in Bylae 3 omskryf (Nasionale Krugerwildtuin, landdrosdistrikte Barberton, Gordonia,

Ingwavuma, Kuruman, Letaba, Mafeking 1, Mafeking 2, Marico, Messina, Pilgrim's Rest, Potgietersrus, Soutpansberg, Thabazimbi, Vryburg 1, Waterberg en Witrivier) inbring, laat inbring of toelaat dat dit ingebring word nie of 'n spleethoewige dier of besmetlike ding van een grondeiendom binne die omskrewe gebied na 'n ander hetsy binne of buite die gebied beweeg of laat beweeg nie, behalwe kragtens 'n skriftelike permit uitgereik deur 'n staatsveearts en onderworpe aan die voorwaardes in so 'n permit gestel.

Ooskuskoors en Buffelsiekte
Beperkings en Beperkte Gebiede (Aanhangsel B):
1 (1) In hierdie regulasie beteken "beeste" ook makgemaakte buffels
2. Niemand mag, behalwe kragtens 'n permit uitgereik deur 'n staatsveearts enige bees in of vanuit die beperkte gebiede (hele provinsie Natal, landdrosdistrikte Mt Currie, Matatiele, Barberton, Letaba, Nelspuit, Piet Retief, Pilgrim's Rest, Sibasa, Witrivier en (sekere dele van Carolina en Ermelo wat aan Swaziland grens)) beweeg of die beweging daarvan toelaat nie.

WILDE DIERE

(G K no R 1924: 25 Oktober 1974; Deel XI 18):
Wilde diere — Beperkings op Bewegings: Niemand mag enige wilde dier van enige grondeiendom binne die Republiek (insluitende die Gebied) na enige ander grondeiendom in die Republiek (insluitende die Gebied) beweeg, laat beweeg of toelaat dat dit beweeg word nie, behalwe kragtens 'n skriftelike permit deur 'n staatsveearts uitgereik en onderworpe aan die voorwaardes in so 'n permit gestel.

PERMITTE

Is 'n permit nodig vir die beweging van buffels?
Wat die siekte betref, word permitte vereis in die Ooskuskoors- en bek-en-klouseerbeheergebiede soos bespreek onder die betrokke siektes, en het dit net betrekking op sekere dele van Suid-Afrika. Onder die beperkings op die bewegings van wilde diere word die hele land en Suidwes-Afrika ingesluit. Laasgenoemde het betrekking op lewende diere alleen maar nie produkte van wilde diere nie. In die bek-en-klouseerbeheergebied is daar 'n permit nodig vir die vervoer van rou produkte (besmetlike ding) van spleethoewige diere, dit wil sê ook van buffels.

Watter oorwegings geld by die uitreiking van 'n permit?
Daar moet eerstens, wat buffels betref, wetlike magtiging wees soos vermeld by Ooskuskoors, bek-en-klouseer en Wilde diere. Tweedens word daar ook voorwaardes gestel op die permit waaraan daar uitvoering gegee moet word.

Die toestaan van 'n permit en die voorwaardes wat daarop aangebring word is onderhewig aan die beleid wat die beheerinstansie neerlê en nodig vind met inagneming van beskikbare inligting

Beleid aangaande Buffelbewegings
Bek-en-klouseer
Buffels in bogenoemde beheergebied word as draers van die SAT I, II en III virus beskou. Bewegings word gevolglik nie toegelaat van 'n plek binne na een buite die beheergebied nie. Insgelyks word dit ook verhoed dat permitte uitgereik word vanaf die Nasionale Krugerwildtuin na die eerste beheergebied (rooilyngebied) wat grens aan die park of vanaf die rooilyngebied na die tweede gebied of vanaf laasgenoemde na die derde gebied (ook genoem res van die beheergebied).

Permitte sal wel oorweeg word vanaf buite na binne, dit wil sê vanaf plase in die gebied met 'n laer risiko na plase met 'n hoër risiko, byvoorbeeld vanaf die derde- na die tweede gebied ens. Dieselfde geld vir 'n beweging vanaf een plaas na 'n ander binne dieselfde risikogebied.

Korridor- en Buffelsiekte
Buffels wat in die Ooskuskoorsbeheergebied voorkom word as moontlike draers van Buffelsiekte *(Theileria parva lawrencei)* beskou. Dit is veral die gebied waar die bruinoorbosluis, *Rhipicephalus appendiculatus*, wat die oordraer van *Theileria parva* is, voorkom. (Aanhangsel C).

Die bruinoorbosluis word in Suid-Afrika aangetref vanaf die weste in dele van die Mafikeng-distrik en in Transvaal in 'n gebied noord van 'n lyn deur Krugersdorp, Pretoria en Middelburg, sowel as die oostelike dele van Carolina, Ermelo en Piet Retief, Swaziland en die dele van Natal en Oos-Kaap onderkant ongeveer 1 500 meter bo seevlak en na die binneland langs die riviervalleie na die Drakensberge toe in die kusgebied suid van die bergreeks tot by Kaapstad.

Die rooipootbosluis *(Rhipicephalus evertsi)* (Aanhangsel D) wat ook *Theileria parva* kan oordra, kom in dieselfde en 'n groter aanliggende gebied voor as die bruinoorbosluis en moet hierdie gebied ook ingedagte gehou word as 'n moontlike gevaargebied vir siekteoordraging na beeste as draerbuffels daarin sou kom.

Permituitreikings
Die uitreiking van vervoerpermitte ingevolge Wet 13/1956 is vir buffels onderhewig gemaak uitsluitlik aan die goedkeuring van die Direkteur van Veeartsenydiens.

Uit gebiede waar buffels moontlik draers van bek-en-klouseer en Ooskuskoors kan wees word uitreiking van permitte nie goedgekeur nie.

Vanaf Addo, waar die buffels nie draers van bogenoemde twee siektes is nie, word toestemming wel verleen.

Daar word egter 'n beperking geplaas op die bestemming van hierdie "skoon" buffels, naamlik net gebiede wat nie bek-en-klouseer- of Ooskuskoorsbeheergebiede is nie.

Die rede hiervoor is om die buffels teen moontlike besmetting met een of albei die siektes te beskerm sodat daar weer, sonder belemmering, bewegings van hierdie plase af toegelaat kan word.

Daar word ook landwyd 'n register opgestel van eiendomme waarop daar buffels is, met 'n aanduiding van die geslag en benaderde ouderdom van diere op die plaas. Laasgenoemde getalkontrole is veral van belang vir plase waar "skoon" buffels voorkom en permitaansoeke in die toekoms oorweeg moet word. Waar daar 'n vermoede ontstaan dat "skoon" buffels deur onwettige bewegings of andersins aan besmetting met een van die twee siektes blootgestel is, sal permitte vir vervoer weerhou moet word.

VERSPREIDING VAN BUFFELS: OORWEGINGS VIR DIE TOEKOMS

Nuwe aansoeke vir permitte

Belangstelling gestimuleer deur hoë pryse, neem toe vir die aanhou van buffels in veral die gebiede wat buite die bek-en-klouseer- en Ooskuskoorsbeheergebiede geleë is.

Die daarstelling van 'n groot aantal plase met buffels op, hou die volgende moontlikhede in:

'n Aaneenskakeling van eiendomme soos 'n ketting tot in die beheergebiede met verspreiding van besmetting langs die ketting op, veral weens die byna onbeheerbare beweging van dwaalbuffels.

Die groter risiko van besmetting van buffels in die "skoon" gebiede weens onwettige bewegings of dwaalbuffels, waarvan daar alreeds voorbeelde van laasgenoemde bekend is.

Daar sal dus besin moet word oor die wenslikheid al dan nie om onbeperk NUWE fokusse van buffels toe te laat.

Embrio-oorplasing

Dit word as een moontlikheid gesien om aan die behoefte van "skoon" buffels te voorsien, deur oorplasings op beeste te doen. Dit is egter duidelik dat die tegniek eers bemeester, en bewyse voorgelê moet word, dat byvoorbeeld bek-en-klouseer en buffelsiekte nie deur hierdie tegniek oordraagbaar is nie alvorens 'n beginselbesluit geneem kan word of die vervoer van sulke materiaal uit die beheergebiede na die res van die land veilig sal wees

Skakeling tussen Natuurbewaring en Veeartsenydiens

Die Direktoraat Veeartsenydiens en die Afdelings Natuurbewaring van die Provinsies het belang by die uitreiking van permitte vir die vervoer van wild. Om verwarring by die publiek te voorkom en hulle bewus te maak van die verpligting word op die permit van Veeartsenydiens aangedui dat 'n aparte permit van die Afdeling Natuurbewaring ook vereis word en doen laasgenoemde die omgekeerde wanneer hulle permitte uitreik.

Ordonnansie

Wat die vervoer en jag van buffels betref is dit dus ook nodig om te kyk oor watter wetlike mag Natuurbewaring in die verband beskik om die taak uit te voer. Dit vind ons vir Transvaal in die Buitengewone Offisiële Koerant Vol 228, nommer 4298 van 14 Desember 1983, nommer 519 (administrateurs), waarin " 'n Ordonnansie tot samevatting en wysiging van die wetsbepalings met betrekking tot natuurbewaring en om vir bykomende aangeleenthede voorsiening te maak", verskyn.

Onder *woordomskrywing* — Artikel 1 beteken:

"wild" enige beskermde wild, gewone wild of beskermde wilde diere, het sy lewend of dood, in artikel 15(1) beoog.

"wilde dier" enige werweldier, met inbegrip van 'n voël en 'n reptiel maar uitgenome 'n vis, behorende tot 'n soort wat nie 'n erkende huisdiersoort is nie ... en waarvan die natuurlike tuiste die Republiek is ... In Bylae 2 word die spesies genoem wat "Beskermde Wild" is, in Bylae 3 — "Gewone Wild" en in Bylae 4 — "Beskermde Wilde Diere". Dit is onder laasgenoemde Bylae waar die Afrikaanse buffel *(Syncerus caffer)* verskyn ingevolge die magte verleen by artikel 15(1) in Hoofstuk III.

Vir die beheer van dieresiektes, veral waar uitbrekings van bek-en-klouseer voorkom, is die jag van en vervoer van buffelkarkasse 'n aangeleentheid wat probleme kan veroorsaak en daarom is die volgende bepalings van belang.

Jag van beskermde wilde diere (dws ook buffels)

Behoudens die bepalings van hierdie Ordonnansie mag niemand 'n beskermde wilde dier jag nie:

Met dien verstande dat —

(a) op skriftelike aansoek van die eienaar van grond 'n permit uitgereik kan word —

1. aan die eienaar
2. aan iemand anders wat die eienaar in die aansoek aanwys,

(b) die eienaar van grond of 'n familielid aan wie hy skriftelike toestemming verleen het...

1. 'n buffel kan jag indien daar beeste op daardie grond aangehou word;
2. Wanneer iemand 'n buffel... in paragraaf (b) beoog doodmaak rapporteer hy dit binne 24 uur by die polisiekantoor of kantoor van die natuurbewaarder...

Buffels kan dus onder (b) hierbo geskiet word sonder 'n permit van Natuurbewaring, maar moet die persoon dit binne 24 uur rapporteer. Noue samewerking tussen Natuurbewaring en Veeartsenydiens is nodig sodat met die karkas op 'n wyse gehandel kan word wat siekteverspreiding sal voorkom.

Skenking van wild

Art 35 (1) Iemand wat wild skenk, oorhandig saam met die wild aan die ontvanger 'n dokument wat — (dan volg al die besonderhede wat op die dokument moet verskyn)

(3) Iemand aan wie wild geskenk is, dra die dokument by hom wanneer hy die wild vervoer

Vervoer van wild

Art 39 (1) Niemand mag lewende wild aanhou of vervoer nie, tensy hy die houer is *van 'n permit* wat hom magtig om dit te doen.

Invoer

Art 41 (1) Niemand mag 'n wilde dier uit die Provinsie uitvoer of wegneem nie, tensy hy die houer is *van 'n permit* wat hom magtig om dit te doen...

Verskuiwing

Art 46 (1) ... die Administrateur kan ... na raadpleging met die eienaar... 'n beampte van die Afdeling Natuurbewaring skriftelik opdrag gee om daardie soort wilde dier te vang en op die grond in die opdrag omskryf, los te laat.

Uitreiking van permitte

Natuurbewaringsregulasies maak voorsiening daarvoor by:

Administrateurskennisgewing 2030 van 14 Desember 1983 Hoofstuk VI

41 (1) Bepaal dat die Administrateur 'n permit kan —

— uitreik vir die tydperk in die permit genoem

— weier om so 'n permit uit te reik

— uitreik onderworpe aan die voorwaardes wat hy dienstig ag
— wysig, opskort, intrek ens.

Aangesien wild dus ook nie vervoer kan word sonder 'n permit van Natuurbewaring nie, kan laasgenoemde en Veeartsenydiens mekaar aanvul deur aan voornemende vervoerders van wild te wys op die nodigheid om 'n permit van albei bogenoemdes te kry voor die beweging mag plaasvind.

Indien bogenoemde nagekom word, is dit vir Natuurbewaring en Veeartsenydiens moontlik om beheermaatreëls en beleid ten opsigte van die vervoer en verspreiding van buffels uit te voer.

SUMMARY OF PROCEEDINGS OF BUFFALO SYMPOSIUM OF WILDLIFE GROUP OF SAVA

R D BIGALKE; Director, Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa

The purpose of this meeting has not come out clearly from the papers and interesting discussions. Possibly the resolutions will cast more light on the matter. Reading between the lines, however, I think the purpose is to try to find a way to marry the aspirations of game farmers, hunters and game conservationists with the problems of the authorities responsible for the control of foot-and-mouth disease (FMD) and Corridor disease (CD).

One cannot get away from the fact that FMD and CD are the two big obstacles in the way of the freer movement of buffalo.

CD is a killer of cattle and it is consequently not possible to farm with carrier buffalo and cattle on the same farm. CD is not difficult to control. If one has an outbreak on a farm you can stop it relatively quickly by getting rid of either the cattle or the buffalo. The infected ticks will cease to be a problem within a few months.

East Coast fever (ECF) on the other hand is most difficult to control. The big fly in the ointment is the possible transformation of CD into ECF. Although we have not been able to confirm the Kenya results of about 20 years ago in which South African and Kenyan strains of CD were transformed into ECF by passage in cattle, our investigation are not sufficiently comprehensive to be conclusive. We have to accept that transformation is possible and the more carrier buffalo there are around, the better the chances of transformation will be.

We still have a lot to learn about the epidemiology of FMD, but the buffalo in the Kruger National Park is undoubtedly the culprit. Somehow, somewhere and sometime it serves as a source of infection for cattle.

The important point is that one cannot keep buffalo which originate from areas where either FMD plus CD (Kruger National Park) or CD alone (Zululand reserves) occur, together with cattle.

An interesting legal point which emerged, is that movement of Addo buffalo, which are free from CD and FMD, is possible into areas where FMD restrictions do not apply and where the potential vector ticks of CD do not occur. To me this means that movement is more restricted that I had thought. Maps showing the current distribution of buffalo in game reserves and game farms seem to indicate that the abovementioned restrictions are not being consistently applied.

I wish to sound a warning note to the Directorate of Veterinary Services in this regard. The extension of the enzootic areas of FMD and CD into "clean" areas could well occur if there is coalescing of infected and "clean" buffalo populations. This would make control much more difficult and adversely affected agricultural exports in the case of FMD.

Despite the research being done at Onderstepoort and elsewhere, successful control of these diseases in cattle or their elimination from buffalo is unlikely in the short and medium terms.

It seems as if there is a good case for preservation of the Addo gene pool from a conservation point of view. The supply of small numbers of buffalo to game farmers seems unwise on account of the evidence that at least 10 animals are necessary for a population to thrive. The habitat must also be suitable.

The buffalo seems to be in no danger of extinction in the RSA. On the contrary its status seems perfectly safe. The shooting of small numbers of buffalo under false pretences on farms adjoining the KNP is therefore not a problem from a conservation point of view. The high perfection of immobilization and translocation techniques, are a boon not only to preservation but also to studies on FMD and CD.

The social organization of buffalo seems important from a genetic and possibly also epidemiological viewpoint as far as the two diseases are concerned. This should perhaps be considered in the current and future epidemiological studies.

Buffalo can apparently be raised FMD- and CD-free. Our own tests have, however, cast some doubts on the absolute CD-free status of the Zimbabwe herd. The possibility of producing such herds should be investigated in the RSA but should be under official control or supervision for obvious reasons.

Embryo transfer is being investigated but it is still in the embryo stage. Semen of carrier bulls should be checked for FMD virus.

A serious omission from today's deliberations, in my opinion, is a study of buffalo in an integrated management system on game farms. There appears to be in fact an apalling general lack of knowledge of the management of game farms. This is resulting in irreparable damage to an ever enlarging surface area of the habitat concerned, as game farms become more and more fashionable. I think the supply of buffalo to game farmers should not be a priority until such parameters as minimum size of farms, and stocking and cropping rates have been determined.

To summarize, it is clear that we as yet are unable to render buffalo from enzootic FMD and CD areas safe for export to non-enzootic areas. We are also unable to protect cattle from CD. More widespread vaccination of cattle against FMD would bedevil agricultural exports.

Continued research may offer solutions, but only in the longer term. Such research will have to be slotted into research programmes with other higher priorities. Research on FMD and CD is fortunately also being done in other countries.

It would be wise to do much more research on the management of game farms which harbour buffalo before a high priority is given to the supply of Addo buffalo to private farmers.

Better co-ordination of buffalo-orientated research would be useful.

This cannot be enforced but requires a spirit of co-operation. This meeting has gone a long way to foster such a spirit.