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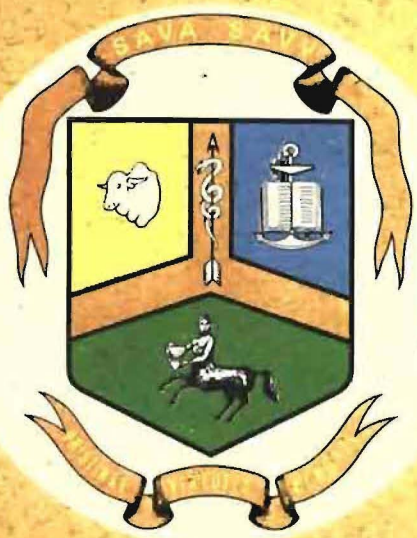

# JOURNAL OF THE SOUTH AFRICAN VETERINARY ASSOCIATION

# TYDSKRIF VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING

SEPTEMBER 1982

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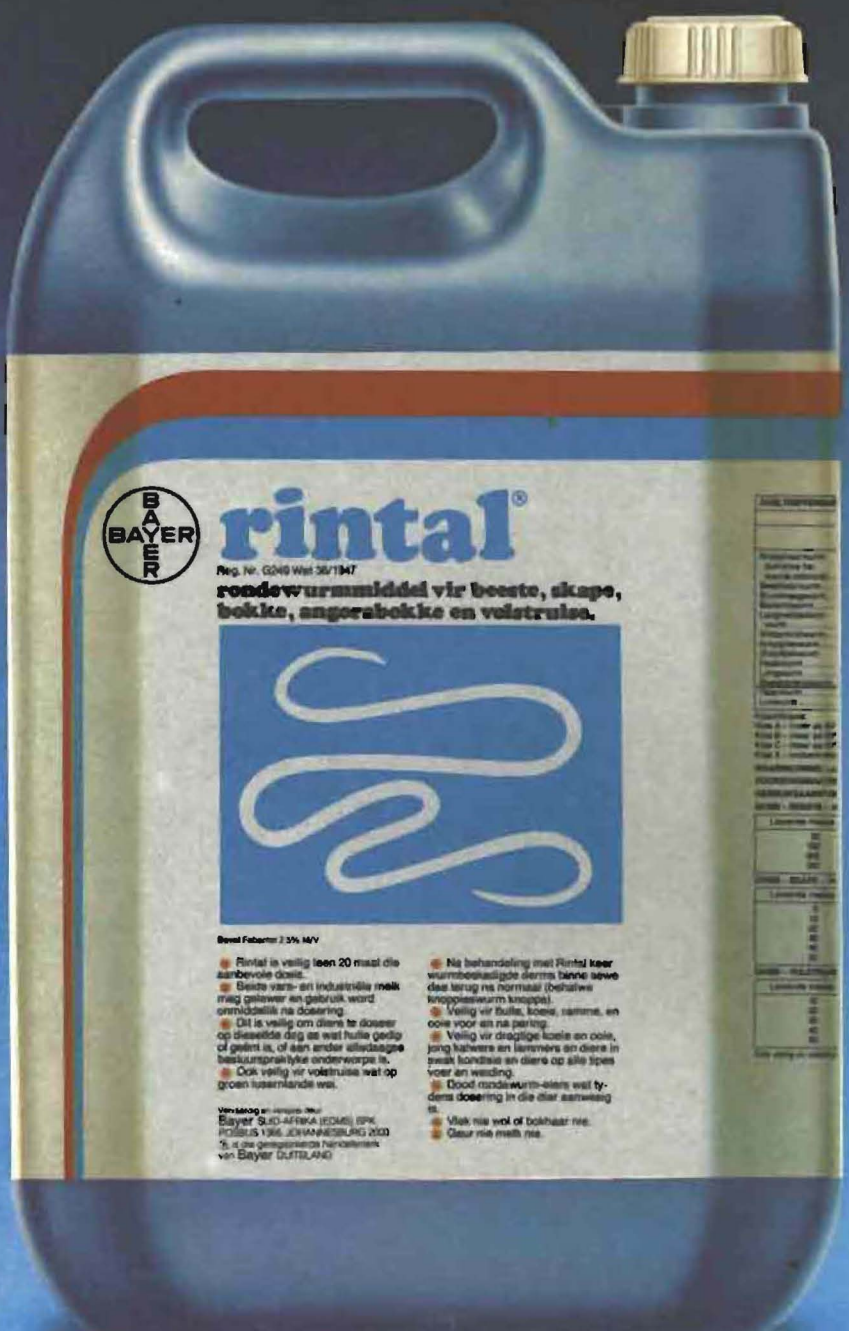


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## TYDSKRIF VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING

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

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# THE IMAGE OF THE VETERINARY PROFESSION VIS-A-VIS THE PUBLIC

We are all members of a proud and honourable profession and whether we work for the State, a Municipality, a University, Commerce and Industry, or as private practitioners, each and everyone of us has a grave responsibility to the public and our profession. It is our individual and collective responsibility to ensure that the image of our profession remains respected in the eyes of the public.

What must always be borne in mind is that the public image of a profession, any profession, is that set by those members of the profession with whom an individual member of the public has contact. Thus, if any of us in our professional capacity does anything that could mar the image of the profession, it is not only the veterinarian concerned who gets a bad name but the profession as a whole. It could be said that such a generalisation is not fair to the profession as a whole (and you would be correct) but one must take cognisance of things as they are and not as one would necessarily like them to be.

Over the past few years, members of our profession have been concerned that there is a tendency for the public to consider that some members of the profession are charging excessively high fees. While this may also be unfair to the profession when all the facts are taken into consideration, it is the duty of the individual veterinarian to be aware of his public relations role and to go out of his way to avoid communication problems with clients.

While nobody will deny the veterinarian the right to a good living by virtue of his training, expertise and service he renders, it must not be forgotten that any animal has either a market, replacement or sentimental value. Because of this every animal has a certain value in the eyes of his owner and this determines what money the owner is prepared to spend on the treatment of it.

Numerous complaints are received by the SAVA from animal owners regarding the fees charged by veterinarians for treating their animals. In most cases the owner has not been informed by the attending veterinarian what the possible course or duration of treatment is likely to be and thus has no idea of what the ultimate costs are likely to be. Consequently when he is finally presented with an account which is higher than he was expecting or was prepared to pay in relation to "his" valuation of the animal, he feels that he has been "milked" and becomes upset with the veterinary profession as a whole.

In other cases, owners complain that they have been "talked into" expensive operations or long courses of treatment for an animal, with the resultant charges being completely out of proportion to the value that the owner places on that particular animal.

Veterinarians are therefore strongly advised to discuss the possible costs likely to be involved with an owner before treatment is initiated thus avoiding later argument and heartache. To discuss costs with an owner is not as *infra dig* as some people seem to think.

Having said all that, we end off by reminding readers that the old adage, "the best form of advertisement is a satisfied client", is as true today as it ever was. We also know that some clients will never be satisfied however hard one tries or whatever you may do, but fortunately they are in the minority and are part of the cross every professional person must bear.

**VOORDRAG** **ADDRESS**

# BESKOUINGS OOR DIE FAKULTEIT VEEARTSENYKUNDE, UNIVERSITEIT VAN PRETORIA*

PROFESSOR J.M.W. LE ROUX

As u kyk na die verspreiding van die omsendbrief wat ek aan u gestuur het, sal u opmerk dat dit 'n baie skerp omlynde gebiedsafbakening inhou, nl. Onderstepoort in sy klassieke drieledigheid: die Navorsingsinstituut, die Fakulteit en Veeartsenydiens. Dit herinner my aan die afgebakende gebied van 'n ou hond wat 33 jaar lank daar ronddwaal – vol ekseem, kroniese nefritis en mastositomas. By tye lig hy sy been bokant en onderkant die pad om daar in "Barbara Woodhouse"-styl sy "quickies-quickies" te maak. U kan my tereg daarvan beskuldig dat dié gebied te klein, te omskrewe, te staties is, sonder verbeelding; dat ek in 'n diep slaap verkeer. As teenprestasie sal ek u dit aanstons voorhou dat daar diegene is wat geredeliker toekomsvisioene ervaar, vooruitskouings kan maak en filosofeer as ek, oor belangrike sake met betrekking tot onderrig, navorsing en die akademiese ontwikkeling van die Fakulteit. Hulle stel dit op skrif. Dit lê onder die stof in kaste en laaie en staan op boekrakke vir 10 tot 20 tot 30 jaar om daar deur die vismotte opgevreet te word. Dan verontskuldig ek myself en sê: ek is nie die enigste Rip van Winkel nie. Gun my daarom hierdie afgebakende gebied, hier by u op Onderstepoort.

Geagte kollegas, ek wil u hartlik welkom heet en wens u 'n baie voorspoedige Nuwe Jaar toe. Ek wil graag my termyn as dekaan begin deur met u te praat. En as my termyn eindig, hoop ek dat ek nog sal kan sê dat ons gereeld met mekaar gepraat, gedagtes gewissel en gesprek gevoer het. Dit is kort en saaklik my administratiewe beleidsverklaring. Dit sal u miskien verbaas want dié van u wat my beter ken, weet dat ek meer oomblikke van stilte ervaar as andersom. Ek het myself egter voor die keuse gestel: óf praat óf briefwisseling, en aangesien vismotte ook briewe opvreet, verkies ek om te praat.

## 1. ONDERRIG

### 1.1 Navorsinggerigte onderrig

Gedurende die afgelope maande het u uself seker afgevra: het hy enige voorkeure? Het hy 'n filosofie? Daarop is my antwoord: Ja, wel! Maar as u van my verwag om my voorkeure te stel en u 'n filosofie voor te hou, wil ek dit graag koppel aan pogings tot doelgerigte praktiese uitvoering. As ek dit, wen of verloor, nie kan of durf doen nie, stel ek nie verder belang nie en sink ek liewers terug in my oomblikke van stilte.

Die primêre funksie van die Fakulteit is in die eerste plaas onderrig. Dit sou treurige dae in die geskiedenis van die Fakulteit gewees het as elke dekaan 'n ander siening, 'n ander filosofie daarop nagehou het. Ek het gaan soek vir 'n leidmotief in ons onderrig. Hoe het ons groot geeste, leermeesters en leiers, die onderrigtaak benader? Die Fakulteit Veeartsenykunde is op 1 April 1920 gestig. Op 9 April van daardie jaar sê Sir Arnold Theiler by geleentheid van 'n gradeplegtigheid: "On the one hand the research officer will, by teaching, remain in constant touch with the literature of his subject as a whole, while on the other hand the student will receive instruction from a teacher who masters his subject from the research point of view as well. Indeed the combination of the two functions will be of the greatest benefit to research itself, and I venture to predict a period of constant progress in the near future, and the solution of problems which have hitherto proved refractory. He (the student) will participate in the investigations of the professors. He will be able to follow their research, and to sharpen his critical mind during the progress of the work itself. It is the training of the critical mind, so necessary for the interpretation of facts obtained by observation and experiment, that makes a successful scientist:" Theiler bepleit hier navorsinggerigte onderrig. Alhoewel sy opmerkings verband gehou het met die intieme wisselwerking tussen die Navorsingsinstituut en die Fakulteit – 'n unieke instelling vir daardie tyd – is hulle vandag nog geldig. Een van die grootste leermeesters wat ons Fakulteit nog opgelewer het, H.P.A. de Boom, sê in 1954 in sy presidentsrede voor die S.A. Biologiese Vereniging soos volg: "Early practical training in the simpler techniques, fairly detailed explanation of the more advanced ones, mention of the importance of ingenious innovations, stressing the value of simplicity and simplification of techniques, active discussion and demonstration of research work done by members of the teaching staff are superb aids in stimulating interest and thought, above all in removing that sense of inferiority that grasps every conscientious student sooner or later and holds him in its inhibiting and frustrating grip." Dit beteken navorsinggerigte onderrig. Na sy terugkeer uit Amerika in 1955 maak De Boom die volgende opmerking: "Die tradisie van klem op kliniese aspekte ter uitsluiting van die meer akademiese benadering gebaseer op breë wetenskaplike beginsels het hier sy nadelige effek." In 1962 na sy terugkeer van 'n tweede besoek aan Amerika sê hy dat die vraag aan hom gestel was waarom Onderstepoort se kursus orals met soveel agting bejeën word. Sy antwoord: "Omdat ons nog soveel klem lê op die basiese onderrig". In 1973 sê B.C. Jansen: "Weens die breë basis van veeartsenykundige opleiding is die graduandi ideaal geskik vir toetrede tot verskeie rigtings en is daar ook alreeds ver hiermee gevorder. Veeartse sal ten alle koste hulle self in dié rigtings moet handhaaf en tred moet hou met die snelle vordering in wetenskaplike kennis op hierdie gebiede." In 'n redaksionele artikel (1973) oor veeartsenykundige

*Rede gehou op 15 Januarie 1982 voor lede van die doserende personeel en ander belangstellendes.

opleiding in Suid-Afrika, word dit soos volg gestel: "'n Ander tradisie wat steeds met trots gehandhaaf is, is die deeglike sorg wat aan basiese, wetenskaplike gefundeerde opleiding gegee word. Alle pogings, van watter aard ookal, om die kursus af te water, is kragtig teengestaan. 'n Gees van kritiese self-analise met volgehoue pogings om die kursus te verbeter en by nuwe eise aan te pas, is 'n verdere kenmerk van die Fakulteit se benadering tot sy taak." By geleentheid deel C.M. Cameron my mee dat die voorlesings van professore Weiss en Jansen hom as student so geïnspireer het dat hy besluit het om navorsing sy loopbaan te maak. In 1981 sê R.I. Coubrough in sy professorale intreerede: "The vitality of this research program must reflect on undergraduate exposure. The undergraduate student must feel the vibrant excitement of research, must experience the thrill of success, must know the pang of disappointment which serves to provide the incentive to try again with undaunted enthusiasm. For it is during these student formative years that the seedbed for the future is indelibly laid."

Verwag u nog dat ek 'n ander filosofie daarop moet nahou? Ek kan dit net herbevestig en beklemtoon, nl. *'n breë, wetenskaplik gefundeerde navorsingsgerigte onderrig*. Dit was so van Theiler in 1920 tot Coubrough in 1981. Ek wil hê ons moet hieroor gesprek voer en onsself afvra: is dit ons erns? As die antwoord bevestigend is, moet ons dit ook doelgerig uitvoer. Ons moet besin hoe elke dosent in sy dissipline probleemstelling in sy onderrig kan voorhou, enkelinge of groepies wat belang stel aan die hand kan neem, verder lei en aan hom of hulle die probleem verduidelik, hoe dit aangepak en nagevors kan word. Dit sal opofferings verg. Die Navorsingsinstituut, Veeartsenydiens en ons kollegas daar buite kan ons baie help. Dit sal ook probleme en navorsing van landsbelang by ons studente inskerp.

### 1.2 Intradepartementele integrasie

In sommige departemente is een man-dissiplines wat daar tuishoort of wat gerieflikheidshalwe daar afgelaai is omdat daar nie 'n ander departement vir hulle is nie. Dit plaas 'n groot las op daardie dosente se skouers. Hulle moet man alleen toetse en eksamens afneem en dit veroorsaak ernstige vertraging met die bekendmaking van toetsuitslae. As sulke dosente vir lang tydperke studieverlof neem, het dit roosterimplikasies. Opvolging kom ook ter sprake. Hoe moet hulle lesings behartig word? Integrasie behoort so te ontwikkel dat daar minstens 3 panele eksaminatore by mondelinge en skriftelike toetse en eksamens is. Memorandums kan opgestel word om by skriftelike toetse en eksamens gebruik te word. Ons moet waak dat oorspesialisasie nie in so 'n mate posvat dat 'n dosent nie meer bruikbaar is in sy eie departement nie. Elke departement het sy eie behoefte en probleme. Ek sal dié aangeleentheid mettertyd met u kom bespreek om vas te stel hoe integrasie sinvol toegepas kan word.

### 1.3 Voortgesette opleiding

Die belangrikheid van voortgesette opleiding hoef beswaarlik beklemtoon te word. In 1973 sê Jansen: "Dit is verblydend om te sien dat voortgesette opleiding in die vorm van opknappingskursusse al posgevat het in ons professie. As ons veeartse nie aan voortgesette opleiding deelneem nie, sal hulle kennis baie gou verouder: hulle sal nie meer bekwaam wees om hulle professie volgens die nuutste metodes te beoefen nie en die agting van die lede van ander beroepe verloor." Ons het verlede jaar 'n baie suksesvolle opknappingskursus oor beeste aangebied. In Maart vanjaar bied ons een oor kleinvee aan. Dié kursusse word nou 'n jaarlikse instelling en word aangebied binne die raamwerk van ons onderrigprogram en beskikbare fasiliteite. Gepaard hiermee beplan ons ook boeredae op die fakulteitsterrein of elders. Die eerste een wat oor melkbeeste handel word reeds in Julie vanjaar aangebied. Op hierdie manier wil ons ook probeer om die fakulteit beter bekend te stel. Die onkunde by die algemene publiek oor Onderstepoort is skokkend, nl. dat daar twee instellings is, tewete die Navorsingsinstituut en die Fakulteit. Ek wil 'n beroep doen op ons kollegas van die Instituut en van Veeartsenydiens om, waar hulle in raadsale vergader en waar hulle met die publiek in aanraking kom en die geleentheid paslik is, met dié voorligting behulpsaam te wees. Die nou verband tussen onderrig en navorsing en die onderlinge wisselwerking moet beklemtoon word. Die een kan nie sonder die ander gedy nie.

### 1.4 Die kuddebenadering

Die kuddebenadering in veeartsenykunde is niks nuuts nie en dit is ook nie onlangs ontdek nie. Dit is o.a. wat ek in gedagte gehad het toe ek aanvanklik gesê het dat dit wat vir onderrig en navorsing belangrik is, op skrif gestel word, en dan in kaste en laaie gebêre word om daar deur vismotte opgevreet te word. 30 Jaar gelede, in 1951, sê S.W.J. van Rensburg: "By virtue of our training and our close contact with agriculture we are in a better position than any other to observe defects and offer suggestions for improvement. To that end we must realise that the treatment of sick animals forms but the smallest part of our responsibility to the country and its people. Apart from it being a national duty, we must realise that the ability to detect and suggest remedies for allied subjects like over-stocking, wrong veld management, incorrect feeding and faulty methods of breeding and management enhance the status and the prestige of the veterinarian far more than his mere skill as a clinician." Van Rensburg herhaal hierdie pleidooi in 1952. Sedertdien is die kuddebenadering bepleit deur K.M. van Heerden in 1962 en deur M.C. Lambrechts in 1963. In daardie jaar word dit ook beklemtoon in 'n simposium oor die rol van die veearts in die moderne wêreld.

Ek vra myself af: hoe gebeur dit dat ons die kuddebenadering in veeartsenykunde sedert 1951 gereeld propageer en dit eers 30 jaar later as beleidsrigting in ons onderrigprogram opneem? Hoe is dit moontlik dat ons dit in werking stel en 3 jaar later ervaar dat 'n leerplan vir die kursus ontbreek? Daarop is my antwoord: ons praat nie genoeg met mekaar nie, daar is nie gereelde gedagtewisseling, gesprekvoering, onderlinge raadpleging en besinning nie.

Met die klem op kuddebandering beland ons in die gebied van die veekundige. Die veearts se rol in veeproduksie is duidelik omlyn in 'n redaksionele artikel in ons joernaal in 1974. Elke professie kan in hierdie verband 'n waardige rol speel en sy deel bydra sonder om mekaar met agterdog te bejeën. Die veearts moet ook in staat wees om die biologiese potensialiteite van 'n kudde te bepaal en die boer se bankbestuurder dienooreenkomstig te adviseer. Die fakulteitskomitee wat die leergang van bevorderende dieregesondheid ondersoek, moet hieroor doelgerigte leiding gee.

### 1.5 Die eerste studiejaar

Die eerste studiejaar word nie sinvol benut nie. Sedert

1979 vind keuring op matriekvlak plaas en is anatomie in die eerste studiejaar ingevoer. In die Fakulteite Geneeskunde en Tandheelkunde is dié stelsel reeds geruime tyd in werking. Jansen het dit ook in 1973 na aanleiding hiervan vir ons Fakulteit bepleit. Dis nie genoeg nie. Die aanbieding van makroskopiese anatomie sonder histologie, fisiologie en embriologie is die gevolg van 'n ongelukkige dwaling. Dit is eweseer 'n dwaling in onderwysmetodiek. En dit is hoog tyd dat die pleitbesorgers van die veel geprese tersiêre onderwysdiploma hulle stemme hieroor laat hoor. Jaarkursusse in die tradisionele vakke van die eerste studiejaar is nie die alfa en omega van die oorgang van skool na universiteit nie. Plant- en dierkunde is reeds tot semesterkursusse gereduseer sonder enige nadelige gevolge. In 1981 spreek Grosskopf die mening uit dat chemie en fisika ook tot semesterkursusse gereduseer kan word. Hierdie siening verdien ons ernstige aandag en dit bied 'n praktiese oplossing om minstens 200 periodes van die tweede studiejaar na ander studiejare oor te dra waar dit sinvol gebruik kan word. Ons kerm heeldag dat ons kursus oorlaai is. Dan kan hierdie tyd gebruik word om studente in staat te stel om meer van die biblioteek gebruik te maak of dit kan as lesingvrye periodes ingedeel word. As dit bots met die gebruik in die Fakulteite Geneeskunde en Tandheelkunde, moet ons ons saak stel en behoort die Universiteit ons Fakulteit op eie meriete te behandel.

Ons keuringsmeganisme is op akademiese prestasie gebaseer, suiwer rekenkundig en daarom objektief. Daar is geen ruimte vir misbruik nie. Die matrikulant het egter 'n agterstand teenoor kandidate met een of meer jare universiteitsopleiding. Die twee groepe behoort afsonderlik op eie meriete gekeur te word.

Die toelating van buitelandse studente moet ook hersien word. Ek is die laaste een wat die deur in buitelanders se gesig wil toeklap. Ons durf nie toelaat dat hulle ons Fakulteit gebruik om krediete te verwerf om in hulle eie lande se Fakulteite toegelaat te word nie. Dan staan hulle in die pad van hulle eie mense wat die kursus hier wil voltooi of in die pad van Suid-Afrikaners. Ek verwys na Amerikaanse studente – om enige misverstand uit die weg te ruim.

### 1.6 Geskiedenis van die Fakulteit

Dit is jammer dat ons studente teenswoordig weinig of niks ervaar van die geskiedenis van Onderstepoort en die Fakulteit se verbintenis met die Navorsingsinstituut nie. Hulle weet nie wie ons mense is nie. Hulle ken nie ons helde, ons leermeesters, ons navorsers nie. Nogtans is hulle trots op die "Onderstepoort-gees" bloot op grond van oorlewering. Ek haal aan: "For on analysis this "spirit of Onderstepoort" is found to be neither more nor less than the reflection of Theiler's own spirit in a devoted band of followers: it still exists and will exist, notwithstanding his passing. . . . it is that ideal of service, that conscientiousness of endeavour, that pride in a task, that confidence of success in the face of difficulties, of which Theiler was as true an example as the world will ever know." Daar rus 'n morele verpligting op ons om hierdie gees aan hulle oor te lewer. Ek pleit vir een voorlesing per jaar per departement oor die geskiedenis van ons Fakulteit en sy mense.

## 2. DEPARTEMENTELE NAVORSING

Die universiteit erken die beginsel dat dosente wat hulle primêr op onderrig toespits ook die hoogste sport kan bereik. Die "publish or perish"-siening geld nie noodwendig nie. Ekonomiese omstandighede dwing die Universiteit om nuwe doseerposte te beperk. Laat ons vir die doel van my argument aanneem dat die dosentegetal in elke departement die versadigingspunt bereik het. Weens doseer- en roetineverpligtinge is daar 'n gebrek aan tyd vir navorsing en vordering is stadig. Dit lei tot vertwyfeling. Oordraging van fondse word aangevra en vorderingsverslae word in 'n mate kunsmatig ingeklee. Ons kan nie bekostig dat dosente 'n stadium bereik dat hulle nie meer navorsinggerig is nie. In 1953 stel E.M. Robinson dit soos volg: "The veterinary profession cannot carry on without research. If it attempts to do so it will become static and finally consist of quacks." Ons moet 'n stelsel in werking stel waarvolgens 'n departement 'n dosent vir 'n tydperk van 3 of 4 maande vrystel van doseer- en roetineverpligtinge sodat hy kan navorsing doen. Dit sal opofferings van almal verg maar uiteindelik sal almal meer werksbevrediging ervaar. Op dié wyse sal dosente wat nagraadse studies doen ook die geleentheid kry om hulle vir eksamens voor te berei en vordering te maak met navorsing vir verhandelings en proefskrifte. Ek vind dit jammer dat dosente hulle studieverlof hiervoor moet inboet.

## 3. DEPARTEMENTELE INSTELLINGS

Oor ons departemente neem ek sterk standpunt in. Onafhanklike akademiese departemente is die hoekstene van ons Fakulteit. Daarin kan dosente hulle uitleef en kan onderrig en navorsing gedy en tot volle ontplooiing kom. Dit bied geleentheid vir sinryke integrasie in die vakgebiede van die departement en maak voorsiening vir opvolging. In die departemente as funksionele eenhede van die fakulteit kan die grootste werksbevrediging ervaar word. As lid van die begrotings- en aanstellingskeurkomitee gedurende die afgelope jare weet ek dat dosente wil spesialiseer, sowel op vakgebied as op spesievlak. Hulle vra selfs oorplasing na ander departemente waar hulle hulle voorkeure op sowel vak- en spesievlak kan bevredig. Jansen (1969, 1973) bepleit hoofsaaklik die spesiebenadering. Vir my hou dit ook departemente in. Ons kliniese departemente kan opgedeel word vir groot- en kleindiere. En waar kan die kuddebenadering beter beklemtoon word as juis in so 'n departement? Die rol van kleinvee – skape, bokke en varke – in die volkshuishouding regverdig 'n onafhanklike Departement vir Kleinveesiektes. In fakulteitsverband kan dit aan ambulatoriese kliniek gekoppel word waar dit die beste tuishoort. Die landelike praktisynsgroep by monde van Wilson (1981) het ook 'n sterk pleidooi gelewer vir so 'n departement. Die instelling van sulke departemente hoef nie afbreuk te doen aan die breë basis van ons onderrig nie, ook nie aan basiese onderrig nie. Dit kan dit net versterk. Daar gaan reeds stemme op vir 'n Departement Bevorderende Dieregesondheid.

Ons het 16 departemente gehad. Dit is tot 9 gereduseer. En hier lê die kern van ons personeelprobleme. Ons loop heeldag en kanker oor die vestiging van die Fakulteit, of dit by die Chryslerfabriek of by die spookhuis (dis nou Erasmusrand se spookhuis) moet wees. Maar oor die gebrek aan departementele instellings bewaar ons die swye. Omdat daar nie departementele ontwikkeling is nie, is daar ook nie personeelontwikkeling nie. Ons dosente sit vasgevang in 'n fakulteit waar daar

geen beweegruimte is nie. Hulle word gedwing om dit gelate te aanvaar of te bedank en na die winsgewender privaat praktyk terug te keer. Dit is vir my onaanvaarbaar dat Veterinêre Volksgesondheid 'n agterryer, houthaker en waterdraer van die Departement Patologie is; dat Farmakologie en Toksikologie in ons land met sy ryk gifplantflora 'n aangenaaide lap van die Departement Fisiologie is; dat Pluimveesiektes 'n aanhangsel van die Departement Infeksiesiektes is terwyl 35% van alle eiwit wat verorber word van pluimvee afkomstig is. In elke departement van ons Fakulteit word hoofstukke gewy aan die pasgebore dier, sy anatomie, fisiologie, voeding, versorging en siektes – lammers, kalwers, vullens, varkies, hondjies en katjies. Dit is van landsbelang dat vrektes op dié vlak bekamp word. Ek pleit vir die instelling van 'n Departement Veterinêre Pediatrie waar doelgerigte en gekoördineerde navorsing gedoen en onderrig gegee kan word.

En nou kan ek ons akademiese filosofie formuleer, nl, 'n basiese, navorsinggerigte onderrig binne die raamwerk van 'n onbeperkte aantal onafhanklike, akademiese departemente. Ek praat nie van 'n sinryke, ideale of realistiese aantal departemente nie want dit impliseer beperking.

Die beperking op die aantal departemente in ons fakulteit het 'n ander ernstige implikasie wat ook met personeelontwikkeling verband hou. As gevolg daarvan is hier kollegas in die Fakulteit wat nie departementshoofde kan word nie. Hulle word hulle regmatige aansprake op erkenning ontsê, tot by die stoel van die Dekaan. Dit is 'n ongesonde en onhoudbare toestand.

Die eerste 3 punte hier bo genoem, nl onderrig, departementele navorsing en departementele instellings is my persoonlike voorkeure, my skerp omlynde gebiedsafbakening. Daarvoor wil ek my graag beywer.

## 4. VESTIGING VAN DIE FAKULTEIT

Die Fakulteitsraad het besluit dat die Fakulteit op 'n ander terrein gevestig moet word. 'n Komitee van die Adviserende Raad op Universiteite is besig met 'n ondersoek en sal aan die Universiteitsraad verslag lewer. Die aangeleentheid is daarom uit ons hande uit tensy dit terugverwys word. En dan neem ek nie die besluit nie, maar die Fakulteitsraad. Dit weerhou my egter nie om my persoonlike mening uit te spreek nie en ek het dit ook by geleentheid aan kollegas meegedeel.

Daar word spottenderwys gesê, openlik en bedek, dat die Navorsingsinstituut op die Fakulteit se rug ry. Dit weerspieël diegene wat so sê se lekker humorsin. Afhangende van die gesindheid waarin dit gesê word, beindruk dié wat dit sê niemand nie behalwe hulleself. Die teendeel is egter die waarheid, maar dit moet in die regte perspektief gestel word. Sedert 1958 en ook sedert 1973 was die dosente van die Fakulteit verantwoordelik vir ingrypende akademiese hervorming – ten goede. Te oordeel aan die aantal publikasies het hulle ook pragtig op navorsinggebied gepresteer. Die roem en glorie van Onderstepoort in die afgebakende gebied waarin ek dit gestel het, is ons almal se besit. Die Navorsingsinstituut sal die eerste wees om die onderlinge akademiese wisselwerking in sy eie ontwikkeling te erken.

Die probleme en knelpunte van die Fakulteit het niks met hervestiging te maak nie. Trouens, ons voorleggings voor die Komitee van die ARU het suiwer om fasiliteite gegaan. Ons kan nie ons studente onderbring nie. Ons probleme word verder in 'n stilstaande personeelontwikkeling weerspieël.

Veeartsenykundige navorsing en onderrig in Suid-Afrika is sedert 1908 gekoppel aan die naam Onderstepoort. Dit geld sowel binnelands as buitelands. En dié van ons wat dink dat ons dit in ons leeftyd gaan ontkoppel, leef in 'n gekkeparadys.

Ons beweer dat die gebied om die Fakulteit geïndustrialiseer raak en dat die kliniese materiaal opdroog. Aan die oostekant van Pretoria sal dit blykbaar in die toekoms nie gebeur nie. Dit klink vir my kunsmatig. Maar as dit 'n werklikheid word, moet die owerhede vir ons 'n privaat praktyk op die randgebied van die stad koop sodat ons daar in ons eie reg kan praktiseer en ons regmatige aandeel van die kliniese materiaal kan bekom.

Die Bek- en Klouseerinstituut is by die Fakulteit geleë. As die virus ontsnap word ons onder kwarantyn geplaas. so beweer ons. As die fakulteitsterrein aan die oostekant van Pretoria geleë is en die virus ontsnap, word ons ook onder kwarantyn geplaas. Maar dit verswyg ons gerieflikheidshalwe. Dit klink vir my ook kunsmatig en ek betwyfel of ons die mense wat die besluite moet neem hiermee gaan beïndruk.

As ons ons saak wil bevorder, enige saak, en in die gang van sake ons tradisies soos pêrels voor die swyne werp, ons rug keer op monumente wat ons self help oprig het, ons in die woestyn wil isoleer, daarby aangespoor word deur persoonlike vooroordele, persoonlike vetes en gerief, wetenskaplik gefundeerde feite wil verdoesel, dan verkrag ons ons eie gewete, beveg ons onsself en het ons die stryd verloor voordat ons dit aangeknoop het. En as u nou miskien dink dat ek om die saak heen praat en nie ter sake kom nie, sê ek: ek hoop en bid dit gebeur nie. En as ek as gevolg van my persoonlike siening as Dekaan moet val, dan val ek.

## 5. MEDUNSA

Ek wil prof. Owen baie sukses toewens met sy onderneming. Die stigting van 'n tweede fakulteit is 'n gesonde ontwikkeling. Hulle is daar besig met 'n ongeëwenaarde proefneming van interfakulteits-, interdepartementele- en interdissiplinêre integrasie wat ons met belangstelling sal volg. Op die biomediese terrein het die veeartsenykundige professie nog altyd konstruktiewe bydraes gelewer. Daar word beweer dat Medunsa dosente van ons gaan afrokkel. Dit gebeur gereeld tussen universiteite. En as een van ons dosente met verhoogde status en bevordering daarheen kan gaan, hoekom nie? Ek beskou dit nie as 'n bedreiging nie. Enigeen wat weet en wat ons fakulteite besoek, die een oud die ander nuut, sal besef dat die ervaring by die moederfakulteit lê. En dit is na alles waarom dit gaan.

'n Minder aangeneme faset is dat die twee Fakulteite en die Navorsingsinstituut nou met mekaar kompeteer vir die indiensneming en behoud van deskundiges. Dit maak die deure oop vir 'n mate van afpersing waaraan ek nie graag wil deelneem nie. Met die tekort aan deskundige mannekrag kan óf navorsing in landsbelang óf onderrig daaronder ly en ek kan nie 'n bevredigende oplossing aanbied nie. Maar daaroor kan ons besin en gesprek voer sonder dat identiteit prysgegee word.

## 6. DERDE FAKULTEIT

Daar bestaan meningsverskil oor 'n 3e fakulteit. Per-

soonlik is ek ten gunste van 'n 3e fakulteit as die land dit kan bekostig en daar vir almal werksgeleentheid is. Lede van ons professie het baie tyd gespandeer en opofferings gemaak om dit te propageer. Geen regdenkende mens kan egter van my as Dekaan en van ons as 'n instelling verwag om 'n 3e fakulteit te propageer terwyl die moederfakulteit in 'n verknorsing sit nie. Ons hier op Onderstepoort en die professie daar buite moet liewers ons kragte saamspan en ons daarvoor beywer om ons eie fakulteit in orde te kry en dan kan ons weer gesamentlik voorspraak maak vir 'n 3e fakulteit.

## 7. ADVIESKOMITEE

Daar is myns insiens 'n ernstige gebrek aan gesprek op alle vlakke in ons Fakulteit. Om te verwag dat besinning en beplanning van 13h15 tot 13h45 in die Dekaan se komiteekamer moet plaasvind as almal op hulle horlosies kyk, is 'n bietjie vergesog. Die vak Interdissiplinêre Veeartsenykunde of Bevorderende Dieregesondheid soos dit nou genoem word, is 'n sprekende voorbeeld. Na verloop van 3 jaar is daar vir hierdie vakgebied nog nie 'n leerplan beskikbaar nie. Ek beskou dit ook nie as die alleenreg van die Dekaan om te besin nie. Ek wil graag gereeld en op gesette tye met u gesels oor alles wat op die Fakulteit betrekking het. Met gereelde gesprekvoering kan ons ons gedagtes orden, kan daar terugvoering wees, kan kontrole uitgeoefen word, kan almal op 'n beskaafde manier hulle standpunte stel en kritiek uitoefen, kan praktiese uitvoering in rat gegooi word en kan ideë wat geopper word, na die regte kanale gevoer word. Ek het besluit om u almal en by geleentheid kollegas van al die sektore van ons professie by 'n advieskomitee te betrek wat my van raad sal bedien. Niemand sal sy identiteit prysgee nie. Daar word nie besluite geneem nie. Absolute informaliteit is egter 'n voorvereiste. Daarom kan sulke samesprekings nie op die fakulteitsterrein gehou word nie. Ek sal u na my privaat woning uitnooi waar ons oor 'n koppie tee gedagtes kan wissel.

## 8. TRIBUTE

It has been my good fortune to study, work or teach under all directors of the Veterinary Research Institute except Sir Arnold Theiler and the present one, namely: Drs. P.J. du Toit, De Kock, Quin, Alexander, Jansen and Weiss. It has also been my good fortune to study, work or teach under all Deans of this Faculty except Sir Arnold Theiler, namely: Prof. P.J. du Toit, De Kock, Quin, Fourie, Graf, R. du Toit, Jansen and Hofmeyr. I have worked in the shadow of respected colleagues, to name but a few at random: Jackson, De Boom, Clark, Thomas, Schultz, Neitz, Robinson, Scheuber, Parkin, Quinlan, Van Rensburg, Steyn, Bisschop, Groenewald, Haig, Coles, Van Drimmelen, Henning, Sterne, Diesel, Lambrechts, Polson, Louw, Ortlepp, Gertrude Theiler, Henrici, Reimerschmied, Rossouw, Van Wyk.

They and their contemporaries have built the monuments of Veterinary Science in this country – monuments of veterinary research and education, original thought and scientific experiment, perseverance and personal sacrifice. I am too hesitant to state that I am following in the footsteps of any one of them. Ons this occasion, however, it is my personal and sincere desire to tell you that in my dealings with Faculty and all matters related to it, I shall always honour them, I shall never forget them.


### DANKBETUIGING

Geagte kollegas, met die dekaansverkiesing verlede jaar het u my onselfsugtig ondersteun. Sedertdien het u my baie welkom laat voel. Ek wil graag my opregte dank teenoor u uitspreek. Ek verbind my nou tot die diens van ons Fakulteit en: tot u diens!


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# HIGH INCIDENCE OF SQUAMOUS CELL CARCINOMA OF THE VULVA IN MERINO EWES ON A SOUTH AFRICAN FARM

R.C. TUSTIN*, D.J. THORNTON** and H. McNAUGHTON***

ABSTRACT: Tustin R.C.; Thornton D.J.; McNaughton H. **High incidence of squamous cell carcinoma of the vulva in Merino ewes on a South African farm.** *Journal of the South African Veterinary Association* (1982) 53 No. 3, 141-143 (En) Department of Pathology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

Squamous cell carcinoma of the vulva of 11 ewes has occurred in a flock of 450 Merino ewes in the Graaff-Reinet district during the last 3 years. Mules' operation and tail docking of the lambs are routine managemental procedures and their possible aetiological role, together with that of the resultant increased exposure of the part concerned to solar radiation, is discussed.

Key words: Carcinoma, perineum, solar radiation, sheep.

## INTRODUCTION

Neoplasms of the skin of sheep are relatively uncommon in the Republic of South Africa. During the last decade, however, squamous cell carcinomas of the skin have been diagnosed with an apparent increasing frequency, the most commonly affected sites being the skin of the perineal region including the vulva, the ears and, rarely, the muzzle.

This report describes the occurrence of squamous cell carcinoma of the vulva in a flock of Merino ewes in the Graaff-Reinet district of the Cape Province.

## HISTORY

The owner of the affected flock has been farming with sheep on the same farm for the last 21 years. He runs a flock comprising 450 mature ewes, 200 wethers, 150 rams and 200 young ewes. The first case of the condition was observed in October 1980 and has subsequently been seen in 10 other ewes. All affected animals have been mature ewes whose precise ages are not on record but all have been older than 4 years of age. The Mules' operation has been performed for the last 11 years on all the sheep on the farm when lambs are about 6 weeks of age. The tails of all lambs are docked.

## CLINICAL SIGNS

Most of the tumours have been first noted to occur at the junction of the skin and mucous membrane of the vulva. They grew rapidly and reached the size of approximately a man's fist within about 2 months of first being observed. The overlying skin and mucous membrane frequently ulcerated, became covered by a dirty black exudate and was unpleasantly odoriferous. Interference in the passage of urine in several of the cases due to the presence of the tumour led to wetting of the wool of the hindquarters and the subsequent decomposition of the urine and fly strike added to the unpleasant nature of the condition. Only one ewe, however, showed any noteworthy clinical signs apart from the neoplasm; this was frequent tenesmus.

All the affected animals were destroyed before the neoplasms became excessively large. Two of these were necropsied. One of them was 6 and the other 7 years old.

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## PATHOLOGY

Both of the ewes showed similar lesions which consisted basically of large irregularly shaped neoplasms which involved and protruded from the floor and lateral walls of the vulva to the externum (Fig. 1 & 2). The part of the tumour exposed to the air was covered by a black crust of dried exudate and was ulcerated. It had an uneven surface somewhat resembling that of a cauliflower. On dissection of the part concerned, the neoplasms were observed to have infiltrated irregularly and deeply into the perivulval and perivaginal connective tissues, thus exerting pressure on the adjacent wall of the rectum which itself was not involved. An approximately 160 mm length of the caudal vaginal walls of both cases was affected. The neoplastic tissue was firm and pinkish white. No metastases to regional lymph nodes or elsewhere were observed macroscopically or microscopically in either of the cases.

Neoplasms from 5 of the cases were examined histologically. All were typical squamous cell carcinomas.

## DISCUSSION

Squamous cell carcinoma of the perineal region has not previously been reported in sheep in the Republic of South Africa although it has been encountered in relative high incidence on at least 3 other farms. Apart from Australia, particularly Western Australia, and France it appears that the neoplasm does not occur in other countries[1-4]. It has also been diagnosed recently in the pinnae of the ears of 3 mature Merino ewes in a flock of sheep at the Veterinary Research Institute, Onderstepoort and in the skin of the muzzle of an Ile de France sheep.

Lloyd[3] drew attention to the possible role of ultraviolet radiation in the aetiology of the neoplasm as its predilection sites in the skin are those parts lacking pigmentation and with sparse hair covering and which are exposed to sunlight, such as the ears, muzzle and, in docked animals, the perineal region. He also speculated on the role that Mules' operation and prior photosensitisation might have on the development of the tumour. Mules' operation is a radical surgical procedure to remove skin folds of the perineal region, and is carried out to reduce the incidence of cutaneous myiasis.

Vandegraaff[4] also suggested that the high incidence of vulval squamous cell carcinoma was related to an increased exposure of the perineal region to direct sunlight as a result of the short docking of the tails and the performance of Mules' operation. He states that field observations indicate that tumours of the perineal skin


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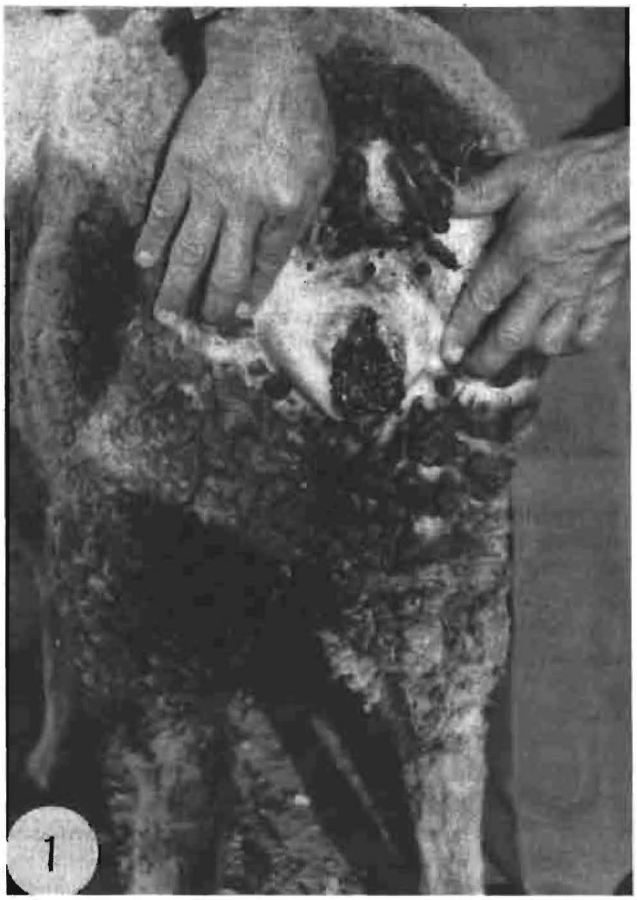

Fig. 1. A protruding squamous cell carcinoma of the vulva.

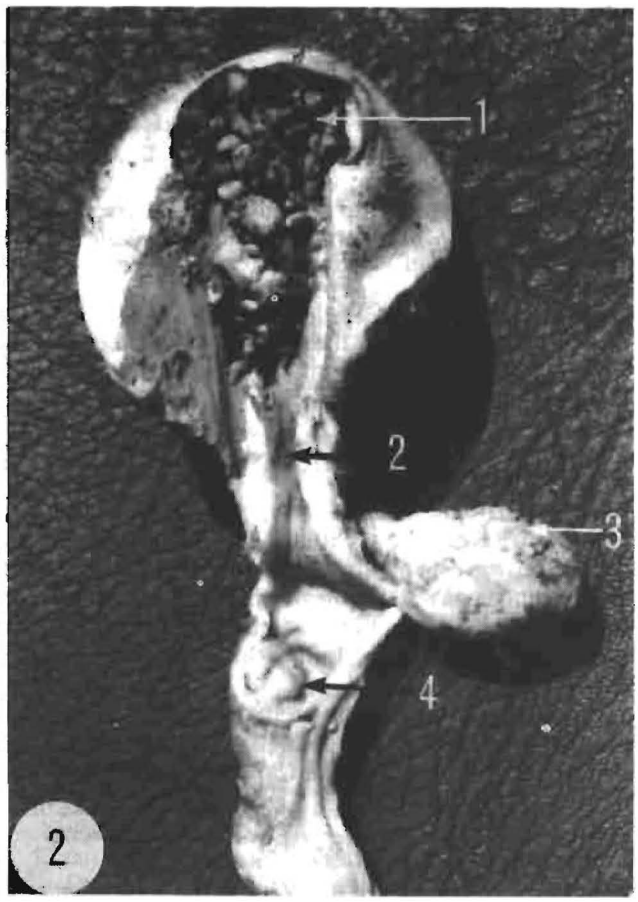


Fig. 2. Part of the genital tract of an affected ewe after formalin fixation and removal of the dorsal vaginal wall to reveal the squamous cell carcinoma. 1 = neoplasm in vulval and vaginal wall; 2 = vagina; 3 = urinary bladder; 4 = cervix.

rarely developed in sheep subjected to the Mules' operation if the tails, when docked, have been left sufficiently long so that they cover the ventral tip of the vulva. In addition, contraction of the surgical skin wound during the healing process leads to a partial opening of the lips of the vulva, thus creating an increased exposure of the mucocutaneous junction of the vulva to sunlight. The results of a recent survey in Australia also implicate the possible roles played by Mules' operation and solar radiation, as well as the time of the year of shearing; a higher incidence occurring on farms where ewes are shorn between January and June[1].

Mules' operation in Merino sheep is practised to some extent in this country while the docking of sheeps' tails of many breeds including Merinos is a very common procedure. On the farm under review Mules' operation had first been practised as a routine procedure on all sheep 9 years before the first case of squamous cell carcinoma of the vulva was encountered, while the tails of all sheep are docked. If these plus solar radiation were the only factors involved in the aetiology of the condition, one would expect a much wider distribution and higher incidence of the condition in the sheep population of this country. Vandegraaff[4] also suggests that the possible influence of a co-carcinogen must be considered and speculates on the possible additional role played by the repeated application of organo-phosphate compounds to the perineal region of sheep in order to treat and prevent cutaneous myiasis, although as far as is known none of these compounds is carcinogenic. Such compounds and others are regularly used by farmers in South Africa to control blowfly larval and tick infestations in their sheep and on the farm in question compounds containing pyrethrin had been used until a year ago until being superseded by one containing an organo-phosphate as active ingredient.

Although the precise age of the affected ewes was not known, they were all mature animals more than 4 years of age. This corresponds with the findings of other reporters[1-4]. Legadic[2], who described a high prevalence rate of vulval squamous cell carcinoma in a flock of Ile de France sheep in France, considered that the principle predisposing factor seemed to be the age of the ewe. In that particular flock the ewes are kept to an advanced age. The incidence ranged from no tumours in animals aged 6 years or under, to 27,7 % in those aged 9 years or over. In South Africa on the 4 farms on which the condition affecting the vulva of sheep has occurred, 3 have involved the Merino breed and one Ile de France. In Australia, Merino ewes have mainly been affected but Border Leicester – Merino cross ewes have also been involved[1 3 4].

Hawkins at al.[1] have recently recorded the results of a survey which was conducted to determine the epidemiology and incidence of squamous cell carcinoma of the perineal region of sheep in the wheat belt of Western Australia where the disease is particularly prevalent. Eighty-two per cent of 80 flocks were affected, and the

mean incidence in ewes of the affected flocks was 2,29 % (range 0,12 % – 4,0 %). Incidence increased with age, from 0,05 % in 1-2 year old ewes to 3,09 % in ewes over 5 years of age.

Squamous cell carcinomas of the ovine perineal region grow by expansion and infiltration. No reports of metastasis have been encountered in the literature and none were seen in the 2 cases necropsied by us. It seems, therefore, that this neoplasm metastasizes at a late stage, if at all, but this particular aspect requires further investigation. Lloyd has, however, observed metastases to regional lymph nodes in 3 of 28 Merino sheep suffering from squamous cell carcinomas of the ears[3]. Treatment of the vulval lesion to be effective, would require wide surgical excision of the tumour at a very early stage of the disease. Owners of animals who have had cases of the disease in their sheep should be advised to keep their ewes under careful observation so that surgical intervention can be resorted to as soon as possible. They should also be informed of the possible role played by sunlight, short tail docking and the Mules' operation in the aetiology of the condition. Legadic[2] reports that recurrence of tumour growth following surgical extirpation occurs if the neoplasm is greater than 20 mm in size.

ACKNOWLEDGEMENT

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## BOOK REVIEW — BOEKRESENSIE

## SURGERY OF THE REPRODUCTIVE TRACT IN LARGE ANIMALS

E. COX

Liverpool University Press, 1981 pp. 212 – xix (Appendices) Figs 57 Publ. Price £5.00

This soft-covered volume deals with common surgical conditions of the reproductive tracts of cattle, sheep, horses and pigs, with chapters on castration, cryptorchidism, hernias, the equine and bovine penis, preparation of teaser males, vaginal prolapse, the caesarean operation, trauma to the female reproductive tract and miscellaneous disorders.

The appendices contain comprehensive, well-illustrated descriptions of pudendal and paravertebral nerve blocks in the bovine.

Each chapter includes a review of appropriate anatomy (using NAV nomenclature in English) and investigative techniques. Well-annotated diagrams simplify the text considerably.

For commoner conditions, various surgical alternatives are given and the merits of each discussed. In many cases non-surgical treatment is recommended and discussed where these may be more appropriate.

One shortcoming of the book is the rather too frequent typographical errors and rough corrections. It is, however, an inexpensive volume providing material of interest and value to students and practising veterinarians.

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# ONKOTERAPIE IN HUISDIERE

J.S.J. ODENDAAL* en J.D.E. CRONJE†

ABSTRACT: Odendaal J.S.J.: Cronje J.D.E. **Oncotherapy in Pets.** *Journal of the South African Veterinary Association* (1982) **53** No. 3, 145-150 (Afrik) 152 Benade Drive, Fichardt Park, 9322 Bloemfontein, Republic of South Africa.

A clinical impression is given on a pilot study to determine the application of advanced oncotherapy in private practice. Three case histories are also discussed. In this study an attempt at improved patient care and rendering a better service to the client is described. At the same time experimental data for future reference were collected from which survival periods of patients were calculated.

Key words: oncotherapy, pets, neoplasia.

## INLEIDING

Vanweë verskeie maligne gewasse wat van tyd tot tyd in 'n huisdiepraktyk voorkom en die swak prognose wat met so 'n diagnose gepaard gaan, is daar besluit om 'n loodsprojek aan te pak ten einde vas te stel of gevorderde onkoterapie in privaatpraktyk prakties uitvoerbaar is. So 'n behandeling sou dan die wye spektrum van chirurgie, radio-, hormoon- en chemoterapie insluit.

Wanneer 'n mens onkoterapie beoordeel, is dit belangrik om eers vas te stel wat mens kan verwag van so 'n behandeling. Dit sou nie baat om onrealistiese positiewe resultate te verwag nie en dit is net so belangrik om nie te negatief te staan oor die uitkoms van kanker behandeling nie. 'n Kliniese indruk is soms so subjektief dat die afleidings nie 'n gebalanseerde beoordeling toelaat nie. Daarom bring slegs herhaalde toepassing van terapie oor 'n langer tydperk die waarheid na vore. Die wetenskap is geneig om 'n pendulum-effek te toon, deur eers ver na die positiewe kant en dan na die negatiewe kant te swaai. Uiteindelik kom die pendulum tussen die uiterste pole in ewewig tot stilstand. Met hierdie poging wil ons dus slegs 'n begin maak, probeer om die pendulum effek te vermy en die ware plek van onkoterapie in huisdierpraktyk probeer vasstel.

Die volgende doelstellings het as motivering gedien. Eerstens is die waarde wat die eienaar aan die dier, of sy verlengde lewe heg, in ag geneem. Indien die eienaar dus sou belang stel, is gepoog om 'n beter diens aan die kliënt te lewer. So 'n diens kan voorkom dat diere met alle soorte gewasse dikwels summier 'n genadedood toegedien word, met die idee om die dier lyding te spaar.

Tweedens sou die eksperimentele inligting wat verkry word, gerekenariseer word vir toekomstige behandeling van kankers in huisdiere in privaatpraktyk. Derdens is die projek as 'n nuttige raakpunt tussen veterinêre en mediese dienste gesien, deurdat die privaatveearts sy kliënte 'n verdere diens bied en sy pasiënt help, terwyl die onkoterapeut terselfdertyd inligting verkry, veral vir palliatiewe werk.

Laastens sou daar 'n oorlewingsyfer bereken word om sukses of mislukking ten opsigte van onkoterapie in huisdiere te bepaal. Dit was as belangrik beskou om 'n syfer te bereken wat kan help om naastenby 'n oordeel oor "sukses" te verkry.

Praktiese oorwegings het swaar geweeg toe die projek aangepak is.

(1) Die lewensverwagting van huisdiere is relatief tot die mens kort en daarom kan uitgerekte behandelings nie toegepas word nie.

(b) Aangesien kankers dikwels nie vroegtydig aangebied word vir diagnose en behandeling nie, meesal weens 'n gebrek aan kennis by die eienaar, is die gevorderdheid van sommige kankers as 'n praktiese oorweging gesien. Gevorderdheid van kankers kan die behandelingsmoontlikheid, sowel as die resultate en prognose, beïnvloed.

(c) Om die diere korrek te posisioneer en in so 'n posisie stil te hou vir bestraling, word algemene narkose benodig. Vanweë die redelik gevorderde ouderdom van die pasiënte, kan die herhaalde narkoses as 'n praktiese probleem beskou word.

(d) Om kontrole oor behandeling uit te oefen, moet daar so min as moontlik tuisbehandeling voorgeskryf word.

(e) Aangesien die bestraling slegs by die radioterapie eenheid van die hospitaal gedoen kan word, word die vervoer van die diere na die eenheid sowel as die tyd wat dit in beslag neem, as 'n praktiese oorweging in ag geneem.

## MATERIAAL EN METODE

Die projek bestaan uit die behandeling van katte en honde wat as troeteldiere aangehou is. Slegs gevalle wat vir verdere konsultasie by die Departement Onkologie van die Nasionale Hospitaal, Bloemfontein, aangebied is, is vir die projek aanvaar.

Die eerste 34 gevalle, 27 honde en 7 katte, is as 'n loodsprojek beskou om te sien of die projek enigsins in privaatpraktyk uitvoerbaar is. Die tydsduur van die projek het gestrek oor 2 jaar, naamlik van 1 Januarie 1979 tot 31 Desember 1980. Van hierdie diere was aan die einde van die tydperk nog 14 lewend en 20 dood. Van die 27 honde was 11 nog lewend aan die einde van die projek en 16 was dood. Van die 7 katte was 3 nog lewend en 4 dood.

Die katte is slegs ingedeel as langhaar of korthaar katte, aangesien geen bekende rasse betrokke was nie. Die honde se geslagte was feitlik gelykop gedeel tussen manlik en vroulik. Dertien honde was manlik, waarvan 3 gesteriliseer was en 14 was vroulik, waarvan 5 gesteriliseer was. By die katte was daar net 1 manlik en 6 vroulik, waarvan 3 gesteriliseer was. Die totale verdeling van al die diere was 14 manlik en 20 vroulik (Tabel 1).

Die ouderdom is geneem op die datum wanneer 'n diagnose vir die spesifieke kondisie gemaak is. Vir honde was die gemiddelde ouderdom 7,1 jaar en vir katte 7,9 jaar. Die gemiddelde ouderdom vir alle diere was 7,2 jaar (Tabel 1).

Aanvanklik is nie alle diagnoses histopatologies bevestig nie, maar daar is gou besef dat indien 'n behande-

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TABLE 1: IDENTIFIKASIE

| Spesie | Ras | Geslag | Steriel | Ouderdom (jaar) |
|---|---|---|---|---|
| Honde | Boxer | Vroulik | — | 3 |
| | Boxer | Manlik | — | 4 |
| | Boxer | Manlik | — | 2 |
| | Boxer | Vroulik | Steriel | 7 |
| | Fox Terrier | Manlik | Steriel | 8 |
| | Fox Terrier | Vroulik | Steriel | 15 |
| | Fox Terrier | Manlik | Steriel | 10 |
| | Engelse Bulhond | Vroulik | — | 4 |
| | Engelse Bulhond | Manlik | — | 3 |
| | Engelse Bulhond | Manlik | — | 8 |
| | Doberman | Vroulik | — | 9 |
| | Doberman | Vroulik | Steriel | 4 |
| | Doberman | Vroulik | — | 4 |
| | Spaniel | Manlik | — | 7 |
| | Spaniel | Vroulik | — | 7 |
| | Labrador | Vroulik | Steriel | 8 |
| | Labrador | Vroulik | Steriel | 8 |
| | Basset | Manlik | Steriel | 5 |
| | Basset | Manlik | — | 3 |
| | Schipperke | Manlik | — | 12 |
| | Schipperke | Vroulik | — | 2 |
| | Bull Terrier | Manlik | — | 11 |
| | Bull Terrier | Vroulik | — | 6 |
| | Corgi | Manlik | — | 12 |
| | Duitse Herdershond | Manlik | — | 6 |
| | Franse Poedel | Vroulik | — | 13 |
| | Afghan | Vroulik | — | 10 |
| Katte | Langhaar kat | Vroulik | — | 12 |
| | Langhaar kat | Vroulik | — | 2 |
| | Langhaar kat | Vroulik | — | 12 |
| | Langhaar kat | Vroulik | Steriel | 10 |
| | Langhaar kat | Vroulik | Steriel | 5 |
| | Korthaar kat | Vroulik | Steriel | 6 |
| | Korthaar kat | Manlik | — | 8 |

ling vir 'n spesifieke diagnose beoordeel moet word, so 'n diagnose bo alle twyfel moet staan – klinies en histopatologies.

Uit die besonderhede in Tabel 2 vervat is dit duidelik dat 2 kankers oorheers, naamlik 14 plaveiselkarsinoom en 5 mammakarsinoom. Hierdie 2 gewasse verteenwoordig onderskeidelik 41 % en 26 % van die totaal en gesamentlik maak dit 56 % van al die toestande uit. Indien kondisies soos kroniese ulkusse, hiperkeratose, inflamatoriese reaksies en eosinofieliese granuloom buite rekening gelaat word, omdat hierdie kondisies nie maligne is nie, verteenwoordig plaveiselkarsinoom 48 %, Mammakarsinoom 31 % en gesamentlik 66 % van die totaal.

Gedurende die projek is die volgende behandelingsprofiel gevolg. Ná kliniese ondersoek en diagnose is chirurgie op die gewas uitgevoer indien chirurgie moontlik was. Vanaf die chirurgiese monster of biopsie is 'n histo-patologiese diagnose verkry. Daarna is die kanker bestraal en/of chemoterapie en/of hormone toegedien soos nodig. Indien hierdie benadering weens swakker resultate, praktiese of eksperimentele redes gewysig moet word, sal dit gedoen word. Op die oomblik is die benadering bevredigend en die profiel sal seker nie verander word voordat heelwat meer gevalle behandel is, ten einde 'n duideliker beeld oor spesifieke behandeling vir spesifieke diagnose te vorm nie. Die besonderhede word in Tabel 3 saamgevat.

(i) Chirurgie: Die meeste gewasse is eers chirurgies verwyder om die massa kleiner te maak. Op die wyse kon die dosis bestraling verminder word. Met chirurgie is gepoog om soveel moontlik van die makroskopies aangetaste weefsel te verwyder, en soms is die betrokke limfklier ook verwyder. Die operasiewonde het goed genees, behalwe 1 Doberman se wond van 'n mammakarsinoom wat na 2 maande weer oopgegaan het met hergroei van die gewas. Op 23 gevalle, wat verteenwoordig is deur 21 honde en 2 katte, is chirurgie uitgevoer (Tabel 3).

(ii) Radioterapie: Radioterapie is gewoonlik ná chirurgie toegepas, indien chirurgie moontlik was. In totaal is 27 diere bestraal, waarvan 21 honde en 6 katte was. Radioterapie is onder algemene narkose gedoen en vir praktiese doeleindes is gepoog om die aantal bestralings te beperk. Indien herhalings gedoen is, is dit met tussenposes van 1 week gedoen. Die gemiddelde aantal bestralings per geval was 2,6 (Tabel 3).

Die gemiddelde radioterapeutiese dosis was soos volg: Vir goedaardige gewasse is 'n gemiddeld van 534 rads in enkel fraksies toegedien. Die dosis het gewissel van 500-750 rads. Vir maligne gewasse was die gemiddelde dosis 2 044 rads. Die dosis het gewissel van 500-4 200 rads. Die gemiddelde tyd wat die kursusse geduur het was 21,5 dae.

TABEL 2: DIAGNOSES

| Spesie | Ras | Diagnose |
|---|---|---|
| Honde | Fox Terrier | Plaveiselkarsinoom |
| | Bull Terrier | Plaveiselkarsinoom |
| | Fox Terrier | Plaveiselkarsinoom |
| | Labrador | Plaveiselkarsinoom |
| | Bull Terrier | Plaveiselkarsonoom |
| | Engelse Bulhond | Plaveiselkarsinoom |
| | Basset | Plaveiselkarsinoom. Infiltrerend, gekeratiniseerd7 matig gedifferensieerd. |
| | Fox Terrier | Plaveiselkarsinoom. Swak gedifferensieerd, infiltrerend. |
| | Franse Poedel | Plaveiselkarsinoom. Gekeratiniseerd. infiltrerend. |
| | Spaniel | Mammakarsinoom OW Graad II. Infiltrerend. |
| | Schipperke | Mammakarsinoom. Infiltrerend. |
| | Doberman | Mammakarsinoom. |
| | Afghan | Mammakarsinoom |
| | Corgi | Limfosarkoom, veelvuldig |
| | Spaniel | Limfosarkoom |
| | Engelse Bulhond | Limfosarkoom, veelvuldig |
| | Boxer | Mastositoom |
| | Boxer | Mastositoom |
| | Basset | Kroniese bloeiende neusspieël ulkes |
| | Duitse Herdershond | Kroniese bloeiende neusspieël ulkes |
| | Boxer | Vulvakarsinoom |
| | Labrador | Maligne melanoom |
| | Schipperke | Maligna Schwanoom/Neurofibrosarkoom |
| | Doberman | Rhabdomiosarkoom |
| | Doberman | Chondrosarkoom |
| | Boxer | Kroniese hiperkeratose van die neusspieël |
| | Engelse Bulhond | Hipertrofiese gingivitis met akantose |
| Katte | Korthaar kat | Plaveiselkarsinoom |
| | Korthaar kat | Plaveiselkarsinoom |
| | Langhaar kat | Plaveiselkarsinoom |
| | Langhaar kat | Plaveiselkarsinoom |
| | Langhaar kat | Plaveiselkarsinoom |
| | Langhaar kat | Mammakarsinoom, swak gedifferensieerd. |
| | Langhaar kat | Eosinofieliese granuloom |

TABEL 3: BEHANDELINGSPROFIEL

| Spesie | Ras | Chirurgie | Radio-terapie | Chemo-terapie | Hormone | Newe-effekte en komplikasies |
|---|---|---|---|---|---|---|
| Honde | Doberman | Ja | Ja | Ja | Steroïed | Wond oop na 2 maande + velreaksie |
| | Afghan | Ja | Ja | — | Panhisterektomie + Steroïed | Velreaksie |
| | Corgi | Ja | Ja | Ja | — | Velreaksie |
| | Fox Terrier | Ja | Ja | Ja | — | Velreaksie |
| | Bull Terrier | Ja | Ja | Ja | — | Velreaksie + vomisie. PM – CIN |
| | Fox Terrier | Ja | Ja | Ja | — | Velreaksie + vomisie. PM – CIN |
| | Engelse Bulhond | Ja | Ja | Ja | — | Velreaksie |
| | Basset | Ja | Ja | Ja | — | Velreaksie |
| | Fox Terrier | Ja | Ja | Ja | — | Velreaksie |
| | Bull Terrier | Ja | — | Ja | — | Sekondêre infeksie |
| | Franse Poedel | Ja | — | Ja | — | Epilepsie en blindheid. PM – lewersirrose |
| | Labrador | Ja | — | Ja | Steroïed | Sekondêre infeksie |
| | Labrador | Ja | Ja | — | — | — |
| | Schipperke | Ja | Ja | — | — | — |
| | Spaniël | Ja | Ja | — | — | — |
| | Schipperke | Ja | Ja | — | — | — |
| | Engelse Bulhond | Ja | Ja | — | — | Verstik tuis |
| | Boxer | Ja | Ja | — | — | Velreaksie |
| | Boxer | Ja | Ja | — | — | Velreaksie |
| | Doberman | Ja | — | — | — | Longmetastase (X-strale) |
| | Doberman | Ja | — | — | — | Longmetastase (X-strale) |
| | Boxer | Vloeibare suurstof | Ja | — | Steroïd | Velreaksie + ulkus |
| | Boxer | — | Ja | Ja | Panhisterektomie | Sekondêre infeksie |
| | Basset | — | Ja | — | Steroïed | Sekondêre infeksie |
| | Duitse Herdershond | — | Ja | — | Steroïed | Sekondêre infeksie |
| | Spaniël | — | Ja | — | — | PM – CIN |
| | Engelse Bulhond | — | — | Ja | — | Vomisie |
| Katte | Langhaar kat | Ja | — | — | Panhisterektomie + steroïed + geslagshormone | Longmetastase (PM) |
| | Langhaar kat | Ja | Ja | Ja | Steroïed | Sekondêre infeksie |
| | Langhaar kat | — | Ja | Ja | Steroïed | Sekondêre infeksie |
| | Korthaar kat | — | Ja | Ja | Steroïed | Sekondêre infeksie |
| | Langhaar kat | — | Ja | Ja | Steroïed | Sekondêre infeksie |
| | Langhaar kat | — | Ja | Ja | — | Sekondêre infeksie |
| | Korthaar kat | — | Ja | Ja | — | Sekondêre infeksie |

(iii) Chemoterapie: Onder chemoterapie word vir hierdie doel slegs middels genoem wat 'n sitostatiese effek op gewasse het. Die volgende middels is sistemies aangewend:
Ametopterien (Methotrexate Natrium, Lederlé) 1 mg/kg, subkutaan en intramuskulêr (i.m.)
bleomisiensulfaat (Blenoxane, Bristol) 2 mg/kg, i.m.
5-fluorourasiel (Fluoro-uracil, Roché) 25 mg/kg, intraveneus (i.v.)
siklofosfamied (Endoxan, Noristan) 80 mg/kg, i.v.
doksorubisienhidrochloried (Andriblastina, Chemfarma Laboratories) 0,5-1 mg/kg, i.v.
vincristiensulfaat (Oncovin, Lilly Laboratories) 0,5-1 mg per geval, i.v.
dekarbasien (DTIC-Dome, Miles Laboratories) 5-10 mg/kg i.v.

Laasgenoemde 4 middels is slegs vir kombinasietoedienings (sogenaamde "cocktail") in limfosarkoom gebruik. Eersgenoemde 3 middels is in plaveisel- en mammakarsinoom gebruik. Wat resultate betref, het dit duidelik geblyk dat ametopterien geen kliniese verskil aan plaveiselkarsinoom van honde gemaak het nie. In katte het dieselfde middel baie goeie resultate getoon, soos ook blyk uit die oorlewingsyfers van katte. Die verklaring vir dié verskynsel moet dalk gevind word in die feit dat honde moontlik 'n oormaat het aan die ensiem wat nodig is vir die afbreek van ametopterien, óf die middel abnormaal vinnig uitskei, óf moontlik 'n alternatiewe metaboliese pad volg wat foliensuur in foliniese suur omskep[4]. 19 Diere is met chemoterapie behandel, waarvan 13 honde en 6 katte was (Tabel 3).

(iv) Hormone: Sterilisasie (dit wil sê die verwydering van hormone) is ook as hormoonterapie gereken en is slegs toegepas op diere met mammakarsinoom en vulvakarsinoom – 2 honde en 1 kat.

Verder is gebruik gemaak van testosteroon (Metieltestosteroon, NHP) 1 mg/kg per os en stilboestrol (Stilboestroldipropionaat, May & Baker) 0,5 mg/kg i.m. vir mammakarsinoom. Metielprednisiloon (Depo Medrol, Upjohn) 4 mg/kg i.m. is gebruik vir limfosarkoom, mammakarsinoom, plaveiselkarsinoom, kroniese ulkusse en inflamatoriese reaksies. Die totale aantal diere wat met hor-

mone behandel is, was 12 waarvan 7 honde was en 5 katte (Tabel 3).

## RESULTATE

### Bepaling van relatiewe sukses in terme van oorlewingstyd

Volgens Petrick[5] kan die gemiddelde lewensverwagting van honde en katte bereken word op 12 jaar. Vir mense word die volgende lewensverwagting aanvaar, naamlik vir mans 74 jaar en vroue 79 jaar, dus 'n gemiddeld van 77 jaar[1]. In onkoterapie van mense word die behandelingsukses internasionaal gemeet met 'n 5 jaar[6] oorlewingsyfer vanaf die eerste datum van diagnose.

As 'n mens nou 6,4 mens jare (dit is 77 jaar gedeel deur 12 jaar) neem vir 1 diere jaar, kan die relatiewe suksessyfer van honde en katte in onkoterapie as volg bereken word: 2 336 dae (6,4 jaar) vir mense is 365 dae (1 jaar) vir honde en katte. Dus: 1 825 dae (5 jaar) vir mense is 285 dae vir honde en katte. Met die voorgestelde syfers, glo ons dat die suksessyfer van 285 dae in onkoterapie vir honde en katte 'n realistiese syfer kan wees. *Oor hierdie arbitrêre syfer* kan daar egter gedebateer word.

### Newe-effekte

Alhoewel ons deurentyd bedag was op ongewensde newe-effekte, kan mens oor die algemeen konstateer dat meer newe-effekte verwag is as wat gerapporteer is. Die min erge, onverwagte newe-effekte, was 'n positiewe punt in die hele projek. Dit het dit vir ons makliker gemaak om kliënte te oortuig om 'n dier wel aan die behandeling te onderwerp. Feitlik al die kliënte was vóór behandeling baie bedag oor verwagte newe-effekte. Alle newe-efekte is getrou deur al die kliënte gerapporteer. Die enkele erge newe-effekte by ouer diere ondervind, is as onvermydelik beskou.

Die ergste newe-effekte is ondervind in baie ou diere waar chemoterapie waarskynlik 'n relatiewe oordosering veroorsaak het as gevolg van chroniese nier- of lewerversaking. Slegs 1 Franse Poedel het erge epileptiese aanvalle en blindheid getoon as gevolg van 5-fluorourasiel toediening. Die nekropsie het duidelike lewersirrose getoon en die middel het na ons mening 'n relatiewe oordosering veroorsaak vanweë vertraagde metabolisme. In hierdie besondere geval is die dosis wat in die Franse Poedel gebruik is, gekontroleer in 'n Beagle hond met ongeveer dieselfde gewig. Hierdie toets het getoon dat die spesifieke dosis per gewig nie te hoog was nie. Soortgelyke aanvalle by mense is as raar beskryf[2 3].

Twee ander honde het as gevolg van ametopterien toediening vomisie, anoreksie en lusteloosheid getoon, maar hierdie effekte is ook toegeskryf aan relatiewe oordosering omdat nekropsies getoon het dat die diere aan chroniese interstisiële nefritis gelei het. Die diere wat erge vomisie getoon het, is gou 'n genadedood toegedien.

Verwagte velreaksie as gevolg van radioterapie het voorgekom. Ongelukkig het die diere die letsels oormatig gelek, wat bykomende irritasie van die bestraalde areas tot gevolg gehad het. Hierdie letsels is met steroïed/antibiotika salwe topikaal en soms sistemies behandel.

Sekondêre infeksie weens onder andere beenmurgonderdrukking en laer weerstand, is met antibiotika behandel. Hierdie infeksies is ook as verwagte komplikasies beskou.

### Oorlewingsresultate

Gemiddelde lewensduur van behandeling tot dood. Van die 20 diere wat reeds gesterf het, is slegs 4 vanself dood. Vyftien ander is 'n genadedood toegedien en 1 het tuis verstik weens die eienaar se toedoen. Die gemiddelde lewensduur na diagnosegtot dood, van die pasiënte wat gesterf het, was 203 dae. Vir honde was dit 181 dae en katte 289 dae.

*Gemiddelde lewensduur van behandeling tot einde van projek vir steeds lewende pasiënte.*

Die lewende pasiënte het gemiddeld 336 dae gelewe vanaf datum van diagnose tot 31 Desember 1980. Vir honde was dit 313 dae en katte 415 dae.

*Gemiddelde lewensduur van die totale aantal gevalle vanaf behandeling tot dood of einde van projek.*

Die pasiënte in totaal het 'n gemiddelde lewensduur van 254 dae vanaf behandeling tot 31 Desember 1980 gehad. Vir honde was dit 232 dae en katte 343 dae.

*Relatiewe sukses*

Daar is reeds bereken dat die relatiewe sukses in honde en katte op 285 dae oorlewing vanaf diagnose gestel kan word. Dan toon die gemiddelde oorlewingsyfer van die totale aantal gevalle 'n presentasie van 89,1 %. Vir honde is dit 81,4 % en vir katte 120,4 %.

## BESPREKING

Aangesien die relatiewe suksessyfer in terme van oorlewing vanaf die mens se syfer bereken is, kan genadedood die oorlewingsyfer in diere beïnvloed, want by mense word die oorlewingsyfer tot dood bereken sónder dat die lewe aktief beëindig word. As mens dus die oorlewingsyfer van katte en honde relatief tot die mens se syfer wil beoordeel, behoort hierdie faktor van genadedood in ag geneem te word. Alhoewel so 'n faktor nie 'n dramatiese verskil aan die resultate sou meebring nie, meen ons tog dat die resultate meer positief kon vertoon, aangesien 75 % van die diere wat nie meer lewe nie, 'n genadedood toegedien is. Aan die anderkant is genadedood 'n belangrike aspek om in ag te neem in 'n projek soos hierdie wat in privaatpraktyk uitgevoer is, en waar die kliënt se oordeel en wense ook 'n rol speel.

As mens na die eerste doelwit kyk, dan meen ons dat dit vanaf die pasiënt en kliënt se kant gesien, bemoedigend is. Daar was egter uitstaande gevalle wat groot bevrediging en waardering van die eienaars uitgelok het. Eksperimentele inligting is beslis verkry en selfs met hierdie beperkte ondervinding, kon afleidings van die resultate gemaak word wat toekomstige benaderings kan beïnvloed.

Wat die raakpunt tussen die veterinêre en mediese professies betref, kan ons wel meld dat dit nuttig en van waarde was. Ons het ten spyte van herhaalde narkoses en die relatiewe gevorderde ouderdom van die diere, geen pasiënt verloor weens hierdie feite nie. Die grootste praktiese probleem bly nog om in 'n privaatpraktyk en 'n Onkologie Departement, geskikte tyd te vind vir so 'n projek. Met 'n bietjie opoffering van al die betrokkenes, kon die behandelings egter soos beplan, uitgevoer word.

Indien hierdie werk wyer bekend kan word, kan dit

bydra tot beter begrip en kennis van huisdiergewasse by die eienaars van die diere. So 'n kennis kan weer help om gevalle vroeër te diagnoseer en te behandel. Dit mag weer lei tot beter resultate.

Uit hierdie aantal gevalle is dit moeilik om definitiewe afleidings oor rasse te maak, maar oor die algemeen was rasse van honde en katte betrokke wat pigmentlose dele op die vel het. Hierdie deel ongepigmenteerde diere, ongeag ras of spesie, het 'n groot deel van die totale aantal gevalle uitgemaak.

Die aantal pigmentlose dele vel en die onbeskermende lang blootstelling aan sonlig, dra by tot die hoë voorkoms van plaveiselkarsinoom. As 'n mens hierby in gedagte hou dat diere geneig is om gevoelige dele vel oormatig te lek en so verder te irriteer, word dit duidelik waarom plaveiselkarsinoom so algemeen is. 'n Groter totaal van gevalle mag dalk die beeld van die voorkoms van tipe kankers verander, maar plaveiselkarsinoom sal waarskynlik steeds die algemeenste kanker wees.

Geen afleidings kan gemaak word van geslagsverskille of gesteriliseerde diere ten opsigte van die voorkoms van kankers nie, behalwe mammakarsinoom wat altyd by ouer, ongesteriliseerde vroulike diere voorgekom het. Die meeste van hierdie diere het ook swak of glad nie geteel nie. By katte was die verhouding van vroulik tot manlike diere wel 6 tot 1 maar die totaal is na ons mening te klein om dit as verteenwoordigend te neem. Die gemiddelde ouderdom van 7,2 jaar vir die voorkoms van gewassegby huisdiere, kan 'n realistiese syfer wees. Die bestek van ouderdomme met diagnose, het egter gewissel van 2 jaar tot 15 jaar.

Chirurgie bly 'n wesenlike deel van onkoterapie en dit is binne die bereik van elke veearts. Die feit bly staan, indien mens werklik lewe wil verleng in geval van maligniteite, chirurgie alleen nie altyd voldoende is nie. Daarom is die meeste gevalle ook radioterapeuties behandel. Op die oomblik blyk dit, dat indien daar 'n simpatieke Onkoterapie Departement naby genoeg is, radioterapie prakties uitvoerbaar is. Die bemoedigende resultate toon dan ook dat hierdie poging die moeite werd was. Die samewerking van die kliënte wat baie belangrik is, was ook tot dusver baie goed.

Chemoterapie is makliker om by 'n spreekkamer toe te dien, maar aan die anderkant is sommige van die middels duur en die spesialis onkoterapeut kan nie altyd die vordering effektief monitor soos wanneer bestraling onder sy toesig gedoen word nie. Ons glo ook dat aangesien hierdie middels nie dikwels in privaatpraktyk gebruik word nie, mens eers die nodige kennis moet bekom om chemoterapie vir kankers in privaatpraktyk met vertroue toe te pas. Net die tyd sal leer in hoe 'n mate chemoterapie vir praktiese doeleindes in plaas van radioterapie gebruik kan word. Dit is egter te betwyfel of radioterapie totaal uitgeskakel kan word.

Die effek van hormoonterapie kon nie na behore beoordeel word nie, aangesien die enkele gevalle waarin dit gebruik is, reeds te ver gevorderd was.

Weens die beperkte nadelige newe-effekte wat ons gevind het, kan ons rapporteer dat newe-effekte nie 'n ernstige struikelblok sal wees by oorweging van verdere gevalle nie, behalwe by baie ou diere waar chemoterapie moontlik relatiewe oordosering kan veroorsaak. Lewer- en nierfunksie toetse kan dalk help om beter voorsiening te maak vir moontlik newe-effekte van chemoterapie.

Alle faktore in ag geneem, kan mens oor die algemeen konstateer dat met gevorderde onkoterapie, daar tog 'n beter diens aan kliënte gelewer kan word, sonder om die pasiënte aan onnodige eksperimentele lyding te onderwerp. Sommige gevalle se resultate was verrassend positief terwyl die oorlewingsyfer vir die totale aantal gevalle hoog genoeg is om die projek verder te voer. Hoe meer gevalle met gevorderde onkoterapie behandel kan word, hoe beter beeld kan mens vorm van die werklike sukses en plek van onkoterapie in privaatpraktyk. Op die oomblik lyk die globale resultaat versigtig positief en die behandelings prakties uitvoerbaar.

Om iets van hierdie positiewe resultate te illustreer, word ten slotte 3 gevalle afsonderlik bespreek. Daar is reeds genoem dat katte in die algemeen baie goed op behandeling gereageer het. Net honde wat verteenwoordigend is van die 2 algemeenste kankers, naamlik 2 plaveiselkarsinoom en 1 mammakarsinoom, word dus bespreek.

Geval 7: Hierdie was 'n klein Fox Terrier, skraal gebou, ongeveer 4 kg in gewig. Die pasiënt was ten spyte van haar 15 jarige ouderdom by datum van eerste ondersoek, redelik gesond en aan die gang. Die eienaar was besonder geheg aan die pasiënt. Die regter agter voet het 'n groeisel op 'n pigmentlose deel vel getoon. Die letsel was ulseratief en geirriteerd, sodat die hond dit gedurig gelek het. Die diagnose was plaveiselkarsinoom. Die pasiënt is ten spyte van haar hoë ouderdom 4 keer onder narkose bestraal. Hierbenewens is chirurgie ook toegepas. Hierna het die pasiënt 'n normale lewe gelei totdat sy waarskynlik weens ouderdom dood is. Geen kliniese tekens van kanker was teenwoordig ten tye van dood nie.

Ten spyte van die hoë ouderdom kon die pasiënt se lewe 230 dae verleng word, tot groot vreugde van die eienaar en sonder dat die pasiënt newe-effekte van die behandelings getoon het. Ametopterien is ook toegedien, maar het geen effek op die letsels gehad nie. Indien 285 dae as sukses beskryf kan word, het ons in hierdie besonder ou pasiënt, 'n oorlewingsyfer van 80,7 % behaal.

Geval 20: Hierdie geval was 'n 6 jaar oue ongesteriliseerde teef met 'n mammakarsinoom. Sy was onvrugbaar. Die eerste behandeling was chirurgiese verwydering van 'n gedeelte van die mamma. Twee jaar en 3 maande later het hergroei plaasgevind en 'n panhisterektomie is uitgevoer plus chirurgie van die res van die mamma aan die een kant. 'n Jaar later het die kanker aan die anderkant van die bors verskyn en chirurgie is weer toegepas.

'n Reeks bestralings is toegepas en metielprednisiloon is toegedien. Agt maande na die bestraling is die pasiënt 'n genadedood toegedien weens metastase van die mammakarsinoom. Die lokale letsel was skoon. Die pasiënt was reeds oor die 10 jaar toe genadedood toegedien is en het 'n normale lewe tussen behandelings gelei. Die oorlewingsyfer ná bestraling is 84,2 %.

Geval 22: Hierdie is 'n manlike, 3 jaar ou Basset hond met plaveiselkarsinoom aan die ventrale abdomen op pigmentlose vel. Daar is reeds 3 maal chirurgie toegepas en toe een kursus radioterapie. Die volgende chemoterapie is aangewend: ametopterien met metielprednisiloon, daarna bleomisiensulfaat.

Die pasiënt leef nog en geniet andersins goeie gesondheid. Geen newe-effekte is gerapporteer nie en die kanker lyk op dié stadium onder beheer. Hierdie is een van die gevalle wat die beste resultate getoon het op behandeling van plaveiselkarsinoom. Op die tydstip toe die projek afgesluit is, het die pasiënt reeds 305 dae geleef, wat 'n 107 % oorlewingsyfer aandui. Op hierdie

stadium het ons goeie hoop dat die pasiënt nog 'n geruime tyd kan leef, sonder dat die plaveiselkarsinoom die hond se lewe in gevaar stel. Die eindresultate sal met belangstelling gevolg word. Moontlik het vroeë diagnose in dié geval met die beter resultate vir hierdie spesifieke kanker te doen.


## ERKENNINGS

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# OAK (*QUERCUS RUBOR*) POISONING IN CATTLE

J.A. NESER*, J.A.W. COETZER*, J. BOOMKER** and H. CABLE***

ABSTRACT: Neser J.A.; Coetzer J.A.W.; Boomker J.; Cable H. **Oak (*Quercus rubor*) poisoning in cattle.** *Journal of the South African Veterinary Association* (1982) 53 No. 3, 151-155 (En) Pathology Section, Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa.

Two outbreaks of oak poisoning in cattle in the Republic of South Africa are described. In the first outbreak 22 out of 80 head of cattle were severely affected while 40 out of a herd of 135 cattle were affected in the second outbreak. Of these 40, only one survived after 9 months despite vigorous treatment. Only young cattle under 2 years were affected during both outbreaks in spite of an average herd age of approximately 6 years in the second group of cattle. Clinical signs included severe weakness with a swaying gait, diarrhoea and dehydration. Some were pot bellied while others were emaciated and remained stunted. The most prominent macroscopic and microscopic lesion present in 3 animals autopsied, was a non-suppurative interstitial nephritis which was accompanied by oedema and ulceration of the caecum and colon. Histochemical studies were carried out on pigment granules observed in kidney sections.

Key words: Oak poisoning, *Quercus rubor* poisoning, cattle, kidneys.

## INTRODUCTION

Acorns have been used as a valuable cattle feed without causing harmful effects, especially during shortages of grazing[12]. Oak (*Quercus* spp) poisoning occurs sporadically in parts of the United States of America, but on the ranches of the South Western USA it is a severe economic problem[2 12]. In the latter region of the USA, oak poisoning affects both cattle and sheep during the spring and is caused by ingestion of green leaves and twigs[2 12]. In the North Eastern USA poisoning results from ingestion of acorns during autumn[12].

The toxicity of acorns has been poorly understood since they may be completely harmless on some occasions but highly toxic on others. Certain predisposing factors which include large acorn crops, windstorms and rain appear to be necessary for outbreaks to occur[15]. The toxic principal in oaks has been reported to be tannic acid which is hydrolyzed to gallic acid and pyrogallol in the digestive tract and then absorbed into the blood[4 8]. Tannins have also been isolated in variable amounts from leaves and acorns during outbreaks of poisoning[4 10]. However, the well described symptoms and lesions of oak poisoning were not reported after the oral administration of relatively large amounts of tannic acid to cattle, sheep and goats[8]. Oral administration of tannic acid, gallic acid, pyrogallol and *Q. havardi* blossoms did produce mortalities in rabbits[8] but the histopathological changes in the kidneys of these animals did not correspond to the characteristic changes of oak poisoning.

Identification of oak species is difficult and according to Kingsbury[12] some 60 species occur in the USA and Canada alone. In the Republic of South Africa 16 species of oak are known to the National Herbarium in Pretoria (National Herbarium Pretoria 1981, personal communication). In spite of the large number of oak tree species and their worldwide distribution, symptoms and lesions caused by oak poisoning in cattle and sheep do not appear to vary significantly[12].

Outbreaks of oak poisoning have been reported in cattle, as long ago as 1917 in the USA and 1920 in Great Britain[12]. Outbreaks have been reported in France, New Zealand and Germany[12]. This is the first report describing outbreaks of acorn poisoning in cattle in the Republic of South Africa although sporadic outbreaks have previously been described.

## HISTORY OF OUTBREAKS

Two outbreaks of oak poisoning occurred in 2 different herds of cattle on the Eastern Transvaal Highveld.

The first outbreak occurred during late winter near Ermelo where 22 out of 80 head of cattle were affected. Although the age of the animals in this herd ranged from 1-8 years, only young animals under 2 years of age were affected. Of the 22 affected animals 16 died whilst 6 recovered but remained stunted. The cattle had been kept in a camp with large numbers of oak trees for 7 days. The species was identified as English oak (*Q. rubor*). After about 4 days in the oak pasture, some of the young animals developed a severe diarrhoea. They were subsequently removed to another camp in which no oak trees were present. In the new camp more animals developed diarrhoea (which was at first thought to be due to a worm infestation and treated accordingly), but within a short period they became prostrate and died. After 3 animals had died, one was submitted to the Veterinary Research Institute, Onderstepoort where a tentative diagnosis of oak poisoning was made.

The second outbreak occurred near Belfast during autumn of the following year in a herd of 135 cattle, 1-2 years old, 40 of which were severely affected. Only 1 of the 40 animals was still alive after 9 months despite vigorous treatment. The herd had grazed in a camp in which many oak trees (*Q. rubor*) were growing and during this period it had rained continuously. The first symptoms were noted in the cattle 9 days after they had been removed from the camp. Together with the cattle, a flock of sheep were kept in the same camp, but they were not affected.

During the first outbreak abundant dry leaves and acorns on the ground were accessible to the cattle (Fig. 1 & 2). However, during the second outbreak, the relative amount of acorns to leaves could not be determined exactly. The acorns collected during the first outbreak were brown and completely dried out, while those involved in the other outbreak still had a greenish tinge.

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Fig. 1: Camp with many oak trees where the first outbreak occurred during late winter. Note acorns under trees.

Fig. 2: Abundant dry acorns and leaves on ground.

## CLINICAL SIGNS

During both outbreaks only young animals were affected. Severe diarrhoea with light green faeces and copious amounts of mucus was a characteristic sign in all affected animals. A pronounced weakness characterized by swaying of the hindquarters was seen during walking. Many appeared sunken in the flanks while others also had "pot bellies". Several animals showed marked filling of the jugular veins and they were all anaemic. The body temperature was not elevated in any of the animals.

## MATERIALS AND METHODS

### Clinical pathology

Blood was collected in 10 ml vacuum tubes from one of 2 surviving animals and the serum urea (S-Urea), total serum protein (S-TSP), haematocrit (Ht) and aspartate transaminase (AST) values were determined.

### Macroscopic and microscopic pathology

Autopsies were carried out on 3 animals. A range of tissues were collected in 10 % buffered formalin. The tissues were routinely processed for light microscopy and sections from them were cut and stained with haematoxylin and oesin (HE). Special staining techniques were applied to the kidney sections, such as the Pickworth's benzidene method for haemoglobin, Perl's reaction for haemosiderin, Hall's method for bilirubin, Periodic acid Schiffs (PAS) reaction for mucopolysacharides, Ziehl-Neelsen's (ZN) method for fatty acids of high molecular weight, oil red O method for lipids, Schmorl's technique for lipofuscin, Lillie's method for melanin and Von Giesons technique for collagen[1 6 14].

## RESULTS

### Clinical pathology

Serum urea at 33,4 mmol/ℓ was markedly elevated. The S-TSP value at 57 g/L was low, but the Ht at 0,34 and the AST value at 10 iu were within normal limits.

### Macroscopic pathology

All 3 animals were emaciated and showed a marked subcutaneous oedema especially in the submandibular region and ventral areas of the thorax and abdomen. In addition a severe ascites and hydrothorax were present. Haemorrhages of varying sizes were scattered in the omentum and subcapsularly in the kidneys.

There was a marked perirenal oedema in the 3 animals examined. The kidneys appeared pale and swollen and a pronounced oedema of the renal pelvis was present. Numerous small white foci about 1-2 mm in diameter were scattered throughout the kidney cortices (Fig. 3). In one animal the foci just below the capsule appeared as nodules which protruded beyond the surface of the kidney. Numerous petechial haemorrhages were interspersed between these foci. These small haemorrhages became larger and more numerous toward the medulla.

In all 3 cases the urinary bladder contained brown urine while in one animal the wall was markedly oedematous.

One animal showed focal ulcerative lesions, approximately 10-15 mm in diameter, on the lingual mandibular aspects of the gums, the ventrolateral aspects of the tongue and the pharynx (Fig. 4). The lesions became smaller and more regular in outline toward the oesophageal entrance. Similar but smaller and shallower ulcerations which were round to oval in outline were scattered over most of the oesophageal length (Fig. 5).

Fig. 3: Kidney. Small white foci scattered through cortex.

**Fig. 4:** Ulceration in gum (arrow).

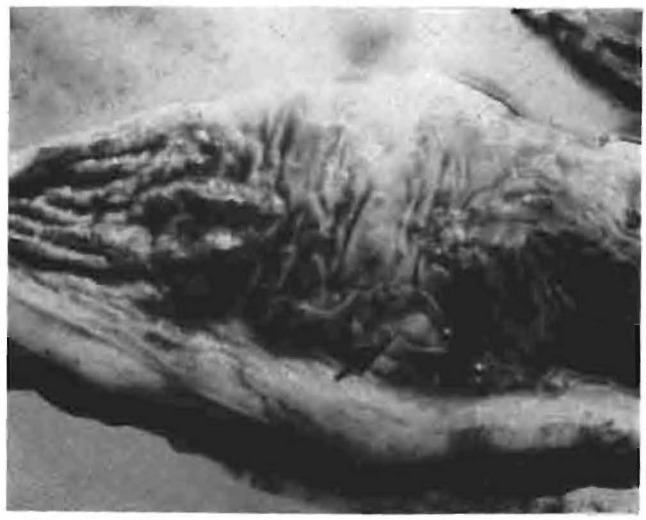

**Fig. 7:** Ulcerations (arrow) in mucosa of colon.

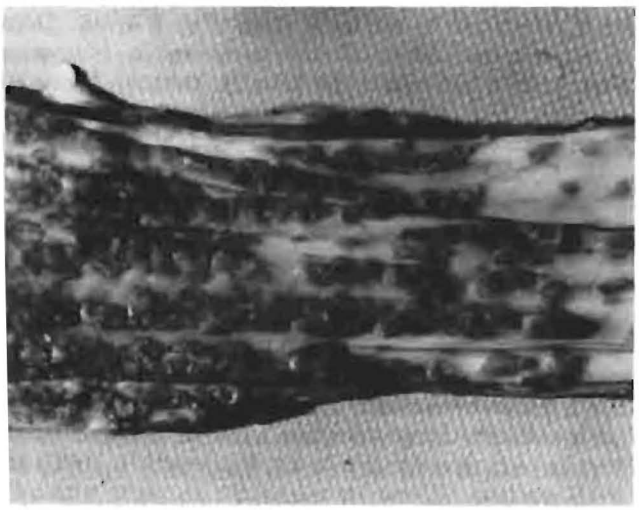

**Fig. 5:** Oesophagus. Numerous round to oval ulcerations in mucosa.

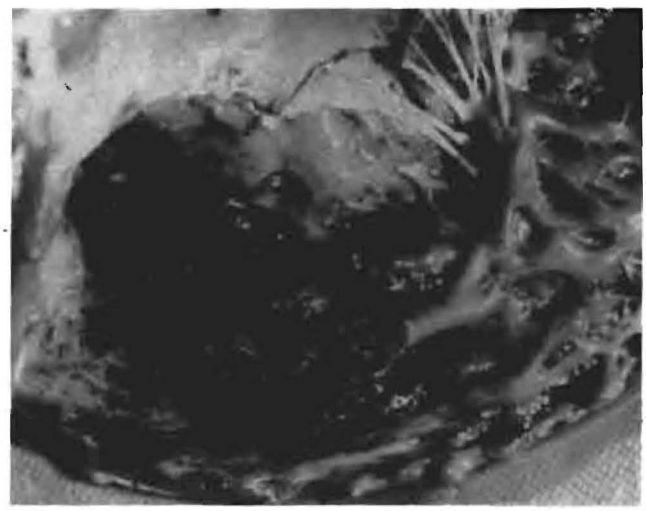

**Fig. 8:** Marked endocardial haemorrhages.

**Fig. 6:** Colon. Desquamated necrotic tissue covered with a blood tinged mucus.

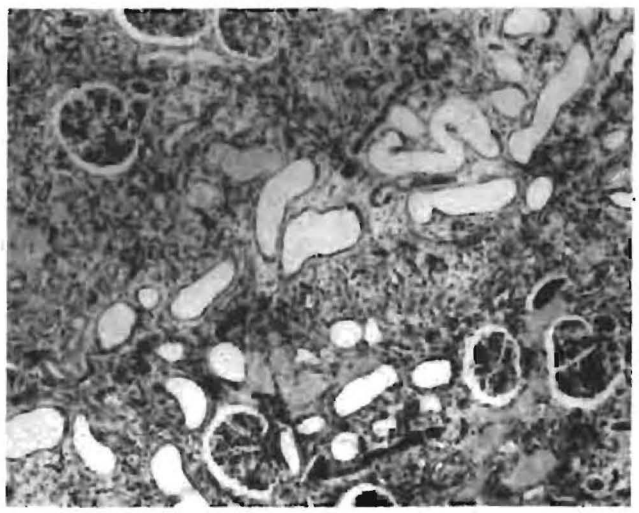

**Fig. 9:** Kidney: Note interstitial fibroplasia, tubular dilatation and necrosis. HE X 53

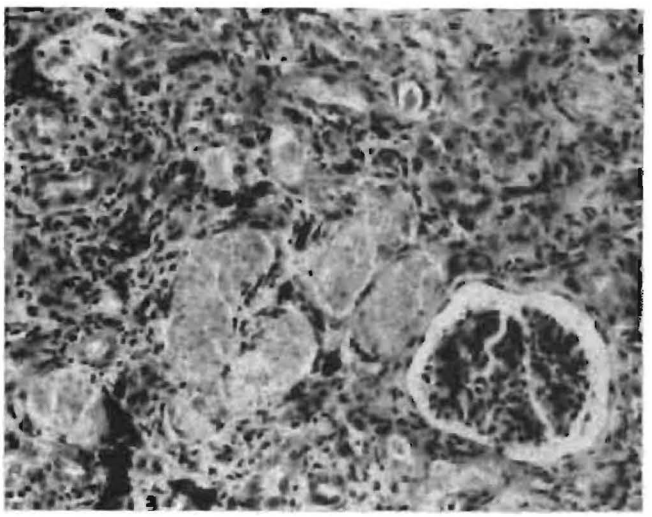

**Fig. 10:** Necrotic tubuli containing abundant pigment granules. HE X 400

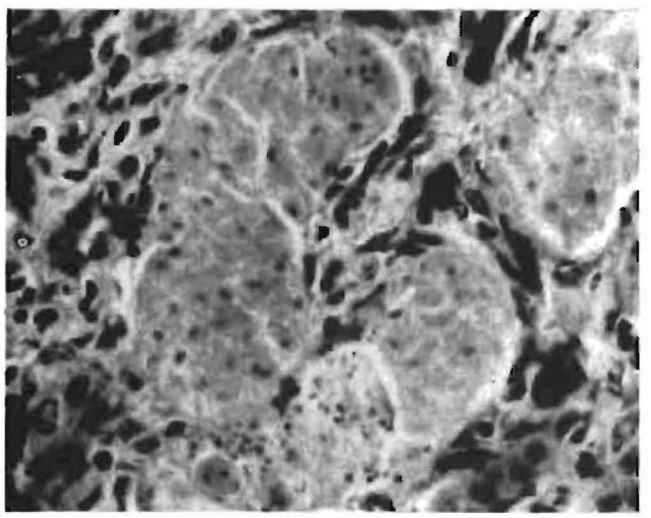

**Fig. 11:** Note pigment granules in necrotic tubular epithelial cells in kidney. HE X 1,000

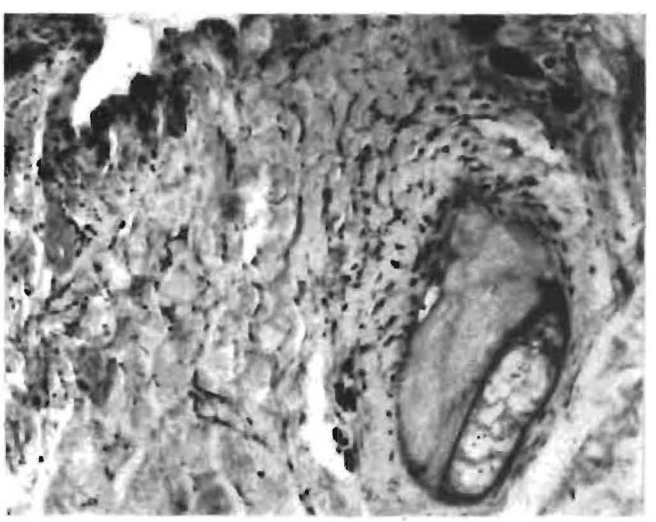

**Fig. 12:** Fibrinoid necrosis and thrombosis of small vessel adjacent to ulcerative lesion on tongue. HE X 160

There was a moderate to severe congestion and oedema of the wall of the rumen, abomasum, small and large intestine, as well as of the mesentery. A catarrhal enteritis and ulcerations were present in the small intestine. The colon of one animal contained a long sausage shaped cast of desquamated necrotic tissue surrounded by blood tinged sticky mucus (Fig. 6). In addition a strong ammonia odour could be sensed when the abomasum and colon was opened. In the other 2 animals the colon and caecum were less severely affected, but ulcerations of the mucosa were present (Fig. 7).

Other lesions included extensive haemorrhages of varying depth and size on the epicardial and endocardial surface of the heart (Fig. 8), mild hepatomegaly and marked distention of the gallbladder was present in one case.

### Microscopic pathology

A subacute to chronic non-suppurative nephritis characterized by interstitial lymphocytic infiltration and connective tissue proliferation were consistently seen (Fig. 9). Groups of tubules throughout the cortex were completely necrotic while others were dilated and showed regeneration of the epithelium (Fig. 9 & 10). Occasional tubules contained many neutrophils. Numerous casts were seen within the tubular lumens. These appeared to be of 2 types; light pink granular eosinophilic casts containing yellowish brown pigment granules (Fig. 10 & 11) and homogeneous pink to bright red casts. The former type were present mostly in the cortex, whilst the latter type were found mostly in the medulla and at the cortico-medullary junction. None of the casts stained positively for haemoglobin or bile pigments with the Pickworth's or Hall's reactions respectively. Both the granular and homogeneous casts were moderately positive for the PAS reaction and weakly positive with the Schmorl's reaction. The brown granules within the casts stained strongly positive with the Schmorl's reaction but negative for the Perl's, Pickworth's, Hall's and PAS reactions. These granules were also seen within the cytoplasm of affected epithelial cells. The Bowman's spaces of most glomeruli were dilated and sometimes filled with a proteinaceous fluid. At the cortico-medullary junction there was a pronounced oedema of the interstitium and around large blood vessels.

Other noteworthy lessions included: ulcerative lessions on the tongue, oesophagus and intestinal tract, characterized by coagulative necrosis accompanied by mononuclear cells and neutrophil infiltration; thrombosis and fibrinoid degeneration and necrosis of small arterioles at the base of necrotic areas in the tongue (Fig. 12); haemorrhage, Zenker's degeneration and necrosis of isolated myocardial fibres in the heart; moderate to severe congestion and oedema of the lungs; cloudy swelling and hydropic degeneration of parenchymal cells in the liver.

## DISCUSSION

Two outbreaks of acorn poisoning are reported from 2 different farms where cattle were allowed to graze in camps containing English oak *(Q. rubor)* trees. During the first outbreak where both young and old animals were present, only young animals less than about 2 years old became affected. Some reports from abroad also state that young cattle are more susceptible[4 5].

The first outbreak occurred toward the end of winter and was due to the ingestion of dry acorns and leaves. During the second outbreak, which occurred in the autumn, acorns and leaves were brown with a greenish tinge. Although mortality rates after both outbreaks were relatively high, little is known about the toxicity of South African oak species. According to Kingsbury[12] all oak species should be regarded as potentially toxic, and futhermore that the toxicity of acorns and leaves does not differ significantly. Several reports also suggest that green leaves and acorns appear to be more toxic[13].

Attempts to produce oak poisoning, by feeding large quantities of oak material at the Veterinary Research Institute, Onderstepoort have failed repeatedly in cattle, sheep and pigs (Kellerman T.S. 1981 Veterinary Research Institute, Onderstepoort, unpublished reports). This has however, been achieved, in other countries in both cattle and sheep by feeding blossoms leaves and acorns[8 10 13]. In the light of our failure to induce poisoning by feeding acorns to cattle, the sporadic nature of natural outbreaks, and the frequency with which acorns are noticeably mould infected, the possibility of a mycotoxin should also be considered in the aetiology. Recently a *Penicillium* sp. was isolated from toxic acorns and when fed to mice produced kidney lessions (Jerrett I.V. 1982 Department of Agriculture, P.O. Box 483 Victoria, Australia, personal communications).

The clinical signs, macroscopic and microscopic lesions closely corresponded to those described in the USA, Europe and New Zealand in cattle that succumb to oak poisoning[2 12 13]. Emaciation and dehydration in the animals reported here were probably the result of the renal damage, uraemia, severe enteritis and prolonged diarrhoea. It is unlikely that these ulcerative lesions are due to the noxious effects of the oak toxins alone. They are more likely the result of the combined effect of the toxins and uraemia. These lessions correspond to those described in the digestive tract of uraemic cattle[11].

Various histochemical methods were applied to study the nature of the casts and yellowish brown pigment granules. The casts gave strong positive results with the PAS reaction indicating their mucoprotein composition. The pigment granules gave a negative reaction with the PAS stain, but were strongly positive with the Schmorl's technique and moderately positive with the Ziehl-Neelsen reaction indicating reducing properties characeristic for lipofuscin[6 14]. Negative fluorescence under ultraviolet light distinguished the pigment from ceroid which is considered to be a precursor compound of lipofuscin[6 14].

Whereas oak poisoning in cattle and sheep has become known as a distinct clinicopathological entity due to its characteristic renal lessions, there is still some doubt concerning the nature of this condition in the horse and pig[7 12]. Mortality rates due to oak poisoning in horses can also be high but the characteristic renal lession have not been described[3 7 12]. Swine appear to be resistant to oak poisoning and usually consume acorns without any adverse effects[12].

Treatment of oak poisoning is at this stage only symptomatic and of questionable ultimate value as seen from the cases treated in these 2 outbreaks. No reports concerning the successful treatment of oak poisoning could be found[13].

Poisoning in oak pastsures can be effectively prevented by providing an ad lib. quantity of a well balanced supplementary feed to which calcium hydroxide ($Ca(OH)_2$) has been added at the rate of 15% by weight[9]. Occasionally this level of $Ca(OH)_2$ in the feed will discourage calves to partake of the lick. In such cases the level should be reduced to no less than 10%[9]. As oak trees frequently provide valuable overwintering fodder to cattle and sheep on the Eastern Transvaal Highveld, one can consider feeding oak leaves or acorns as a supplimentary feed if $Ca(OH)_2$ is added to the supplement at the prescribed ratio.

## ACNOWLEDGEMENTS

The authors wish to express their appreciation to the staff of the Sections of Pathology and Photography for preparing the histopathological sections and photographs, respectively. We also wish to thank Mrs. R. Coetzer for typing the manuscript.

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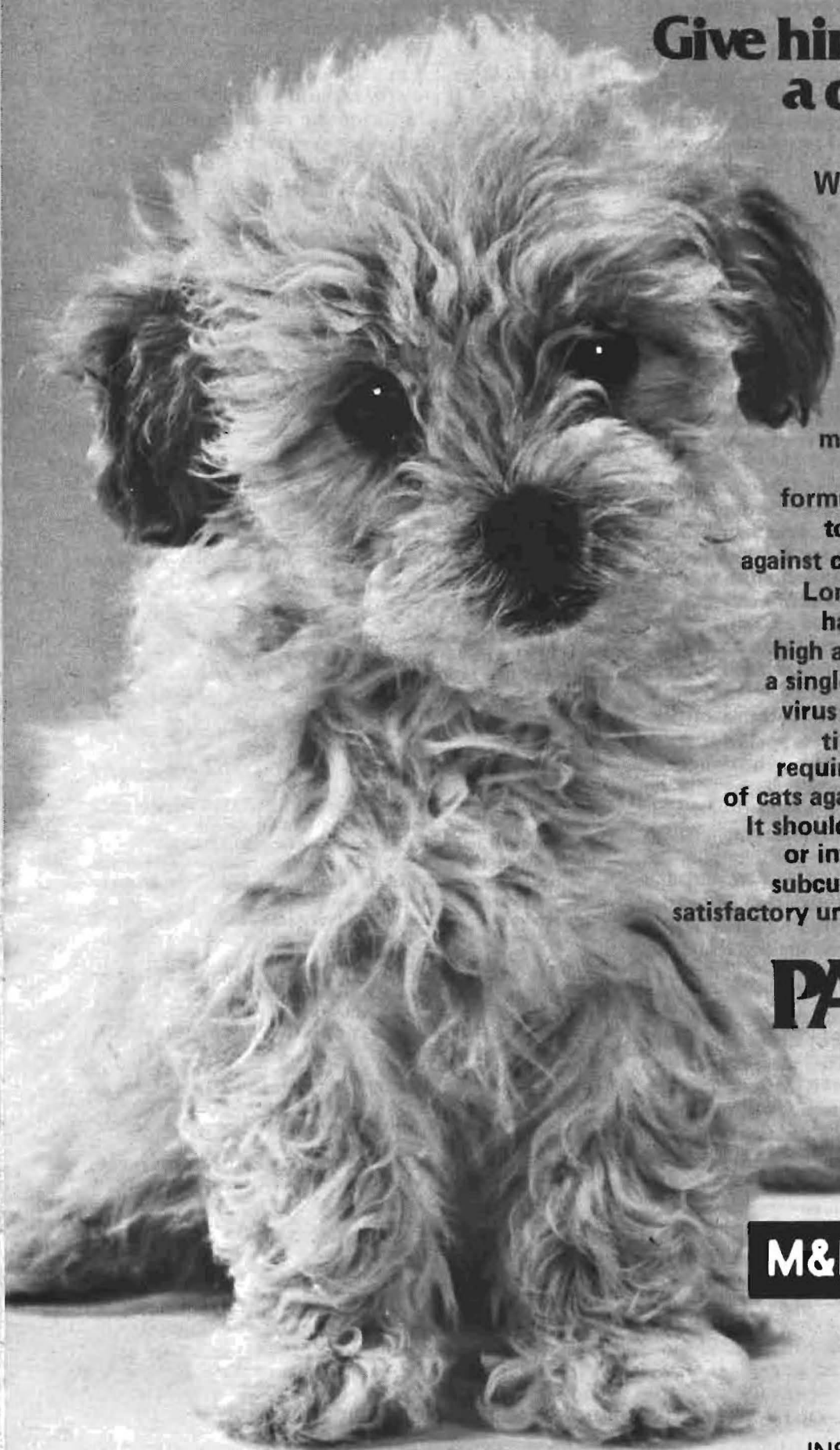

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# PATHOLOGY OF THE BOVINE UDDER PARENCHYMA CAUSED BY ASPOROGENOUS OBLIGATE ANAEROBIC BACTERIA ISOLATED FROM CASES OF BOVINE MASTITIS

J.H. DU PREEZ*, A.S. GREEFF** and W.S. BOTHA*

ABSTRACT: Du Preez J.H.; Greeff A.S.; Botha W.S. **Pathology of the bovine udder parenchyma caused by asporogenous obligate anaerobic bacteria isolated from cases of bovine mastitis.** *Journal of the South African Veterinary Association* (1982) 53 No. 3, 157-159 (En) Department of Pathology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

The gross and microscopic pathology of the udders of lactating cows after experimental infection with pure cultures of *Propionibacterium granulosum*, *Peptococcus indolicus*, *Bacteroides fragilis*, *Eubacterium aerofaciens* and *E. lentum* was studied. Evidence is presented that these asporogenous obligate anaerobic bacteria are capable pathogens of the bovine udder.

Key words: Anaerobic bacteria, bovine mastitis, histopathology, udder parenchyma.

## INTRODUCTION

In the past few years a number of reports have incriminated a variety of obligate anaerobic bacteria in the aetiology of bovine mastitis. Early reports[11 13 14] refer to the consistent isolation of *Peptococcus indolicus* together with *Corynebacterium pyogenes* from cases of summer mastitis and from the udders of healthy cattle[12]. Japanese workers[9] have isolated obligate anaerobic Peptococcaceae, *Bacteroides* spp. and *Fusobacterium necrophorum* from outbreaks of mastitis as well as from the healthy udders of non-lactating heifers.

In recent investigations of the milk and/or udders of South African herds *Bacteroides fragilis*, *Bacteroides eggerthii*, *P. indolicus*, *Eubacterium lentum*, *Eubacterium aerofaciens*, *Eubacterium combesii*, *Propionibacterium granulosum*, *Propionibacterium acnes*, *F. necroforum*, *Clostridium sporogenes* and an anaerobic *Streptococcus* spp. have been isolated from sporadic cases of bovine mastitis[2 3 5].

Very few studies dealing with the possible udder pathogenicity caused by asporogenous obligate anaerobic bacteria were encountered in the literature. Mastitis has been experimentally induced in healthy non-lactating heifers by introducing mixed cultures of *P. indolicus* and *C. pyogenes*[10 13] into the udder through the teat canal and has also been induced in healthy lactating udders with pure cultures of *B. fragilis*, *E. aerofaciens*, *E. lentum*, *P. indolicus*, *P. granulosum* and an anaerobic *Streptococcus* spp.[2 3 5].

Histological evidence is now presented which incriminates various asporogenous anaerobic species as possible causative agents of udder parenchyma pathology.

## MATERIALS AND METHODS

### Bacteriology

The methods described by Du Preez et al.[3] for the cultivation of strict anaerobic bacteria and the experimental infection of healthy lactating udders with pure cultures of 2 strains of *P. granulosum*, one strain of *P. indolicus*, one strain of *B. fragilis* and 2 strains of *E. lentum* were used in this study. The clinical and cytobacteriological criteria used to establish the existence of mastitis were those of the International Dairy Federation[8].

### Pathology

The cows were slaughtered after the establishment of clinical mastitis[2 3], the udders and the supramammary lymph nodes were incised, examined and specimens for histopathological examination were fixed in 10 % formalin from infected and uninfected quarters of all the cows. Tissue blocks from the latter were routinely processed and sections were cut and stained for light microscopy. Haematoxylin and eosin stains were used on all sections, while Gram stain was applied to selected sections.

## RESULTS

### Gross pathology

All the anaerobic bacterial species and strains tested caused acute purulent mastitis within 24 h. This was evident from examination by the strip cup of the secretions from all infected quarters and from the presence of pus in the teat and udder cisterns and the lactiferous ducts. All supramammary lymph nodes were oedematous and swollen.

### Histopathology

Control quarters showed normal udder parenchyma (Fig. 1). No inflammatory cells were present and the alveolar epithelium was intact. In contrast all the bacterial strains tested caused pathological changes of the udder parenchyma.

Experimental udder infection with *P. granulosum* resulted in the presence of high numbers of neutrophils in the lumina of alveoli (Fig. 2 & 3). Some alveolar epithelial cells showed degenerative changes. Mild fibrosis of the interstitial tissue with mild round cell infiltration was evident.

*P. indolicus* infection caused alveoli to be filled with exudate consisting mainly of polymorphonuclear leukocytes (Fig. 4). Lymphocytes were also present in the alveoli. A number of round cells (lymphocytes, plasma cells and a few neutrophils) were found in the interstitial tissue.

Clear signs of an acute purulent mastitis followed infection with *B. fragilis* (Fig. 5). Large numbers of polymorphonuclear leukocytes were present in the alveoli and smaller lactiferous ducts. Neutrophils, some

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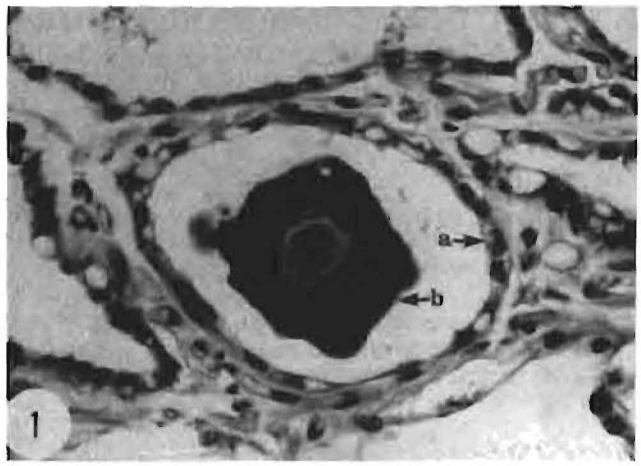


**Fig. 1.** Control quarter. Normal alveolar epithelium (a) with corpora amylacea (b) HE X 400

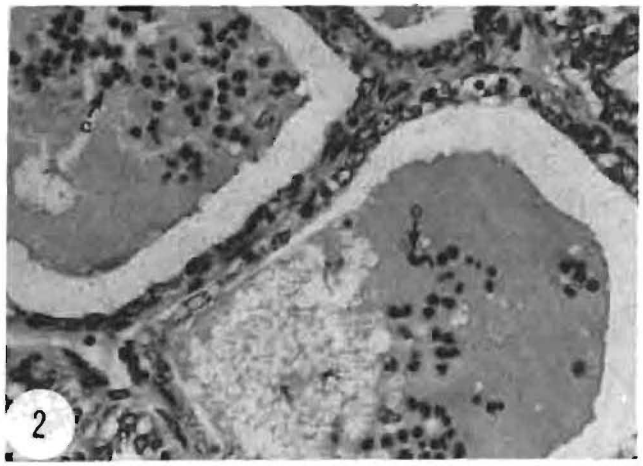


**Fig. 2.** Udder parenchyma after experimental induction of mastitis with *P. granulosaum* Neutriphils (C) in alveoli. HE X 400

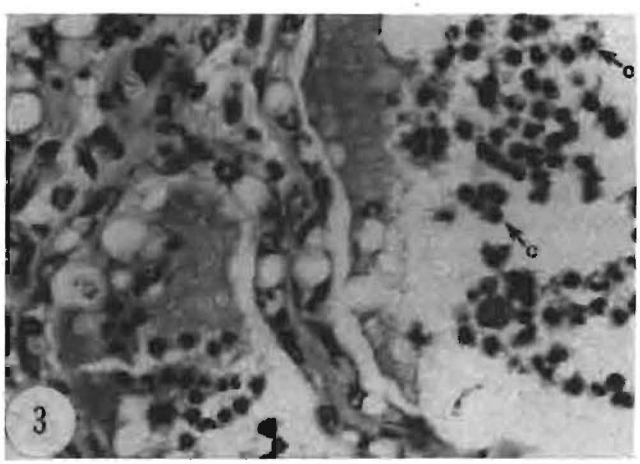


**Fig. 3.** The same as Fig. 2. Neutrophils (C) in alveolus. HE X 400

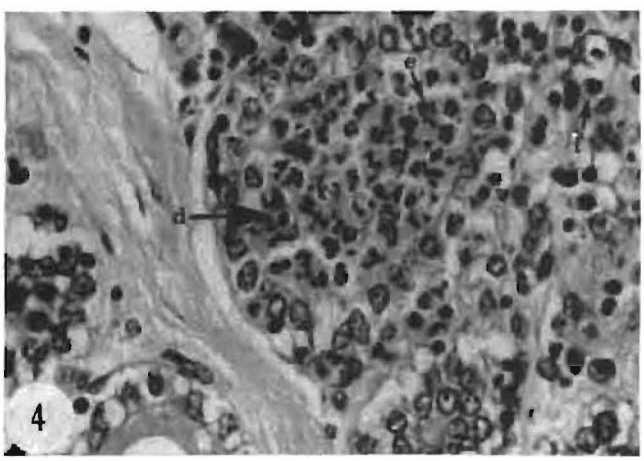


**Fig. 4.** Udder parenchyma after experimental induction of mastitis with *P. Indolicus*. Alveolus is filled with exudate (d). The exudate contains mainly polymorphonuclear (e) and lymphocytes (f). HE X 400

of which appeared pycnotic, dominated while some macrophages were also present. Marcophages contained phagocytosed material which gave a vacuolarised appearance to their cytoplasm. Some eosinophils and plasma cells were found in the interstitial tissue.

Few inflammatory cells were seen in sections from udder parenchyma after infection with *E. aerofaciens*. However, diffuse interstitial round cell reactions were present. The large lactiferous ducts contained large quantities of exfoliated epithelial cells, some neutrophils and many Gram positive bacilli (Fig. 6).

*E. lentum* caused accumulation of many polymorphonuclear leukocytes and fibrin in the alveoli and duct system (Fig. 7 & 8). Vacuolization of the alveolar epithelium was prominent and neutrophils could be demonstrated to be migrating through the alveolar walls. Fibrinopurulent exudate was present in the lactiferous ducts. Some alveoli contained many macrophages with a foamy cytoplasm. Plasma cells were widely distributed in the interstitial tissue udder parenchyma but can also occurred singly in the alveolar walls. Numerous lymphocytes were present in the interstitial tissue.

## DISCUSSION

Little is known about the pathogenic propensities of obligate anaerobic bacteria. A few studies[3 10 13] suggest that they may act as primary pathogens because they are capable of inducing clinical mastitis under experimental conditions. Histopathological evidence, however, is scant.

It was recently found[6] that tissue cultures of human fallopian tubes, experimentally infected with *B. fragilis*, underwent rapid loss of cilial activity as well as tissue destruction.

In the present study we found that all our strains are capable of rapidly (within 24 h) inducing an inflammatory reaction in the udder parenchyma and some of the results suggest that tissue damage also occurred.

Although it is evident that a variety of non-sporulating anaerobic bacteria possess the capability of acting as pathogens, they are almost always isolated together with organisms classically involved in bovine mastitis. This may be explained in terms of the oxygen requirements of the obligate anaerobic bacteria. Since they will grow best under conditions of low oxidation-reduction (redox) potential[7], they often act as secondary invaders after low redox potentials have been created by oxygen consumption and tissue necrosis caused by aerobic micro-aerophilic or facultative organisms[1 4].

Because many anaerobic bacterial species, notably *B. fragilis*, are naturally resistant to penicillins, cephalosporins, tetracycline and aminoglycoside an-

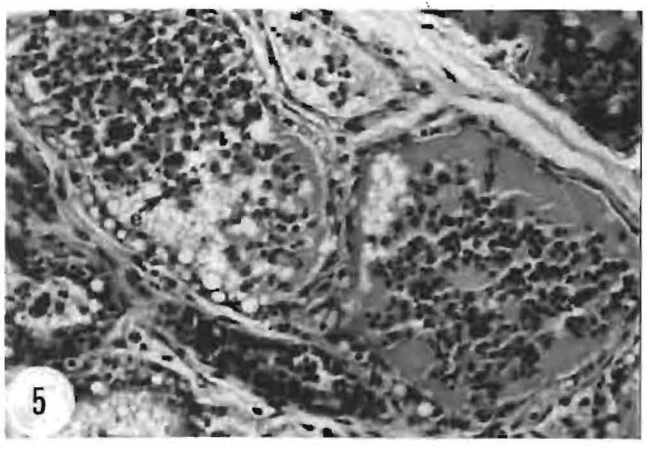


**Fig. 5.** Udder parenchyma after experimental induction of mastitis with B. fragilis. Polymorphonuclear leucocytes (e) in alveolus. HE X 400

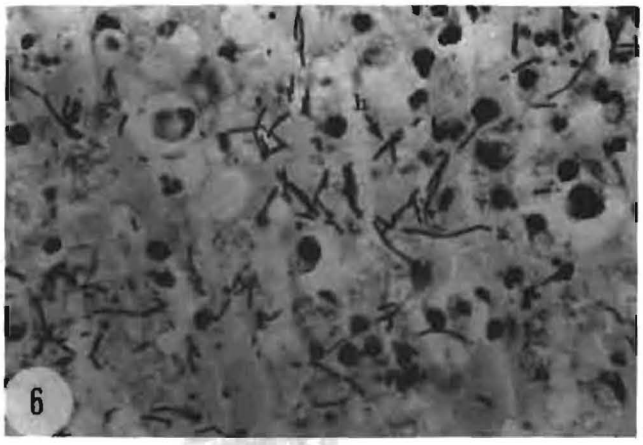


**Fig. 6.** Lactiferous duct after experimental induction of mastitis with *E. aerofaciens*. Desquamated epithelial cells (g) and thin Gram positive bacilliform bacteria (h) Gram X 1000

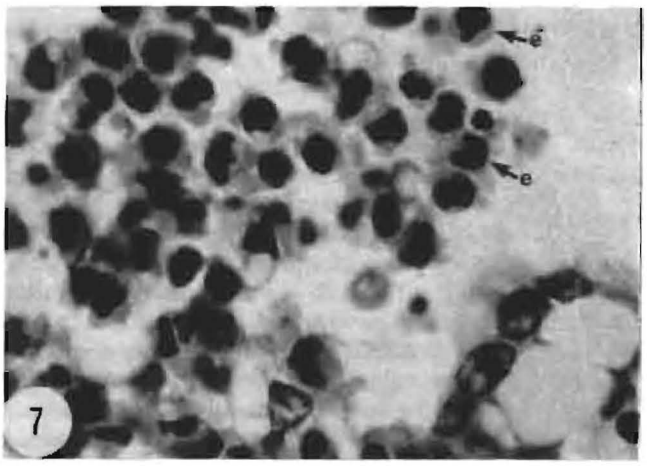


**Fig. 7.** Alveolus with polymorphonuclear leucocytes (e) after experimental induction of mastitis with *E. lentum*. HE X 1000

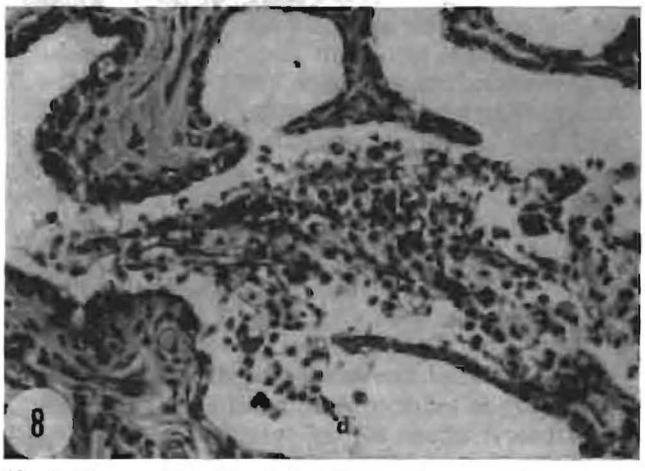


**Fig. 8.** The same as Fig. 7. Lactiferous duct with fibrinopurulent exudate (d) HE X 200

tibiotics, they often survive antibiotic therapy[15]. It thus seems evident from the results of the present study that they are quite capable of causing considerable damage to the bovine udder parenchyma in the absence of other organisms.

ACKNOWLEDGEMENTS

The authors wish to thank Prof L.W. Van Den Heever for valuable discussions.

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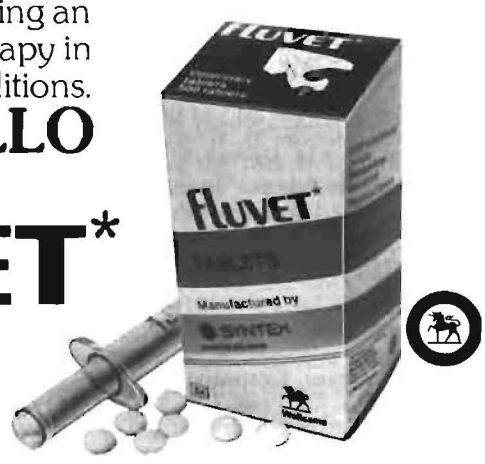


SYNTEX

Coopers Veterinary Speciality Department
68 Rigger Road, Spartan, Kempton Park 1620. Phone 975-1146

# KIKUYU POISONING AND THE ARMY WORM

R.W. BRYSON*

ABSTRACT: Bryson R.W. **Kikuyu poisoning and the army worm.** *Journal of the South African Veterinary Association* (1982) **53** No. 3 161-165 (En) Regional Veterinary Laboratory, Allerton, Private Bag X 9005, 3200 Pietermaritzburg, Republic of South Africa.

Kikuyu poisoning occurred during 1981 in widely separated coastal areas of South Africa. Although the causal agent has not been identified, precautions can be taken to contain mortality. Existing knowledge of the disease is documented.

Key words: *Pennisetum clandestinum*, grass, kikuyu poisoning, *Spodoptera exempta*, army worm.

## HISTORY AND BACKGROUND

The condition was first recorded in 1969 in New Zealand[3] where it has continued to cause sporadic losses to the present day. It has always been associated with kikuyu grass damaged by army worm (*Spodoptera exempta*) and for this reason New Zealand workers named the condition "Kikuyu Poisoning"[9 10 12]. Kikuyu grass (*Pennisetum clandestinum*) was first introduced into New Zealand from Rhodesia in 1920 and has encroached rapidly since, being the dominant grass of many farms in areas of Northland and is still spreading[10].

As far as is known, sporadic outbreaks occurred in South Africa and Rhodesia during the 1960's with the cause of the condition being obscure and at that time not correlated with the New Zealand syndrome. In Natal sporadic ourbreaks appear to occur every 3-4 years, coinciding with the invasion of kikuyu pastures by army worm.

T.S. Kellerman (1977 Veterinary Research Institute, Onderstepoort, personal communication) and Hodkin (1981 State Veterinarian, East London, personal communication) have noted the condition in Zimbabwe, but during 1960 kikuyu grass was only grown on a small scale as a pasture grass chiefly where irrigation was possible. Army worm invasions were fairly common withour reports of toxicity. The condition is thought to occur in Kenya and surrounding territories where kikuyu grass is common and the army worm actually breeds. It is likely, depending on the rainfall pattern, that the invasion occurs regularly. Information on this is being sought from Kenya. The invasion in South Africa appears to affect coastal areas, Eastern Transvaal, Eastern Cape and Natal, and has been recorded in the Rustenburg district of the Western Transvaal.

Some degree of breeding of the worm occurs every year in South Africa and Ardington (1981 Private Practitioner, Mandini, personal communication) has noted army worms present in the Mandini area in 7 seasons out of 8. The potential for a local build-up exists but Bell[1] states that the following factors are required:

1. Optimal temperature, and
2. Availability of young succulent grass.

The caterpillars feed only on grass, and seem to prefer sweet grasses belonging to the genus *Cynoden* but are also partial to kikuyu and *Eragrostis curvula*, but when mature will attack almost any kind of grass, even maize plants and sugar cane.

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## RECENT OUTBREAKS

Regular outbreaks occur in New Zealand[6 10 12]; there were 32 outbreaks in Northland farms in 1970-72, 2 in 1973 and one in 1976. There was then a lull during which biological control of the worm appeared to be succeeding[6] followed by 2 disastrous outbreaks in mid 1979[7]. In the latter at Whangarei in a herd of Hereford steers, 60 were clinically affected, of which 17 died. In a second outbreak, 60 out of 150 Angus steers and heifers died.

In South Africa 3 known outbreaks occurred in 1977: in Natal, 7 cows died out of 25 exposed[2]; in Eastern Cape, 44 Friesland heifers died in a herd of 97[5]; in Western Transvaal, 17 animals were affected of which 14 died[14].

During 1981 7 outbreaks were recorded in which 157 animals died. This is the greatest number of outbreaks and the highest number of known deaths recorded in South Africa from this condition, although it is known that sporadic undocumented mortality has been mentioned since the 1960's. It appears that the increased incidence of the condition has coincided with the increasing use of kikuyu as a pasture grass, as has occurred in New Zealand, or it may be that there has been increased public awareness of the condition because of warnings issued by the Department of Agriculture and Fisheries. These warnings were issued as soon as it was realised that the 1981 army worm invasion was on a massive scale, and it appears probable that where these warnings were heeded, mortality was prevented on many farms.

## PREDISPOSING FACTORS

There can be few diseases where factors are required to appear at the right time to produce the condition known as kikuyu poisoning. These can be listed in order of importance:

1. Cattle grazing on kikuyu pasture which has been damaged by army worm some 10 days or longer previously.
2. Small paddock usually not exceeding 1-2 ha in extent and normally heavily stocked.
3. A period of drought followed by patchy rainfall and a humid atmosphere; often there has been a fall of 18-20 mm of rain within 5 days of toxicity appearing. Optimal minimum temperatures range from 10-14°C, usually 14°C.
4. Paddocks often on a latitude of approximately 02905S, sloping gently and protected on one side by a


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line of mature trees. These can also occur in die middle of the paddock.

5. Cattle not kept under constant observation as the "incubation" period can be up to 96 hours. If toxicity is observed, affected cattle must be removed from the camp at once and will normally recover spontaneously. It was noticed in the majority of the Natal outbreaks that heavily pregnant dairy cows not in milk were affected, as were a group of down calving heifers. It may, of course, be that diet variations are a factor in these animals, but it appears unlikely that pregnant animals are more susceptible. Tracer steers were affected during trials at Cedara, Natal and morality in New Zealand during 1979 involved steers and heifers. In South Africa it is common to allow dairy cattle to calve from May-July in order to obtain a higher winter milk price. Thus many dairy animals are heavily pregnant at this time.

Fertilisation of kikuyu pastures was standard practice in all the affected farms, as kikuyu is known to respond well to nitrogen supplementation. Excessive nitrogen fertilisation, giving rise to toxic concentration of nitrate/nitrite; cyanogenetic glycosides and/or oxalates, was postulated by Steyn[13] as a possible factor in kikuyu poisoning, but analyses for these by Mapham[5] proved negative during an outbreak of kikuyu poisoning in the Eastern Cape. It does seem probable that poisoning on kikuyu due to these substances can occur, and produces symptoms similar in many ways to kikuyu poisoning. One farmer in the King Williams Town district, Eastern Cape who was grazing cattle on kikuyu irrigated by effluent from a tannery reported signs similar to kikuyu poisoning, although no army worm was present.

In Western Australia in 1973, in a herd of 38 steers grazing on luxuriant undamaged kikuyu, 7 died and many were affeccted with kikuyu poisoning-like signs. In a further outbreak, 3 heavily pregnant heifers died out of 16 affected. Lesions in the dead animals resembled those found in kikuyu poisoning. The New Zealand army worm (*Pseudaletia separata*) is not known to exist in this area of Australia nor are many other major insect pests known to affect kikuyu there[4].

## BREED AND SPECIES SUSCEPTIBILITY

Mortality has been recorded in *Bos taurus* breeds, in particular in the Friesland, Hereford, Sussex and Aberdeen Angus. Of the *Bos indicus* breeds, Brahmans have been present on farms where poisoning occurred and have grazed on damaged pastures with impunity, but these pastures were not necessarily toxic[14]. Sheep have never been affected in South African outbreaks where the caterpillar was *S. exempta*, but Steyn states that the commando worm (*Laphygyma exempta*) has produced toxicity on grazing affecting sheep and cattle[13]. This is thought to be a similar but separate condition. In New Zealand, Martinovich & Smith produced the condition in sheep on pastures known to have affected cattle[9]. Previously sheep grazing on affected pastures had been unaffected. This was the case in South Africa in 1977 where kikuyu poisoning was confirmed by Bryson & Newsholme[2]. During the 1981 outbreak farmers were advised to utilise dangerous pastures by grazing sheep after using "tracer" sheep for 96 hours. No mortality occurred.

Horses have been grazed on affected pastures normally without any deleterious effects but Mapham has observed mild colic in one horse[7] and Jacobs states that 2 horses in a toxic paddock were affected with nervous symptoms and one died in convulsions (Jacobs E. 1981, personal communication).

## THE ARMY WORM AND ITS LIFE CYCLE

The following information is provided by Bell[1]. It is thought that the moth (*S. exempta*) regularly breeds in territories such as Kenya, Tanzania and Sudan, and that migration to other territories occurs spasmodically. It is known, however, as stated above, that in certain warm coastal areas of Natal breeding regularly occurs.

The eggs are laid on succulent grass and hatch in 3 days, the larvae climbing to the top of the grass to feed. There are 6 instars and the larvae require a temperature of 24°-32°C to develop. They are fully mature after 3 weeks and then enter the soil to pupate about 40 mm below the surface in a fragile coccoon. The pupal stage lasts 3 weeks and the total life cycle takes about 45 days.

Control is best carried out when the larvae are newly hatched as each instar becomes more resistant to poisons. Controlling fully mature pests is a waste of money because the damage is already done and development beyond the pupal stage is unlikely due to frost and parasites. In New Zealand a wasp (*Apanteles ruficrus*) which parasitises the larvae was introduced from Pakistan in 1971 and appeared to reduce the army worm population to some extent, although kikuyu poisoning continued to occur[6 7].

Wild birds and particularly egrets feed greedily on the worm with impunity although mortality in turkeys was noted by Nixon where crops were found to be packed with army worms (R.C. Nixon 1981 Allerton Laboratory, Pietermaritzburg, personal communication). In New Zealand 500 g of dead army worms were fed to cattle without effect[7]. In most outbreaks faecal material in large quantities is found on grass stalks and dead worms occur at the base of the sward. In the recent Eastern Cape outbreak, Kellerman & Newsholme had difficulty finding dead worms at the height of the toxicity (T.S. Kellerman 1981 Veterinary Research Institute, Onderstepoort, personal communication). For any control measure to be effective, it would require to take place in the breeding grounds with a similar co-operative venture to the red locust control scheme. Political consideration may inhibit this in Southern Africa. Only biological control methods have been used in New Zealand.

## THE DISEASE

As the cause is not known, no definitive name can really be given to this condition. The main effects appear to be on the nervous and digestive systems.

A regular feature has been an "incubation" or "prepatent" period within which potentially toxic pastures appear safe for grazing animals. At one stage recommendations were given to rest the damaged pastures for at least 10 days before resumption of grazing. In fact, this is the worst possible precaution, as it is during this time that the pastures become "toxic". On some farms where grazing was not stopped at all, no mortality was noted and a postulation could be made that the toxic factor is disturbed and inhibited during this period, and does not develop subsequently, but this is pure conjecture. Lightly damaged pastures appear to be as dangerous as those heavily damaged[10 12]. Cutting

of luxuriant growth to expose the roots has had no effect. In our first outbreak during 1981 the owner had severely mowed the grass and left the cut grass to dry on top of the stubble. This may have even produced the toxic agent. In most cases farmers avoided using the grazing for the minimum of 42 days as is recommended by the Regional Veterinary Laboratory at Allerton, although this period was much less in many cases. Other farmers made and stacked hay.

The first indication of the disease is usually the finding of dead animals and the presence of many other very ill cattle. Under experimental conditions where free-choice grazing on toxic kikuyu was allowed, steers were affected within 96 hours after having been placed on the pasture[12].

Mapham starved one heifer for 24 hours after which toxic material was consumed. Clinical signs appeared within 24 hours and the animal was destroyed in extremis 5 days later. Another heifer was not starved but was offered only fresh kikuyu. It showed a similar syndrome[5]. Under natural conditions where maximum pressure is put on grazing in small paddocks, clinical signs have appeared within 24 hours.

The occurrence and severity of the disease appear to depend on the degree of toxicity in the paddock and the concentration of cattle, but it does seem that, if signs are noted early enough, mortality need not occur if the cattle are immediately moved from the toxic pasture.

Drooling of large strings of watery saliva from the mouth and an apparent severe thirst are the earliest clinical signs. The animals stand over the water troughs with the lips dipped into the water but they do not actually swallow ("sham-drinking"); there is some ruminal tympany with very marked colicky pains, the animals grinding its teeth, kicking and looking at the flank. Cyanosis is present and conjunctivae are injected. The expression is anxious. Ruminal and intestinal movements cease; no faeces are passed. The animal becomes hyperaesthetic with twitching of muscles and high stepping gait. Occasionally convulsions are manifested. Periods of recumbency are common, the animal often lying on its side, and sometimes opisthotonus is present. Other cases have the typical recumbent position of milk fever with the head tucked into the flank. Some of these recumbent animals will rise, walk around and even resume cudding, and then revert to their former positions. Prolonged recumbency is always followed by death.

The course of the disease varies from 12 hours to 7 days. In fatal cases most animals die within 48 hours.

A marked feature in all cases is the presence of watery fluid in the rumen which in most cases is so voluminous that it can be heard "sloshing" as the animal walks[2]. Dehydration develops rapidly.

When recovery starts there is a watery diarrhoea, the faeces sometimes containing small quantities of undigested ruminal contents.

Opinions vary as to the reasons for the inability to swallow water. Van Heerden et al.[14] state that in their cases the tonus of the tongue was weak when pulled on. Bryson & Newsholme[12] found the tonus to be fully present and concluded that either pain in the pharynx was responsible for the dysphagia or that it was due to bulbar paralysis (c.f. rabies). Detailed examinations of the pharynx have revealed no obvious lesions[2]. Moribund animals may regurgitate ruminal contents prior to dying.

The severity of the signs probably reflects the amount of toxin ingested, and it is clear that if this is reduced as far as possible by immediate withdrawal from the toxic paddock, recovery is likely.

Blood chemistry in several of the outbreaks showed a consistently low blood magnesium content and in one outbreak, serum sodium was reduced[2 14]. A mild acidosis has been recorded in some cases.

## MORTALITY

In affected animals this is about 80 %. In those animals severly affected, it is 100 % and appears to be little influenced by treatment. Mildly affected animals recover without treatment.

## PATHOLOGY

### Macroscopic Pathology

Dehydration is marked although the animal may still be well-fleshed; eyes are sunken and injected and the blood is thick and dark. The most apparent lesions are in the abomasum and forestomachs. These organs are packed with bright green, well-chewed, sloppy ingesta which resembles that often seen in cases of bloat but without the gas. The omasum in particular is grossly distended with this fluid content and may be almost twice its normal size. Examination of the mucous membranes of these structures usually reveals a slight abomasitis while the other stomachs show focal areas of hyperaemia and erosion which do not often exceed 50 mm in diameter. New Zealand workers noted that the abomasal contents are less acid than normal[10] but in South Africa the ruminal pH has been within the normal range. The intestinal contents are dry and hard.

In most cases extensive epi- and endocardial haemorrhages are present, the latter being most frequently in the coronary and longitudinal grooves of the heart. Ecchymotic haemorrhages are also noted in thegmesenteric, suprarenal, precrural and prescapular lymph nodes.

The livers in some animals show areas of greyish-brown discoloration suggesting pseudo-necrosis. Mapham isolated *Clostridium welchi* and *Escherichia coli* from intestinal cultures[7].

### Histopathology

The most striking lesion in all cases is an intensive necrotising inflammatory process involving the epithelium of the forestomachs particularly that of the rumen and omasum. The superficial and, in places deeper, layers of the mucosa were completely necrotic. This change is accompanied by an intense infiltratrion of neutrophiles. Changes in other tissues are unremarkable save for a mild but consistent nephrosis and hyperaemia and occasionally oedema of the abomasal mucosa.

## TREATMENT

No specific treatment has yet been found and even symptomatic therapy has proved to be of limited value. Despite the low serum magnesium in affected animals, the intravenous or subcutaneous administration of magnesium alone or in combination with phosphorus and calcium (MFC, Maybaker) does not affect the poor prognosis[2 14]. The intravenous administration of glucose saline has no apparent effect.

To counteract the dehydration and to compensate for blood changes, electrolyte solutions have been given intravenously, but these appear to be of limited value. The administration of large doses of sodium bicarbonate to counteract the mild acidosis has no effect[2].

To stimulate stomach and bowel movements linseed oil, carron oil and carbachol have been administered without success. Sodium thiosulphate has been tried (G.F. Bath 1981 Allerton Laboratory, Pietermaritzburg, personal communication) therapeutically as has piracetam to counteract possible unknown toxins[14]. Vitamin B12 has been used on the assumption that a cerebro-cortical necrosis syndrome was present but with little success. The administration of preparations containing corsitone also have no apparent effect on the course of the disease.

Intravenous electrolyte therapy would appear to be the most obvious line to follow, but the value of fluids and/or purgatives administered per os is doubtful, and may possibly even be harmful. Antibiotics have no effect whatsoever.

It can be concluded that there is no effective treatment for this condition and that treatment of animals in the chronic stage of the disease, i.e. 3-5 days after the first toxicity, is particularly unrewarding. Mild exertion, even that produced by clinical examination, will speed up the animal's inevitable death.

## EXPERIMENTAL REPRODUCTION OF THE DISEASE

During 1977 Bryson & Newsholme reproduced the condition in 2 groups of steers introduced into a known toxic paddock. Typical signs of the disease manifested. As soon as these appeared the animals were removed and they recovered without treatment[2].

Mapham fed suspected toxic kikuyu grass to 2 cattle as follows:

Heifer 1 was fed 2 sacks of the grass on 9 June 1977. By 10 June 1977 it showed typical clinical signs and was destroyed. Typical lesions were present on necroscopy.

Heifer 2 received the same treatment. It died after 8 days having shown similar signs and post mortem features.

Feeding trials have also been carried out using a guinea pig and 3 rabbits. The guinea pig refused to eat the kikuyu. One rabbit died 13 days after feeding commenced without having shown signs. Concurrent uterine disease inhibited assessment of post mortem and the trial[7].

During the 1981 outbreaks, kikuyu grass from toxic paddocks on 3 different farms was sent to the Veterinary Research Institute, Onderstepoort for feeding trials in cattle. The grass being transported to the Institute by air and rail. In only one of the animals were mild signs observed. This was a Jersey bull of 380 kg body mass which was fed 22 kg grass. The signs passed off quickly and could not be considered diagnostic (Kellerman 1981, personal communication). All other feeding trials proved fruitless. It is possible that the method of collection, storage and transport may have altered the character of the grass, or the grass was cut from non-toxic areas in the paddock. It does appear, however, that hay made from affected pastures loses its toxicity as farmers in previous outbreaks have made hay from toxic paddocks with no reports of disease after it has been ingested by cattle.

It was noted in New Zealand that rainfall did not have any effect on known toxic paddocks, which indicates that the toxin does not appear to be water soluble[12].

## FUTURE OUTLOOK AND POSSIBLE CAUSAL FACTORS

In all the outbreaks both here and New Zealand toxic fungal material was suspected, and a wide range of cultural tests was made from grass and the army worm and its faeces. This showed a wide variety of potentially toxic fungal species particularly *Fusarium*, *Myrocethicum*, *Helminthosporium*, *Phizoctonia* as well as others[8] (Kellerman 1981, personal communication).

*Myrothecium verrucaria*, a known toxigenic species, was investigated in New Zealand as a possible causal factor and was found widely in material examined, but this fungus is a common contaminant of pastures. In New Zealand cultures of this fungus produced signs and lesions identical to those of kikuyu poisoning when fed to cattle and sheep. Martinovich considers that circumstantial evidence could implicate a particular strain of *M. verrucaria* and in the absence of other identified causal factors should at least be considered a possible factor. Kellerman considered that, in view of the small fungal population isolated in a recent ourbreak, a toxic fungal cause is unlikely. Toxicity in cattle on kikuyu pastures not damaged by army worm occurs sporadically both in South Africa and Australia as mentioned above.

## DISCUSSION

The causal factors of the disease remain obscure although the available evidence does point to an alteration in the composition of the kikuyu grass due to damage by the army worm. It may be that this damage is exacerbated by fungi and it is still conceivable that certain of these produce mycotoxins of a pathogenic nature. Circumstantial evidence and some experimental work does indicate mycotoxin produced by *M. verrucaria*.

It is noteworthy that on the few occasions where the disease was produced experimentally cattle were allowed to graze on pastures where field cases had recently occurred[2] or were fed very freshly cut grass[5]. It is possible that the transport of material involving 2-3 days leads to destruction of the toxic matter. Bath (1981 Allerton Laboratory, Pietermaritzburg, personal communication) noted during geeldikkop trials that if the toxic material was not frozen immediately its toxicity was lost.

Martinovich (1981 personal communication) suggests that a toxic pasture is usually safe after a few days but that when it remains toxic for a prolonged period, this is due to continued production of toxin rather than to the persistence of the toxin. The treatment of toxic pastures with fungicides on a trial basis may be a line of research to follow, but has never been tried. As to our knowledge at this moment, we can do little more than propagate the advice given during the last army worm outbreak. This is:

1. Use tracer animals on kikuyu pastures damaged by army worm. Observe them for a minimum of 96 hours before gradually introducing more and more animals to the pasture. The test animals should preferably be removed during the night because of

the difficulty of keeping them under surveillence.
2. Where the above recommended actions are not possible and where toxicity has occurred, rest the grazing for a minimum of 42 days.

Although sheep have been grazed in South Africa with impunity on many damaged or toxic pastures, New Zealand experience indicates that they are susceptible to kikuyu poisoning and that they possibly avoid toxic areas because of their more fastidious feeding habits. It has been noted that cattle are sometimes reluctant to graze for a time on a toxic paddock.

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# EFFECT OF A HIGH COPPER INTAKE AND DIFFERENT LEVELS OF MOLYBDENUM ON THE HEALTH OF SHEEP

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ABSTRACT: Van Ryssen J.B.J.; Botha W.S.; Stielau W.J. **Effect of a high copper intake and different levels of molybdenum on the health of sheep.** *Journal of the South African Veterinary Association* (1982) 53 No. 3, 167-170 (En) Department of Animal Science, University of Natal, P.O. Box 375, 3200 Pietermaritzburg, Republic of South Africa.

Molybdenum at levels of 0, 20, 40 and 60 mg/kg dry matter was added to a sheep ration containing 82 mg copper and 3,8 g sulphur per kg dry matter. At the 2 higher levels of molybdenum, a substantial accumulation of copper and molybdenum was observed in the kidney cortices of the South African Mutton Merinos (reported elsewhere). The effect of these treatments on the health of the sheep was investigated.

Observations on the 3 molybdenum supplemented treatments were: Serum enzyme levels remained normal throughout the trial. No evidence of kidney damage due to treatment could be obtained from kidney clearance studies. No pronounced liver lesions were evident from histophathological evaluations. A significant increase in Periodic acid Shiff positive stained granules was observed in the cortical tubules of the kidneys at increased molybdenum intakes. However, this was considered to be of minimal physiological significance. It was concluded that molybdenum prevented liver damage due to excess copper and that copper and molybdenum which did accumulate in the kidneys were probably harmless and present in an unavailable form to the body or caused minimal morphological changes.

No haemolytic crisis occurred in the sheep receiving no added molybdenum. However, their concentration of serum enzymes Aspartate transaminase and Lactate dehydrogenase increased throughout the 193 day trial. It is suggested that elevated levels of these enzymes may be unreliable indicators of an approaching haemolytic crisis in the Merino.

Key words: molybdenum, copper, sheep, kidney damage, liver damage, serum enzymes.

## INTRODUCTION

The well-known reaction of dietary molybdenum (Mo) plus sulphur (S) on copper (Cu) metabolism in the ruminant is a depletion of hepatic copper stores up to a stage of a copper deficiency in the animal[16]. However, when the copper reserves are adequate in the body, high dietary molybdenum and sulphur uptakes may cause an accumulation of copper and molybdenum in the body of sheep. This is most pronounced in the plasma and kidneys[14 16 17 18]. This accumulation of the minerals in the body was suggested to be a manifestation of a systemic effect of the copper-molybdenum-sulphur interaction in sheep[14]. Concern has been expressed that the accumulation of the minerals may have a harmful effect on the kidneys[17].

The formation of thiomolybdates in the digestive tract of sheep has been proposed as a mechanism in the copper-molybdenum-sulphur interaction. It was suggested that thiomolybdates could bind copper in the digestive tract and the body to form compounds unavailable to the animal[2]. These copper thiomolybdate compounds may therefore be responsible for these systemic effects in the body. Furthermore, attention has been given to the possible use of thiomolybdates to prevent and treat chronic copper toxicity in sheep[5].

In an investigation to evaluate the effects of different levels of molybdenum on the metabolism of copper and molybdenum in sheep fed high levels of copper, van Ryssen & Stielau[18] observed these systemic effects in the sheep at high molybdenum intakes. When 0 and 20 mg Mo/kg feed were added to the diets, the kidney cortices of the sheep contained 1,7 and 6,7 mg Mo/kg dry matter respectively and 26 mg Cu/kg dry matter in both groups. At the supplementation of 40 mg Mo/kg feed, 55 mg Mo and 94 mg Cu/kg dry matter were measured in the kidney cortices of the sheep. At 60 mg Mo/kg feed the kidney cortices contained 137 mg Mo and 236 mg Cu/kg dry matter[18].

During this trial, various measurements were taken to determine the effect of copper per se and of these so-called systemic effects (possibly thiomolybdates) due to the Cu-Mo-S interaction on the health of the sheep.

## EXPERIMENTAL

Thirty-two South African Mutton Merinos (16 wethers and 16 ewes), approximately one year of age, were randomly allotted within sex to 4 groups. All groups received a basic ration high in copper (82 mg/kg dry matter) and sulphur (3,8 g/kg dry matter) during the first 42 d of the trial. During this period, copper was allowed to accumulate in the livers of the sheep. At 42 d, different levels of molybdenum (as ammonium molybdate) were added to the basic ration of the sheep in each of the 4 groups, resulting in molybdenum levels in the feed of 0,6; 21,4, 39,6 and 60,3 mg/kg dry matter. The treatments were called the 0 Mo, 20 Mo, 40 Mo and 60 Mo groups respectively. After a further 193 d, the sheep were slaughtered. Details of the experimental procedures and rations have been published elsewhere[18].

Serum Aspartate transaminase (AST; EC 2.6.1.1) and Lactate dehydrogenase (LD; EC 1.1.1.27) concentrations were determined regularly using Boehringer Mannheim standard kits (Boehringer Mannheim GmbH Diagnostica) on a Gemsaec Fast Analyser (Electro-Nucleonics).

During the last 2 months of the trial, kidney clearances of inulin, endogenous creatinine and endogenous urea were done on 14 of the 16 ewes according to the method described by Smith[13] and Owen[11]. The ewes were sedated to prevent stress and to reduce the risk of a haemolytic crisis due to copper toxicity[15]. The primary dose of inulin was 40 ml of a solution of 24 g inulin/ℓ sterile saline. Inulin at a concentration of 7,5 g/ℓ saline was infused with an Unita II continuous infusion apparatus (B. Braun-Melsungen) in the left jugular vein at a constant rate of 1,5 to 2 ml/min, depending on ewe

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size. Urine collections (Foley's balloon type catheter, 12 FG) started after at least 45 min of inulin infusion. At least 3 collections were carried out per ewe. Blood was collected at the mid-point of each 20 min urine collection period. Serum and urine were analysed for inulin[12], creatinine (picric acid method based on the "Jaffe reaction" using a Technicon Auto Analyzer II) and urea (diacetyl monoxime method as modified for an Auto Analyzer).

At slaughter a complete autopsy was done on every sheep. Kidney and liver samples were collected in buffered formalin for histopathological evaluation. Tissue sections were stained by the haematoxylin and eosin as well as the Periodic acid Schiff (PAS) methods[8]. During microscopical examination, lesions observed, were rated as none, minimal, moderate or pronounced.

The analysis of variance was used to compare the results statistically and Duncan's Multiple Range Test[3] was used to isolate significant mean differences.

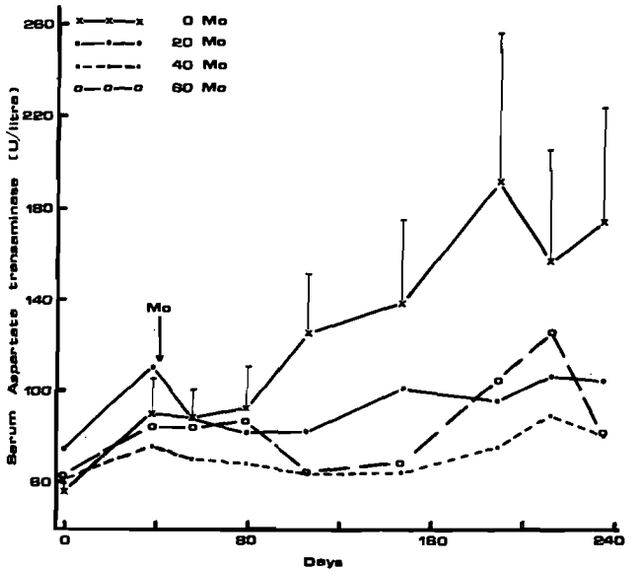


**Fig. 1:** Serum Aspartate transaminase (U/ℓ) concentration of sheep fed high levels of copper and different levels of molybdenum (Mo). Vertical bars in O Mo graph represent positive side of standard error of mean for that treatment.

## RESULTS

### Serum enzymes

The AST and LD levels in serum followed similar patterns within sheep throughout the trial. Therefore, only changes in AST levels are reported (Fig. 1). In the 0 Mo treatment, a correlation coefficient (r) of 0,82 was observed between serum AST and LD concentrations. A summary of serum enzyme levels has previously been published in table form[18].

During the 42 d pre-experimental period, serum enzyme levels tended to increase, but dropped in all 3 of the experimental groups where molybdenum was supplemented. Enzyme levels in the 0 Mo group generally tended to increase throughout the trial. This increase was due to increases in serum enzyme concentrations in 4 (the 4 ewes) of the 8 sheep, resulting in large standard errors of the means in the 0 Mo group (Fig. 1). One ewe in the 60 Mo group showed elevated enzyme levels throughout the trial, but they were apparently unrelated to the Mo treatment.

### Kidney clearance

For comparative purposes, the kidney clearances were expressed on a body mass basis[11] (Table 1). In most of the treatment groups, the variations between individuals within treatment were fairly high.

One ewe in the 60 Mo treatment showed very low inulin (0,83 ml/kg/min), high creatinine (3,14 ml/kg/min) and high urea (2,00 ml/kg/min) clearances as compared to the other ewes in that group. This ewe also had elevated serum enzyme levels throughout the trial, even during the pre-experimental period. Results from this ewe were therefore omitted in the statistical analyses.

The 0 Mo and 60 Mo treatments showed significantly ($P<0,05$) higher inulin clearances (glomerular filtration rates; GFR) than the 20 Mo and 40 Mo treatments. Endogenous creatinine clearance was significantly lower in the 0 Mo than in the 60 Mo treatment. The ratio between creatinine and inulin clearances in the 0 Mo group was lower than this ratio in all the groups receiving molybdenum, reaching a significant level ($P<0,05$) between the 0 Mo and 20 Mo groups. None of the differences in urea clearances between groups were significant.

Table 1: AVERAGE KIDNEY CLEARANCES OF INULIN, ENDOGENOUS CREATININE AND ENDOGENOUS UREA AND RATIOS OF CREATININE AND UREA CLEARANCES TO INULIN CLEARANCE OF EWES FED DIFFERENT LEVELS OF MOLYBDENUM (Mo).

| Treatment** (mg Mo/kg dry matter) | Clearances (ml/kg*/min) | | | Ratios | |
|---|---|---|---|---|---|
| | Inulin ($C_{in}$) | Creatinine ($C_{cr}$) | Urea ($C_{ur}$) | $C_{cr}/C_{in}$ | $C_{ur}/C_{in}$ |
| 0 Mo | 1,70[a] | 1,12[a] | 0,76 | 0,65[a] | 0,44 |
| SE | 0,06 | 0,14 | 0,06 | 0,08 | 0,02 |
| 20 Mo | 12,20[b] | 1,46 | 0,90 | 1,27[b] | 0,78 |
| SE | 0,15 | 0,24 | 0,19 | 0,32 | 0,20 |
| 40 Mo | 1,31[b] | 1,58 | 0,88 | 1,21 | 0.67 |
| SE | 0,10 | 0,10 | 0,15 | 0,08 | 0,07 |
| 60 Mo | 1,88[a] | 1,75[b] | 1,09 | 0,92 | 0,57 |
| SE | 0,13 | 0,24 | 0,16 | 0,07 | 0,05 |

*kg body mass
**Means within columns with superscripts a and b are significantly ($p<0,05$) different.
SE = standard error of mean.

## Necropsy findings

Indications of slight liver degeneration were observed in 4 sheep in the 0 Mo group and in one each from the other 3 treatment groups.

## Histopathological evaluation

*Kidney*

No pathological lesions were apparent in the haematoxylin-eosin stained sections. Distinct PAS positive granules, as small, round, red granules were observed in the cytoplasm of the convoluted tubules in the cortex (Table 2). A significantly ($P<0,05$) higher frequency of these granules was recorded in the 60 Mo than in the 20 Mo group.

Table 2: OCCURRENCE OF PERIODIC ACID SCHIFF (PAS) POSITIVE GRANULES IN RENAL CORTICAL TUBULES OF SHEEP FED DIFFERENT LEVELS OF MOLYBDENUM (Mo).

| PAS + granules rating | Treatments (mg Mo/kg dry matter) | | | |
|---|---|---|---|---|
| | 0 Mo | 20 Mo* | 40 Mo | 60 Mo* |
| | – – – number of sheep per rating – – – | | | |
| none | 1 | 2 | 0 | 0 |
| minimal | 3 | 4 | 3 | 2 |
| moderate | 4 | 2 | 4 | 4 |
| pronounced | 0 | 0 | 1 | 2 |

*Ratings at 20 Mo significantly ($P<0,05$) lower than at 60 Mo.

Table 3: OCCURRENCE OF INDIVIDUAL HEPATOCYTE NECROSIS IN LIVER OF SHEEP FED HIGH LEVELS OF COPPER AND DIFFERENT LIVELS OF MOLYBDENUM (Mo).

| Hepatocyte necrosis rating | Treatments (mg Mo/kg dry matter) | | | |
|---|---|---|---|---|
| | 0 Mo | 20 Mo* | 40 Mo | 60 Mo* |
| | – – – number of sheep per rating – – – | | | |
| none | 0 | 0 | 0 | 0 |
| minimal | 2 | 4 | 7 | 5 |
| moderate | 3 | 3 | 1 | 2 |
| pronounced | 3 | 1 | 0 | 1** |

*Rating of 0 Mo significantly ($P<0,05$) higher than at 40 Mo.
**Ewe with high serum enzyme levels throughout trial.

Table 4: HYPERTROPHY OF KUPFFER CELLS IN LIVERS OF SHEEP FED HIGH LEVELS OF COPPER AND DIFFERENT LEVELS OF MOLYBDENUM (Mo).

| Hypertophy of Kupffer cells rating | Treatments (mg Mo/kg dry matter) | | | |
|---|---|---|---|---|
| | 0 Mo | 20 Mo* | 40 Mo | 60 Mo* |
| | – – – number of sheep per rating – – – | | | |
| none | 0 | 0 | 0 | 2 |
| minimal | 3 | 6 | 8 | 4 |
| moderate | 5 | 2 | 0 | 2 |
| pronounced | 0 | 0 | 0 | 0 |

*Ratings of 0 Mo significantly ($P<0,05$) higher than 40 Mo and 60 Mo.

*Liver*

Significantly ($P<0,05$) higher frequencies of necrosis of the individual hepatocytes and hypertrophy of Kupffer cells were observed in the 0 Mo treatment than in the higher molybdenum groups (Tables 3 & 4). None of the other criteria of liver damage investigated, viz. enlarged nuclei, hydropic degeneration, fatty changes or portal cell infiltration showed any differences between treatments.

## DISCUSSION

Any harmful effect due to the systemic effect of the copper-molybdenum-sulphur interaction is most likely to occur in the kidneys of sheep. In the present trial, an accumulation of copper and molybdenum was observed in the kidneys of the sheep in the 40 Mo and 60 Mo treatments[18]. These copper levels were well above normal levels and within the range observed during a haemolytic crisis due to copper toxicosis when excessive renal tissue damage occurs[14]. Elevated AST and LD concentrations in serum are indicative of tissue damage in the body[15 17]. However, during the present trial, no increase in concentration of these enzymes was observed in the 40 Mo and 60 Mo treatments.

No definite conclusions could be drawn from the kidney clearance study because of lack of numbers per treatment. However, the GFR values of the 0 Mo and 60 Mo groups compare well with values of 1,75 to 2,35 ml/kg/min quoted by Owen[11]. The significantly lower GFRs in the 20 Mo and 40 Mo groups are difficult to explain, and do not correspond with the occurrence of the sytemic effects in the kidneys. The endogenous creatinine clearances of all the groups receiving molybdenum corresponded well with the GFRs of the respective groups. This agrees with observations that inulin and creatinine have approximately the same excretion rates[10 12 13]. The significantly lower clearance of creatinine in the 0 Mo group is of interest and will be discussed later. The clearance of endogenous urea was not affected by any treatment and compares well with urea clearance values in the literature[12].

The only changes in this trial which could be related to the systemic effects were the increases in PAS positive granules in the cortical tubules with increased molybdenum intakes. These granules may represent swollen mitochondria and therefore the early stages of parenchymal degeneration. However, these changes probably caused minimal disturbance in the physiology of the animal.

In general, it seems as if the systemic effect had a negligible influence on the health of the sheep. This would support suggestions that copper and molybdenum accumulated in tissues in an inert form unavailable to the body[7 14 18].

The liver histopathology and serum enzyme levels in this trial suggest that tissue breakdown took place in the livers of some sheep, mainly in those of the 0 Mo group. The addition of molybdenum at 40 and 60 mg/kg to the feed prevented any liver damage in the sheep fed 82 mg copper and 3,8 g sulphur per kg feed. The histopathological changes in the livers of the 0 Mo group correspond with the changes observed by Ishmael et al.[8] during the pre-haemolytic stage of copper toxicity in sheep. No lesion typical of the post-haemolytic stage was present in either the liver or kidneys[6]. Elevated

serum enzyme concentrations were observed in this trial for extended periods without the occurrence of any haemolytic crisis. This does not support the observation by various other workers that a haemolytic crisis can be expected within 8 weeks after the increase in serum enzyme concentrations[14 15 17]. Merino types as used in the present trial, reportedly have a higher resistance to copper toxicosis than other sheep breeds and an ability to survive more than one haemolytic crisis[17]. It is possible that they may also show elevated serum enzyme levels for protracted periods without advancing into a haemolytic crisis. This suggests that elevated AST and LD levels in serum may be unreliable indicators of an approaching haemolytic crisis in Merino type sheep.

Thiosulphate and p-aminohippurate can decrease the creatinine clearance from kidneys without altering the inulin clearance[10]. At high sulphur intakes, as in the trial, relatively high thiosulphate levels may be present in blood[1]. However, the presence of even small quantities of molybdenum in the rumen reportedly reduces sulphide absorption from the rumen substantially[4]. The formation of thiomolybdates in the rumen may also decrease the concentration of other sulphur compounds in the blood[2 4]. It is therefore possible that lower levels of thiosulphate were present in the blood of the sheep fed additional molybdenum than in the O Mo group. This may explain the lower rate of creatinine clearance and creatinine to inulin clearance ratios in the O Mo group as compared to the other treatments.


## ACKNOWLEDGEMENTS

The authors wish to thank Dr. J.P. Kitching of the Epol Feed Company for carrying out the necropsies and Mr. B. D. Thring for assistance during the trial.


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## BOOK REVIEW — BOEKRESENSIE

# A BIBLIOGRAPHY AND KEYWORD INDEX OF THE BITING MIDGES (DIPTERA: CERATOPOGONIDAE)

W.R. ATCHLEY, W.W. WIRTH, C.T. GASKINS and SANDRA L. STRAUSS

U.S. Department of Agriculture, Bibliographies and Literature No. 13. 544 pp. Free from Office of Governmental and Public Affairs, U.S. Department of Agriculture, Washington, D.C. 20250.

This is an invaluable bibliography and keyword index for the specialist worker on these midges as it includes 3 527 primary and secondary references on worldwide literature for the period 1758 to 1978.

Not all references are equally retrievable using the Keyword-in-Context Index because limitations had to be placed on the size of such a publication and various stopwords had to be introduced to eliminate some entries.

Such a bibliography, however, brings home the importance of using highly descriptive explicit titles in scientific papers and the inclusion of keywords at the ends of abstracts. The authors bemoan such titles as "Studies on the Genus X'us" and "Further Studies on the Genus X'us" as these are useless for bibligraphic index systems.

This is worth our noting if we want a wide readership of our publications.

**E.M. Nevill**

# THE SEASONAL INCIDENCE OF THE SHEEP ITCH MITE, *PSORERGATES OVIS* WOMERSLEY UNDER SUB-TROPICAL CONDITIONS

F.S. MALAN and NANCY A. ROPER*

ABSTRACT: Malan F.S.; Roper N.A. **The seasonal incidence of the sheep itch mite, *Psorergates ovis* Womersley under sub-tropical conditions.** *Journal of the South African Veterinary Association* (1982) **53** No. 3, 171-174 (En) Hoechst Research Farm, P.O. Box 124, 1320 Malelane, Republic of South Africa.

Peak numbers of mites were present in the colder months July to September. A single dipping in triazophos (Zipdip, Hoechst Pharmaceuticals, Johannesburg) at a concentration of 60 ppm (parts per million) or 120 ppm and phoxim (Sarnadip, Bayer, Isando) at 1 000 ppm controlled but did not eradicate *P. ovis*.

The *P. ovis* populations of Merino sheep were determined by monthly skin scrapings during the first year and three-monthly scrapings during the second year.

Key words: *Psorergates ovis*, sheep itch mite, triaphos phoxim.

## INTRODUCTION

The literature on *Psorergates ovis* has extensively been reviewed by McHardy[4]. Armstrong[1] in New Zealand claims that diagnosis is difficult during January, February and March (warm months). Downing & Mort[2] agree with Graham[3] that there is a marked reduction in the mite population on recently shorn sheep. This decrease is greater if the sheep are exposed to high temperatures and sunlight, and an increase in mite populations follows a period of cold weather[2]. Sherman et al.[7] mentioned that there was danger in assessing insecticidal action on a falling mite population in summer. It follows therefore, that on recently shorn sheep the mites are affected, their numbers decrease but transfer may occur most readily to other shorn sheep. Downing & Mort[2] agreed and furthermore stated that any results from assessing an insecticide on a "falling parasite" population might be completely the opposite on a "rising active parasite" population (which usually occurs following a cool spell.) Although infestation is reduced by shearing and warm, dry weather, no evidence of self-cure has been encountered[2].

The present paper describes the seasonal fluctuations of mite populations on sheep on a farm in the warm, humid eastern Transvaal Lowveld.

## MATERIALS AND METHODS

### Sheep

Forty-one Merino sheep were bought in the Karoo during October 1979. *P. ovis* was identified microscopically on a few sheep after making skin scrapings and additional sheep were selected on the basis that they showed clinical signs of scurf on the skin, yellowish discolouration of the wool and tufts of wool that had been pulled out of the fleece. The sheep were shorn about 2 weeks prior to their purchase.

On arrival at the Hoechst Research farm near Malelane in the eastern Transvaal Lowveld the animals were dewormed and immunized agaionst enterotoxaemia infection. They were housed on concrete-floored sheep pens which were covered by an asbestos roof but open on the sides. The pen size for each group of sheep was 3,35 m². Clean fresh water and hay were available ad libitum. In addition each sheep consumed 300 gram of sheep finisher cubes per day.

The sheep were shorn with an automatic sheep shear in October 1979, December 1980 and again in February 1982 after the trial had ended.

### Mite examination

Mite examination was done according to the methods described by Downing & Mort[2].

### Acaricides

Triazophos E.C. (Zipdip Hoechst Pharmaceuticals, Johannesburg) 40 % m/m at concentrations of 60 and 120 ppm (parts per million) and phoxim E.C. (Sarnadip, Bayer South Africa) 50 % m/m at a concentration of 1 000 ppm were evaluated as acaricides.

### Dip trial

The sheep were ranked according to their mite counts and thereafter allotted to 3 groups so that each group to be treated had sheep with approximately the same level of infestation as follows

Eight sheep treated with triazophos at 60 ppm with a mean mite count of 15,3 per scraping.

Eight sheep treated with triazophos at 120 ppm with a mean mite count of 10,7 per scraping.

Eight sheep treated with phoxim at 1 000 ppm with a mean mite count of 15,6 per scraping.

Eight sheep with a mean mite count of 6,9 per scraping were left as untreated controls.

An additional control group of 9 sheep was left untreated. Although they showed clinical signs of infestation no mites could be demonstrated.

The dip tank was rectangular with a volume of 1 000 ℓ. The sheep were individually dipped for 1 min and their heads were submerged at least 3 times so that proper wetting was achieved.

During the first year, October 1979 to October 1980, examinations took place at monthly intervals and during the second year, October 1980 to 1981, at 3 monthly intervals.

The sheep were only dipped once on 24 October 1979 and not again and scrapings were made at regular intervals thereafter as described above. For the first 3 months of the trial one area was scraped per inspection i.e. the upper left lumber area, thereafter 3 areas i.e. one on the left, one on the top line (dorsal) and one on the right side. No area was scraped twice during the year. During

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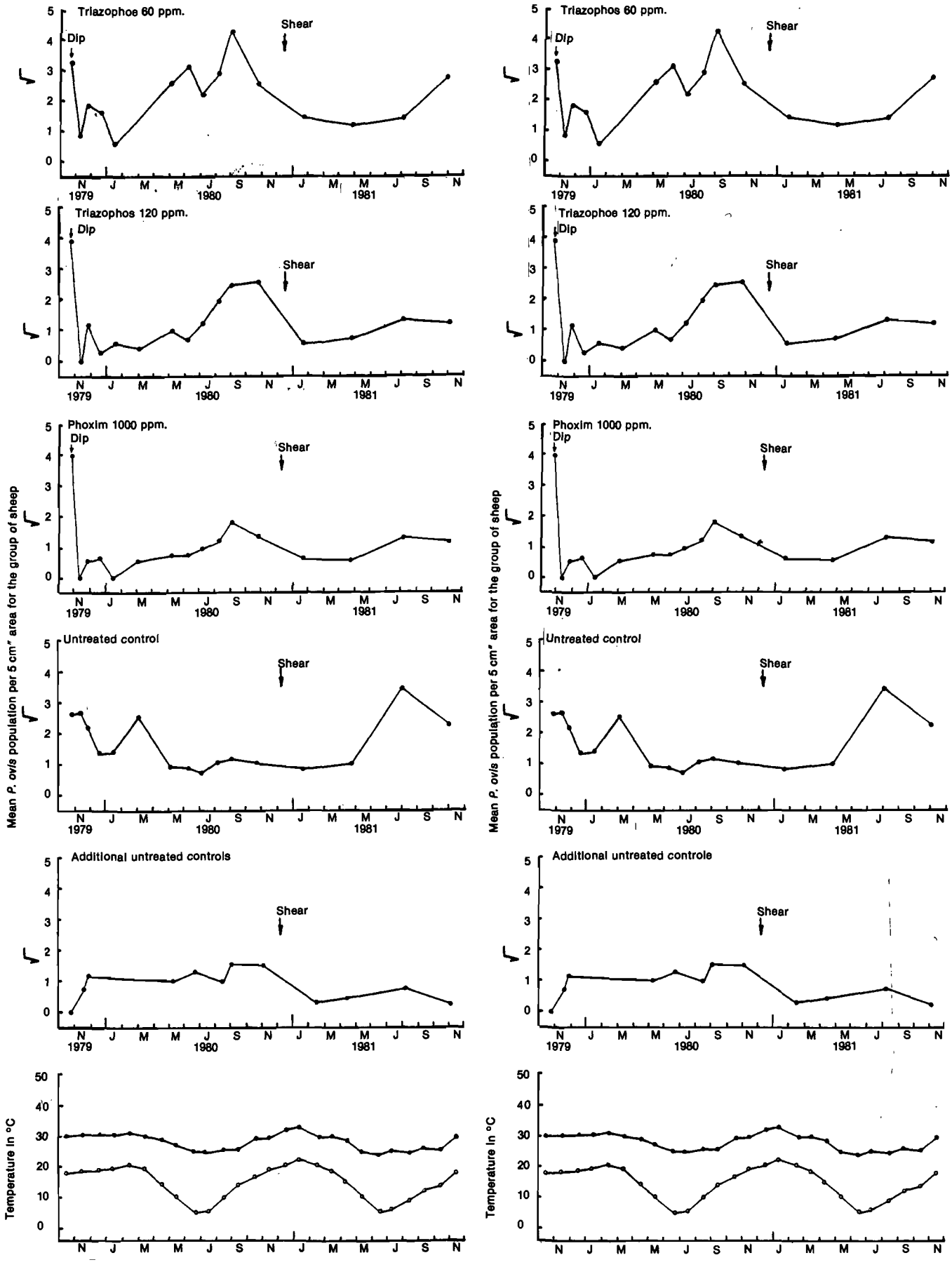


**Fig. 1** Variations in the mean *P. ovis* per 5 cm² area (√ scale) in groups treated either with triazophos (60 ppm), triazophos (120 ppm) phoxim (1 000 ppm) compared with untreated controls and an additional control. Mean maximum ●—● and mean minimum ○—○ temperatures.

November (spring) each sheep with negative mite counts from these 3 areas had further examination until as many as 14 areas had been looked at. Only when all these sites yielded no mites were the sheep regarded as being mite free. The mites were classified as dead, immature (either larvae or of 3 nymphal stages) and adults.

## RESULTS

Results are illustrated in Fig. 1

### Triazophos 60 ppm

On the day of dipping the mean mite count was 10,7 ranging from 1 to 60. Nineteen days after treatment only 2 mites were found. The sheep with the highest count initially (60) rose dramatically from 0 in March to 46,7 in May and to 126,3 in September 1980. One sheep died in March 1980. In November 1980 only one sheep was positive, 6 being negative. After shearing in December 1980 with the higher temperatures of summer the mean count fell from 6,6 to 2,2 rising again to 7,5 in November 1981. Four sheep were positive in August 1981 and by making an additional 14 scrapings per sheep a further 2 were found to be positive. No scrapings were done during March 1980 for this group.

### Triazophos 120 ppm

The mean mite count for the group of 8 sheep per scraping on the day of treatment was 15,3 with a range of 1 to 63. Nineteen days after treatment no live mites could be found. Mite counts increased in August 1980 with the highest mean count per scraping being 6,3 during October 1980, with a range from 0 to 22,6. After scraping up to 14 patches per sheep in November 1980 and November 1981 only 2 sheep were negative. After shearing in December 1980 the mean count dropped to 0,3 rising to 1,9 in August 1981, the highest individual count being 8,3.

### Phoxim 1 000 ppm

On the day of dipping the mean count per scraping for the group was 15,6 ranging from 2 to 73. Nineteen days after treatment no live mites could be found. During September 1980 the highest count was 25, four animals being positive and the mean count being 3,3. Shearing and higher temperatures decreased the mean count from 1,8 in October 1980 to 0,4 in January 1981. In November 1981 five out of 7 animals were positive with a mean count of 1,3 mites per slide.

### Controls

The mean mite count per scraping for the group on the day of treatment was 6,9 with an individual range of 1 to 30. During the first year after shearing, there was a decrease in mean count in January 1980 to 1,9 rising to 6,7 in March 1981, the highest count being 38. No marked rise took place during September 1980 and all the sheep were still positive at the time of shearing in December 1980. During the examination in August 1981 one sheep which always had low counts showed a rise in mean counts from 6,3 mites in April to 76,4 in August. All the sheep were still positive in November 1981.

### Additional controls

On the day the dipping trial commenced no mites were recovered from the scrapings. The mean count for the group rose to 2,4 in September 1980 with the highest individual count being 16,4 per scraping. At shearing 2 animals were positive. During the next year the mite counts remained very low i.e. 0,6 in August 1981 being the highest mean count.

In November 1981 when up to 14 sites per animal were examined only 2 sheep were positive. Scrapings were not done from November 1979 to May 1980.

## DISCUSSION

A single dipping controls but does not eradicate *P. ovis*. The reason for this may be two-fold in that some stages are protected in the upper layers of the skin while other stages e.g. the egg and moult, are resistant to the acaricide and thus survive treatment. A second dipping three weeks after the first is therefore advocated.

Mite numbers per scraping showed a similar trend in all the treated groups. After dipping in October 1979 mite numbers decreased rising slowly during late winter to reach a peak in the spring.

The untreated controls didn't follow the same trend as the treated animals during the first year (1980).

After shearing in October 1979 mite numbers decreased until January 1980 rising in March 1980, decreasing in May to remain low during the year only to rise again in August 1981. These observations confirm Graham's[3] results that by exposing the mites to high temperatures and light after shearing the mite population decreases. Furthermore Skerman et al.[7] observations were confirmed that there is a danger in assessing insecticidal action on a falling mite population in summer. As misleading deductions can be made from these results assessments should only be made after a full winter and spring have passed, following treatment the previous summer. The necessity of making additional scrapings (up to 14) was also shown in that 11 sheep with negative results after 3 scrapings proved to be positive for *P. ovis* after additional sites were examined.

One sheep from the control group showed an interesting phenomenon: for 20 months the mite count per scraping was less than 4 mites; then in August 1981 there was a dramatic increase to 76,4 mites per scraping. Sheep with high mite counts tended to maintain higher counts throughout the trial period. From this it may be deduced that in a positive flock some animals may serve as reservoirs for the spread of the disease. Why the mite counts of the control group didn't increase during the late winter and spring of 1980 as was the case in the treated groups is unknown. Although the untreated animals were confined in small pens no increase in mite population took place. Some animals showed sporadic positive counts e.g. Sheep 45, 40, 27 and 32, only to become negative again.

Murray[5], Toop[8] and Graham[3] cited by McHardy[4] stated that infestation by direct contact occurred soon after shearing when short wool favours the transmigration of the parasite e.g. ewe to lamb, and that migration of mites from full fleece sheep is unlikely.

New Zealand investigators[1,6] showed that apart from a short period immediately after shearing the transmission of mites from one woollen sheep to another was extremely slow. Infestation spread very slowly so that 3 or 4 years could elapse before the condition became widespread in a flock. From the results of the present study it seems logical that when sheep are sheared at a time when *P. ovis* numbers are high the chances of

transmission are much greater. When *P. ovis* is rife in a flock close contact between animals brought about by herding should be as short and as infrequent as possible.


## ACKNOWLEDGEMENTS

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ABSTRACT: Gray, J.S. & Potgieter, F.T., 1981. **The retention of *Babesia bigemina* infections by *Boophilus decoloratus* exposed to imidocarb dipropionate during engorgement.** *Onderstepoort Journal of Veterinary Research*, **48**, 225-227 (1981).

*Babesia bigemina* was retained in the vector *Boophilus decoloratus* for a complete generation despite the use of the babesicide, imidocarp dipropionate, to prevent reinfection. This drug did not sterilize ticks of the *B. bigemina* infection as has been suggested for *B. bovis*.



ABSTRACT: Reinecke, R.K., Bruckner, Christel & De Villiers, I.L., 1981. **Studies on *Haemonchus contortus*. IV. The effect of *Trichostrongylus axei* and *Ostertagia circumcincta* on challenge with *H. contortus*.** *Onderstepoort Journal of Veterinary Research*, **48**, 229-234 (1981).

Worm-free Merino yearlings were dosed with either a mixture of infective larvae of *Trichostrongylus axei* and *Ostertagia circumcincta* or with *O. circumcincta* only, and challenged 90-93 days later with infective larvae of *Haemonchus contortus*. Neither of these methods protected sheep against challenge and slight protection was afforded sheep predosed with *T. axei* and *O. circumcincta* and challenged with a trickle dose of *H. contortus*.



ABSTRACT: Reinecke, R.K., Bruckner, Christel & De Villiers, I.L., 1981. **Studies on *Haemonchus contortus*. V. Chemoprophylaxis and its effect on worm egg counts and the haematocrit.** *Onderstepoort Journal of Veterinary Research*, **48**, 235-238 (1981).

A group of 12 10-month-old, worm-free Merino lambs were given a single injection of disophenol at 10 mg/kg. This group and another group of 12 worm-free lambs were infested with infective larvae of *Haemonchus contortus* 1-3 times a week with a maximum dose of 4000/week from 0-91 days. From 95-168 days the dose was increased to a maximum of 6000/week. Every 7 days faeces were examined for worm eggs and blood samples for haematocrit (Ht). Worm egg counts in the controls rose from 21-56 days, fluctuated, and rose to another peak at 84 days, while the Ht fell from 28 days to rise again after 105 days. In the group treated with disophenol egg counts were negative until 161 days and Ht remained at normal values throughout.

# PRELIMINARY REPORT ON THE STIMULATION OF IMMUNITY TO THE LARVAL STAGE OF *TAENIA MULTICEPS*

ANNA VERSTER* and R.C. TUSTIN**

ABSTRACT: Verster A.; Tustin R.C. **Preliminary report on the stimulation of immunity to the larval stage of *Taenia multiceps*.** *Journal of the South African Veterinary Association* (1982) 53 No. 3, 175-176 (En) Section Helminthology, Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa.

The efficacy of Oncosphere Secretory Antigen (OSA) in protecting sheep against the larval stage of *Taenia multiceps* was assessed in 47 sheep in 2 trials. In the pilot trial no cerebral coenuri were found in 3 sheep which were treated with OSA 28 and 14 days before challenge with 6 000 eggs of *T. multiceps*. Cerebral coenuri were present in 3 untreated controls. In the second experiment 30 sheep were similarly treated with OSA and challenged with 5 000 eggs of *T. multiceps*, while 11 sheep served as untreated controls. At necropsy either developing coenuri or degenerate lesions were present in the brain of 5 (16,6%) of the 30 vaccinated animals while 8 out of 11 (72,7%) of the untreated animals had cerebral coenuri or degenerate lesions in the brain. It is concluded that OSA may be used to protect animals against cerebral coenuriosis.

Key words: *Taenia multiceps*, coenuriasis, immunity to *Taenia multiceps*.

## INTRODUCTION

Richard & Bell[5] successfully vaccinated lambs against the larval stage of *Taenia ovis* with an antigen secreted by activated oncospheres during *in vitro* cultivation. Subsequently Rickard & Adolph[3] used a similar antigen to protect calves against the larval stage of *Taenia saginata*. Recently Edwards & Herbert[1] found that antigens derived from activated oncospheres of *Taenia multiceps* protected lambs against infestation with the metacestodes of this species.

This report deals with 2 trials in which an oncosphere secretory antigen (OSA) was used to stimulate immunity in sheep to the larval stage of *Taenia multiceps*.

## MATERIALS AND METHODS

*T. multiceps* was collected at necropsy from experimentally infested dogs. Ova from the gravid proglottids were used to prepare the antigen as described by Rickard & Bell[5], but NCTC 135 (Flow Laboratories) and not medium 858 was used as the culture medium. The antigen used in the pilot trial was stored at −20°C for 2 months and that for the other experiment stored for 13 months before it was used.

### Pilot Trial

Six Dorper sheep, 3-7 months of age, which were bred and reared at the Institute, were divided into 2 equal groups and treated as summarized in Table 1.

Table 1: EXPERIMENTAL DESIGN OF PILOT TRIAL

| Day | Treatment | |
|---|---|---|
| | Group 1 (Unvaccinated) | Group 2 (Vaccinated) |
| −28 | — | OSA* |
| −14 | — | OSA |
| 0 | 6 000 *T. multiceps* eggs per os | 6 000 *T. multiceps* eggs per os |
| +33 | Necropsy | Necropsy |

*Oncosphere Secretory Antigen.

At necropsy the brain, spinal cord, heart, lungs, liver, kidneys and all the muscles were examined for lesions.

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### Trial 2

The Merino sheep were approximately 4 months old when the trial commenced and were obtained from a farm in the Standerton district where *T. multiceps* does not occur. The 41 animals were divided into 2 groups of 11 (Group 3, unvaccinated) and 30 animals (Group 4, vaccinated). The animals in Group 4 were vaccinated as described for Group 2 in Table 1. On Day 0 all the animals in both Group 3 and 4 were challenged with 5 000 *T. multiceps* ova. With the exception of an animal in Group 3 which died 19 days after challenge, all the animals were slaughtered and examined post mortem 36-38 days after challenge.

At necropsy the animal that died 19 days after challenge as well as 4 other animals in Group 3, and 4 in Group 4 were examined as described for Trial 1 above. In the remaining animals the brain, heart, lungs, liver, kidneys and masseters were examined for lesions. In addition, the viscera of all the animals were examined for cysticerci of *Taenia hydatigena*.

## RESULTS

### Pilot Trial

Group 1 (Unvaccinated): Cerebral coenuri (9-104) were present in all 3 animals in Group 1 and one animal also had a coenurus in the spinal cord. All the animals had degenerate lesions in the heart (9-25), masseters (4-10), tongue (1-3) and skeletal muscles (2-30), while 2 animals had them in the kidney (1 and 5) and one had 4 in the lungs.

Group 2 (Vaccinated): There were no cerebral lesions in these 3 animals. One animal had 2 degenerate lesions in the heart. The other 2 animals had numerous greyish-white lesions with a denser centre in the liver; the lesions varied from 2-3 mm in diameter.

### Trial 2

The numbers of sheep in Groups 3 and 4 in which lesions were found in the brain, heart, liver and kidneys are summarized in Table 2.

Group 3 (Unvaccinated): In 5 of the 8 animals with cerebral lesions, the coenuri were viable and contained developing scolices, but in 3 animals the lesions were degenerate. In one animal there were 4 degenerate lesions, 4-5 mm in diameter, in the skeletal muscle which were classified as degenerate larvae of *T. ovis*. Viable larvae of *T. hydatigena* and/or degenerate lesions caus-


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Table 2. GROUPS 3 AND 4: NUMBERS OF SHEEP WITH LESIONS OF *T. MULTICEPS*

| Group | Treatment | No. of animals PER GROUP | Sheep with lesions | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | Brain | | Heart | | Liver | | Kidneys | |
| | | | No. | % | No. | % | No. | % | No. | % |
| 3 | Unvaccinated | 11 | 8 | 72,7 | 8 | 72,7 | 5 | 45,5 | 2 | 18,2 |
| 4 | Vaccinated | 30 | 5 | 16,6 | 9 | 30,0 | 24 | 80,0 | 6 | 20,0 |

ed by the larvae of this cestode were present in 8 animals.

Group 4 (Vaccinated): Three animals had viable cerebral coenuri, one animal had one viable and one degenerate lesion and another animal a single degenerate cerebral lesion. The majority of the animals (80,0 %) had degenerate lesions in the liver and in one animal these were too numerous to be counted. Large degenerate lesions, classified as those of *T. ovis*, were present in 9 of the sheep while viable and/or degenerate cysticerci of *T. hydatigena* occurred in 14 animals.

### Development of the coenuri

The viable coenuri recovered from the brain of the sheep in Group 3 (Unvaccinated) varied from 3-15 mm and those from animals in Group 4 (Vaccinated) from 5-7 mm in diameter. Developing scolices were present in varying numbers in the coenuri from both groups, but while there were incompletely developed rostellar hooks on the scolices in the coenuri from Group 3 (Unvaccinated) no such hooks were found in the coenuri from Group 4 (Vaccinated).

## DISCUSSION

These results confirm the findings of other workers [1 3 5] that secretory products of developing embryos of *T. multiceps*, *T. saginata* and *T. ovis* may be used as a vaccine to protect animals against infestation with the metacestodes of the *Taenia* spp. concerned.

The fact that 16,7 % of the vaccinated animals failed to become immunized may be the result of a variety of factors. There is a degree of cross-immunity between *T. hydatigena* and *T. ovis* when the animals are immunized with OSA antigen[5], but the presence of *T. hydatigena* in a lamb does not prevent subsequent infestation with *T. ovis*[6]. Moreover Edwards & Herbert[1] found that previous exposure to *Taenia* oncospheres may counteract the effects of vaccination.

When pregnant ewes are treated with OSA colostral transfer of *T. ovis* antibodies confers a passive immunity to the lamb which may persist for 9 weeks[4]. There is, however, no statistical evidence that naturally infested ewes pass immunity to their lambs[2].

Further investigations must be carried out to determine whether vaccination of pregnant ewes will result in passive immunity to coenuri of *T. multiceps* in their lambs, and if so, how long this immunity persists. The age at which the lambs themselves should be vaccinated and the duration of their immunity must also be determined. Lambs 3-4 months old are highly susceptible and 80 % become infested while only 16 % of 7-8 months old lambs and 3 % of older animals are susceptible[7]. It is not known whether vaccination will interfere with the development of such age immunity.

### ACKNOWLEDGEMENTS

Mesdames R. de Kock, Z. Haarhoff and E. Venter and Messrs. F. de Villiers, D. Meyer and P. Minnaar are thanked for their technical assistance.

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# THE SURVIVAL OF *TAENIA SAGINATA* CYSTICERCI IN BEEF CARCASES SUBJECTED TO ELECTRICAL STIMULATION

J.T.R. ROBINSON*

ABSTRACT: Robinson J.T.R. **The survival of *Taenia saginata* cysticerci in beef carcases subjected to electrical stimulation.** *Journal of the South African Veterinary Association* (1982) 53 No. 3, 177-178 (En) Department of Pathology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

*Taenia saginata* cysticerci present in 2 bovine carcases were subjected to high voltage electrical stimulation during routine slaughter. A total of 125 cysts were dissected out of each carcase and tested for viability over a period of 23 and 28 days respectively, during which the carcases were held at chilling temperatures. Despite showing organoleptically unacceptable putrefactive changes at the end of the storage period, both carcases yielded viable cysts (36 % and 40 % of those tested respectively).

Key words: *Taenia saginata*, cysticerci, cestode, electrical stimulation.

## INTRODUCTION

Electrical stimulation (ES) of carcases is a technique recently developed as part of dressing procedure in many abattoirs around the world and an outline of the reasons for its introduction is appropriate.

Increasing attention has been paid to meat hygiene over the past 20 years, stimulated in part by the greater tempo of international movement of meat, much of it in chilled form. One of the important measures taken to limit the multiplication of the inevitable contaminant organisms on dressed carcases is to reduce the carcase temperature as rapidly as possible after leaving the slaughter floor. Modern quick chilling facilities are capable of high efficiency in this regard, particularly where small carcases such as lambs are concerned.

Unfortunately, when muscle fibres are cooled to below 10°C before their pH has decreased by means of anaerobic glycolysis to 6,1 or less, the cold stimulus causes actin and myosin fibrils to overlap to an exaggerated degree. The resultant shortening of the muscle fibre persists until fixed in that position by rigor mortis and this leads to abnormal and irreversible toughness of the meat. The phenomenon is known as "cold shortening" and attracted attention in the early 1960's when it became a problem in exported New Zealand lamb carcases. Since that time cold shortening has been observed and well studied in beef and lamb in various countries including the Republic of South Africa[1 2 3].

Two possible methods of control of the phenomenon presented themselves: either to reduce the speed of chilling of carcases by holding them at more than 10°C for the time required to allow the pH to drop to 6,1 or below (approximately 10 h in normal carcases) or to speed up post mortem anaerobic glycolysis so that the critical pH level was reached before the carcase musculature cooled to 10°C in the quick chillers.

The first alternative has obvious meat hygiene disadvantages, but the second has proved successful by means of ES.

Electrical stimulation consists of passing an electric current through the carcase as soon after slaughter as possible and in any case no later than 40 min after bleeding in order to accelerate the rate of breakdown of muscle glycogen to lactic acid in the anaerobic myofibrils and thus reduce the muscle pH very rapidly. Where ES has been applied, it has not only been successful in eliminating cold shortening but has brought other meat quality benefits in its wake: more rapid maturing, the possibility of hot deboning without the risk of thaw rigor in frozen cuts, brighter meat colour and other desirable properties.

One of the possible major advantages which could possibly flow from ES and which had not been reported in the literature was its effect on cysticerci in beef. It was conceivable that the passage of a powerful electric current through carcases may kill or render non-viable the scolices of *Taenia saginata* in "measly" beef. A trial was therefore undertaken to investigate this possibility.

## MATERIALS AND METHODS

Two *T. saginata* infested carcases which had passed through a routine ES system were held at chill temperatures (−1°C to +3°C) for 23 and 28 days respectively, by which time both showed sufficient organoleptic changes to make them aesthetically unacceptable to normal consumer tastes.

*T. saginata* cysts were dissected out of various muscles from both carcases at intervals during chill-holding and were then incubated at 37°C in 40 % fresh ox bile for 18 hours. Any cysticercus showing complete or partial evagination of the scolex was scored as viable. No infectivity tests were done.

The ES system consisted of an electrified stainless steel bar making contact with the withers region of carcases, with the overhead rail acting as earth via the hoof and roller. The current employed had a potential of 500 V rms at 10 HZ with 14,3 cycles/second and with the polarity reversed every 20 seconds. Period of contact: 60 seconds.

## RESULTS

These are expressed in Tables 1 and 2.

Table 1: NUMBER OF VIABLE *T. SAGINATA* CYSTS/TOTAL NUMBER TESTED; CARCASE NO. 1.

| Post-slaughter period: | 5 days | 18 days | 28 days |
|---|---|---|---|
| Muscles sampled | No. viable | No. viable | No. viable |
| *Triceps brachii* | 9/10 | — | 2/5 |
| Neck muscles | 10/10 | — | — |
| *Gluteus medius* | 10/10 | — | 1/5 |
| *Long. dorsi* | 10/10 | 3/10 | 3/5 |
| H/limb adductors | 9/10 | 10/20 | — |
| *Quadriceps* group | — | 6/20 | 3/10 |
| Totals: | 48/50 (96 %) | 19/50 (38 %) | 9/25 (36 %) |

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Table 2: VIABLE CYSTS/TOTAL TESTED; CARCASE NO. 2.

| Post slaughter period: | 4 days | 14 days | 23 days |
|---|---|---|---|
| Muscles sampled | No. viable | No. viable | No. viable |
| *Triceps brachii* | 8/10 | 19/20 | 3/5 |
| *Long. dorsi* | 18/20 | — | 2/5 |
| *Biceps femoris* | 17/20 | — | 2/5 |
| Neck muscles | — | 8/10 | — |
| *Semimembranosus* | — | 18/20 | 3/5 |
| *Quadriceps* group | — | — | 0/5 |
| Totals: | 43/50 (86 %) | 45/50 (90 %) | 10/25 (40 %) |

## CONCLUSION

Two conclusions can be drawn:

1. Whatever the benefits that may ensue from the electrical stimulation of beef carcases, the destruction of the cysticerci of *T. saginata* is not one of them.
2. Although the percentage of viable cysts decreases with storage of beef carcases at chill temperatures, live cysticerci are liable to persist for at least 28 days or until marked putrefactive changes supervene.


## ACKNOWLEDGEMENTS

My thanks to Mr M. Shaer of Bull Brand Foods (Pty) Ltd. and Dr A.J.P. Machado of the Meat Hygiene Division of the Department of Veterinary Services for their cooperation in making carcases available for this trial.


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ABSTRACT: Littlejohn, A. & Bowles, Felicity, 1981. **Studies on the physiopathology of chronic obstructive pulmonary disease in the horse. V. Blood gas and acid-base values during exercise.** *Onderstepoort Journal of Veterinary Research*, **48**, 239-249 (1981).

The haemoglobin concentration, the partial pressures of oxygen and carbon dioxide, the oxygen content and the pH were determined in the arterial and mixed venous blood of 5 normal and 3 horses with chronic obstructive pulmonary disease (COPD) at 3 stages of an exercise distance of 1200 m. Arterial and mixed venous samples were collected simultaneously by means of an automatic technique during the walk, trot and gallop at 0-100 m, at 500-600 m and at 1100-1200 m.

The standard bicarbonate and the lactic and pyruvic acid concentrations were also determined in arterial and mixed venous blood.

Highly significant changes in the mean values of $P\bar{v}O_2$, $O_2$ content and $\Delta$ a-$\bar{v}$ $O_2$ content occurred during exercise in COPD subjects, and significant changes in $P\bar{v}O_2$ and $\Delta$ a-$\bar{v}$ $O_2$ content occurred during exercise in normal subjects.

We concluded that COPD subjects compensated for respiratory dysfunction during exercise by extracting more oxygen from the blood than did normal horses.

There was a highly significant correlation between the changes in standard bicarbonate and the changes in lactic acid concentration during exercise in both normal and COPD subjects. This led to the conclusion that lactic acid production was primarily, but not completely, responsible for the metabolic acidosis of exercise in horses.



ABSTRACT: Schneider, D.J., 1981. **First report of annual ryegrass toxicity in the Republic of South Africa.** *Onderstepoort Journal of Veterinary Research*, **48**, 251-255 (1981).

The occurrence of annual ryegrass toxicity (ARGT) in sheep and cattle is reported for the first time in South Africa. To date is has been diagnosed conclusively in South African Mutton Merino sheep on a farm in the Caledon district and in cattle on 3 farms, 2 of which are situated in the Bredasdorp district and 1 in the Ceres district. It is a neurological disease characterized by symptoms of tremor, ataxia, intermittent epileptiform seizures, nystagmus, opisthotonus, abortions and high mortality.

The history, clinical signs and experimental reproduction of the disease as well as the pathology of 4 experimental and 10 natural cases in sheep and 2 in cattle are described.



ABSTRACT: Fripp, P.J., 1981. **Non-specific esterase isoenzymes of adult schistosomes from the hippopotamus (*Hippopotamus amphibius*).** *Onderstepoort Journal of Veterinary Research*, **48**, 257 (1981).

Extracts of adult schistosomes collected from *Hippopotamus amphibius* in the Kruger National Park gave alpha naphthyl acetate isoenzyme patterns after polyacrylamide gel electrophoresis that did not correspond with those of either *Schistosoma mansoni* or *S. rodhaini*.



ABSTRACT: Herr, S., Pieterson, P.M. & Boshoff, J.A., 1981. **The relationship between the minimal and the 50 % haemolytic dose in complement titrations end-point ($C'H_{50}$)** was found to be more reproducible than the 100 % end-point. The relationship of the 100 % end-point (MHD) to the ($C'H_{50}$) was 1: 2,5. The use of thrombin tubes induced clotting within 2 min and enabled the process of complement preparation to be completed well within 1 hour.

# THE SEASONAL INCIDENCE OF HELMINTH PARASITES OF CATTLE IN THE EASTERN TRANSVAAL LOWVELD

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ABSTRACT: Malan F.S.; Reinecke R.K.; Roper N.A. **The seasonal incidence of helminth parasites of cattle in the eastern Transvaal Lowveld.** *Journal of the South African Veterinary Association* (1982) 53 No. 3, 179-184 (En) Hoechst Research Farm, P.O. Box 124, 1320 Malelane, Republic of South Africa.

Two helminth-free tracer calves were exposed to natural helminth infestation on a farm in the eastern Transvaal Lowveld for one month. On a nearby farm calves grazed with the herd for at least 3 months before slaughter so that the cumulative worm burden could be determined. The gastro-intestinal parasites increased markedly after rain. The major species and order or prevalence were: *Cooperia pectinata*, *Cooperia punctata*, *Haemonchus placei* and *Oesophagostomum radiatum*; minor species were *Bunostomum phlebotomum*, *Trichostrongylus falculatus*, *Nematodirus helvetianus*, *Trichuris* spp., *Parafilaria bovicola* and *Paramphistomum* spp. The highest worm burdens of *Cooperia* spp. on the first farm were between December and March and with the exception of *O. radiatum*, which had a peak in January, the other species reached peak worm burdens on the other farm in March.

Key words: cattle helminths, nematodes, epizootiology

## INTRODUCTION

Acocks[1] described the Lowveld as that area between the eastern foot of the Drakensberg and other mountains southward through Swaziland and Zululand and the western foot of the Lebombo lying 150-600 m above the sea. The vegetation is the "Characteristic open *Acacia nigrescens-Sclerocarya – Themeda* Savanna". *Panicum coloratum* grows under the *Acacia* spp., and *Eragrostis superba* and *Themeda* spp. are common.

We carried out a survey of common helminth parasites of cattle in this area and the results are presented in the present paper.

## MATERIALS AND METHODS

### Farms

Riverside: This farm is situated 25° 26′ S and 31° 35′ E 5 km north east of Malelane, the Crocodile River and Kruger Park forming the northern boundary (Fig. 1). About three quarters of the farm is fairly level and low lying. The climate is described by Acocks[1] as semi-arid with hot summers and mild winters. Mean annual rainfall ranges from 590 mm to 730 mm with a long term mean of 639 mm.

The animals drink water from irrigation canals running through the farm or from gravel dams receiving their water from these canals. Two-thousand-two-hundred ha are available for natural grazing. The farm is divided into 16 camps and a rapid rotational grazing system is followed i.e. the animals are kept in a camp for 3 days only and then moved to the next camp. This grazing system has been used for at least 8 years.

Te Kort: The farm is situated 25° 34′ S and 31° 43′ E 20 km south east of Riverside and consists of 1 200 ha divided into 20 camps (Fig. 1). Underground water is pumped into reservoirs which drain into concrete drinking troughs which are regularly cleaned. Furthermore there is one gravel dam, numerous streams running through the camps and vleis which are very wet in the rainy season. Animals graze for 2 weeks before they move to a new camp, but this is shortened to one week during the rainy season.

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### Experimental animals

Riverside: The breeding herd consist of 300 AfrikanerX BonsmaraXSimmentaler cows. All the calves were treated with fenbendazole, levamizole, resorantel and nitroxinul before the trial commenced but neither the breeding herd not the calves were treated with anthelmintics for the duration of the trial. The trial ran for 16 months, commencing in June 1980 and ending in October 1981.

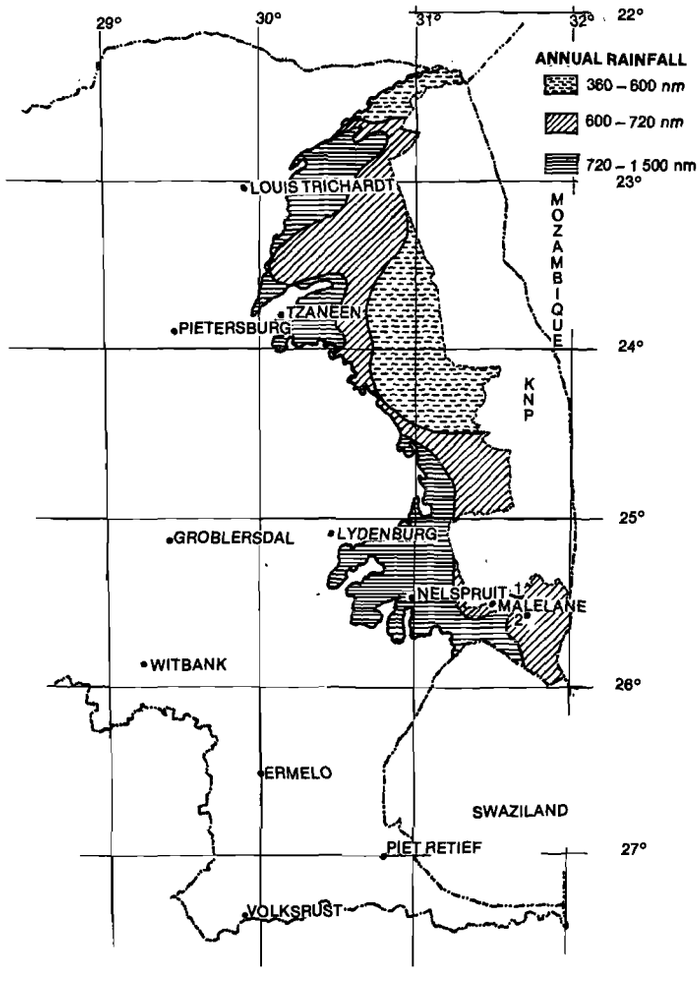


**Fig. 1** The Lowveld showing the rainfall distribution (Frans Ehlers with kind permission of Dept. of Agricultural Technical Services) ● 1 Te Kort 25° 34′ S and 31° 43′ E. ● 2 Riverside 25° 26′ S and 31° 35′ E.


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Two calves were slaughtered at intervals of 2,5-3,5 months, as follows: 13 October 1980, 7 January 1981, 24 March 1981, 7 July 1981 and 20 October 1981 respectively. The age of the animals at slaughter ranged from 8 to 14 months but the minimum period that any group of 2 calves was exposed to infestation was 4 months. These calves are hereafter referred to as 'grazers'.

Te Kort: The breeding herd consists of 300 AfrikanerXBonsmara cows. Fifty cows with their calves and 10 heifers, 18 months old were separated from the herd to infest the pasture and are referred to as 'seeders'. The trial ran from 12 June 1979 to 1 July 1980.

Two steer calves, about 5 months old, were treated with fenbendazole, levamizole, terenol and rafoxinide, housed worm-free in stables with concrete floors for one month and the put to graze for periods ranging from 24-42 days with the seeders. These calves known as 'tracers' returned to the worm-free stables for one month before they were slaughtered.

## Faecal examination

Faecal samples were taken every month from the seeders on the farm Te Kort and worm egg counts (epg) carried out.

## Helminth recovery

After the calves were slaughtered and the viscera removed, worms in the ingesta were killed with iodine solution, fixed with formalin and the ingesta washed with a strong stream of water on a sieve (150 $\mu$m apertures) placed on top of another sieve (38 $\mu$m apertures). The upper specimen was labelled residue and the lower specimen filtrate and after placing the specimens in jars, formalin was added as a preservative. Three 1/10 aliquots were made of each specimen from which worms were recovered with the aid of a sterioscopic microscope. The remaining 7/10 were examined macroscopically and all large worms recovered for identification. The mucosae of the abomasum, small and large intestines were removed by scraping for subsequent pepsin/HCI digestion. Three 1/10 aliquots were again examined with a stereoscopic microscope. Identification of adult species and fourth stage larvae ($L_4$) was carried out with the aid of a standard microscope[5].

In every autopsy the following were also examined: conjunctivae for *Thelazia rhodesii*; the carcass for *Parafilaria bovicola*; the fore-stomachs for adult *Paramphistomum* spp. and the liver for *Fasciola* spp.

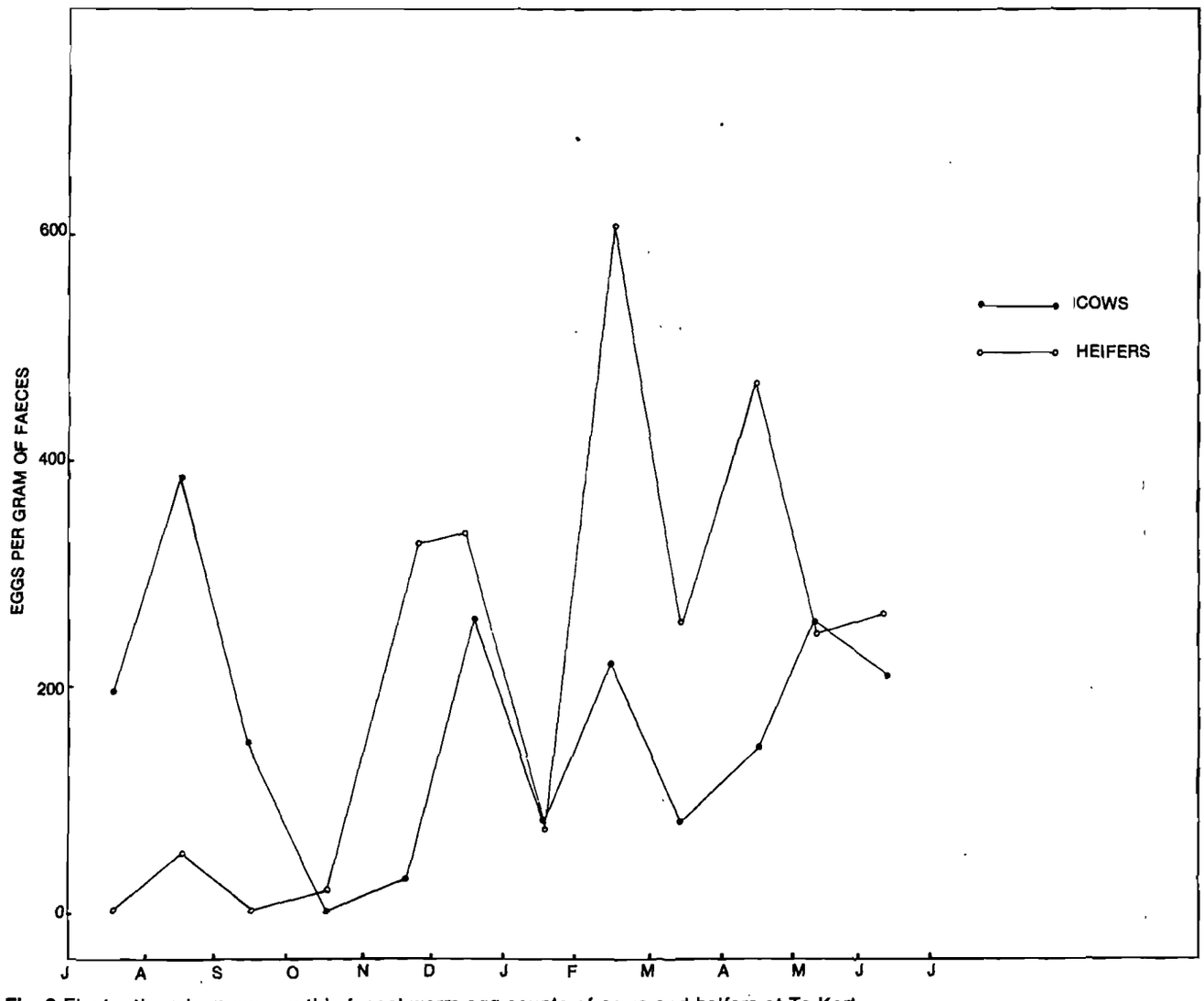


**Fig. 2** Fluctuations in mean monthly faecal worm egg counts of cows and heifers at Te Kort

## RESULTS

Faecal worm egg counts: Fluctuations in mean faecal worm egg counts (epg) are illustrated in Fig. 2.

Initially the heifers had lower egg counts than the cows but once the rains started egg counts in heifers exceeded those in cows. The highest mean count was 603 epg which was recorded in the heifers in February

Nematodes recovered at autopsy: The following major species recorded in Tables 1 and 2 were recovered in order of prevelance:

1. *Cooperia pectinata* and *Cooperia punctata*
2. *Haemonchus placei*
3. *Oesophagostomum radiatum*

The following were also present in individual calves in lower numbers:

1. *Bunostomum phlebotomum*
2. *Trichuris* spp.
3. *Paramphistomum* spp.
4. *Parafilaria bovicola*
5. *Trichostrongylus falculatus*
6. *Nematodirus helvetianus*
7. *Moviezia expansa*
8. *Stilesia hepatica*

Variations in worm burdens illustrated in Fig. 3 are described below

*Cooperia* spp.

Te Kort: These remained at a low level through the winter and spring rising to a minor peak in November to reach a maximum in January falling steadily thereafter to a low level in May.

Riverside: Worm counts rose steadily from October to March falling through the autumn to a lower level in the following October.

*H. placei*

Te Kort: Although less worms were present of this species the variations resembled those of *Cooperia* spp.

Riverside: This species also rose to a peak in March.

Table 1: TOTAL WORM BURDENS OF TRACER CALVES GRAZING FOR PERIODS OF ONE MONTH ON TE KORT

| Calf No. | Period exposed | *Cooperia* spp. Ad* | *Cooperia* spp. $L_4$** | *H. placei* Ad | *O. radiatum* Ad | *O. radiatum* $L_4$ |
|---|---|---|---|---|---|---|
| | 1979 | | | | | |
| 1 | 12 June-10 July | 904 | 0 | 11 | 33 | 0 |
| 2 | | 539 | 0 | 17 | 3 | 0 |
| 3 | 10 July-21 Aug | 34 | 0 | 11 | 0 | 0 |
| 4 | | 86 | 0 | 10 | 0 | 0 |
| 5 | 21 Aug-11 Sept | 173 | 0 | 9 | 3 | 0 |
| 6 | | 524 | 0 | 6 | 9 | 0 |
| 7 | 11 Sept-9 Oct | 10 | 0 | 0 | 2 | 0 |
| 8 | | 10 | 0 | 0 | 4 | 0 |
| 9 | 9 Oct-20 Nov | 115 | 0 | 19 | 32 | 0 |
| 10 | | 20 | 0 | 3 | 5 | 0 |
| 11 | 20 Nov-18 Dec | 383 | 0 | 570 | 629 | 0 |
| 12 | | 10 403 | 0 | 470 | 1 036 | 0 |
| 13 | 18 Dec-16 Jan 1980 | 830 | 0 | 283 | 117 | 3 |
| 14 | | 1 229 | 0 | 150 | 53 | 0 |
| | 1980 | | | | | |
| 15 | 16 Jan-20 Feb | 26 477 | 0 | 4 026 | 503 | 106 |
| 16 | | 9 140 | 3 | 677 | 273 | 43 |
| 17 | 20 Feb-20 March | 5 740 | 0 | 247 | 10 | 0 |
| 18 | | 9 893 | 0 | 386 | 33 | 0 |
| 19 | 20 March-24 April | 399 | 0 | 20 | 0 | 0 |
| 20 | | 1 026 | 0 | 20 | 6 | 0 |
| 21 | 24 April-20 May | 6 | 0 | 3 | 4 | 0 |
| 22 | | 13 | 0 | 0 | 1 | 0 |
| 23 | 20 May-1 July | 80 | 0 | 14 | 7 | 0 |
| 24 | | 0 | 0 | 0 | 7 | 2 |

Ad* = Adult
$L_4$** = Fourth stage larvae

Table 2: TOTAL WORM BURDENS OF TRACER CALVES GRAZING FOR PERIODS OF 4 MONTHS OR LONGER ON RIVERSIDE

| Calf No. | Day of slaughter | *Cooperia* spp. Ad* | *Cooperia* spp. $L_4$** | *H. placei* Ad | *O. radiatum* Ad | *O. radiatum* $L_4$ | *B. phlebotomum* Ad | *Trichuris* spp. Ad |
|---|---|---|---|---|---|---|---|---|
| 25 | 13 October 1980 | 177 | 0 | 6 | 11 | 0 | 10 | 10 |
| 26 | | 313 | 3 | 17 | 1 | 0 | 4 | 1 |
| 27 | 7 January 1981 | 1 650 | 0 | 180 | 255 | 0 | 43 | 3 |
| 28 | | 8 166 | 0 | 717 | 817 | 0 | 7 | 0 |
| 29 | 24 March 1981 | 12 839 | 0 | 740 | 823 | 13 | 90 | 1 |
| 30 | | 4 820 | 53 | 656 | 170 | 10 | 37 | 0 |
| 31 | 7 July 1981 | 2 640 | 49 | 17 | 37 | 0 | 0 | 7 |
| 32 | | 2 693 | 0 | 37 | 56 | 0 | 28 | 18 |
| 33 | 20 October 1981 | 2 810 | 13 | 337 | 83 | 3 | 5 | 0 |
| 34 | | 410 | 0 | 50 | 175 | 0 | 5 | 0 |

Ad* = Adult
$L_4$** = Fourth stage larvae

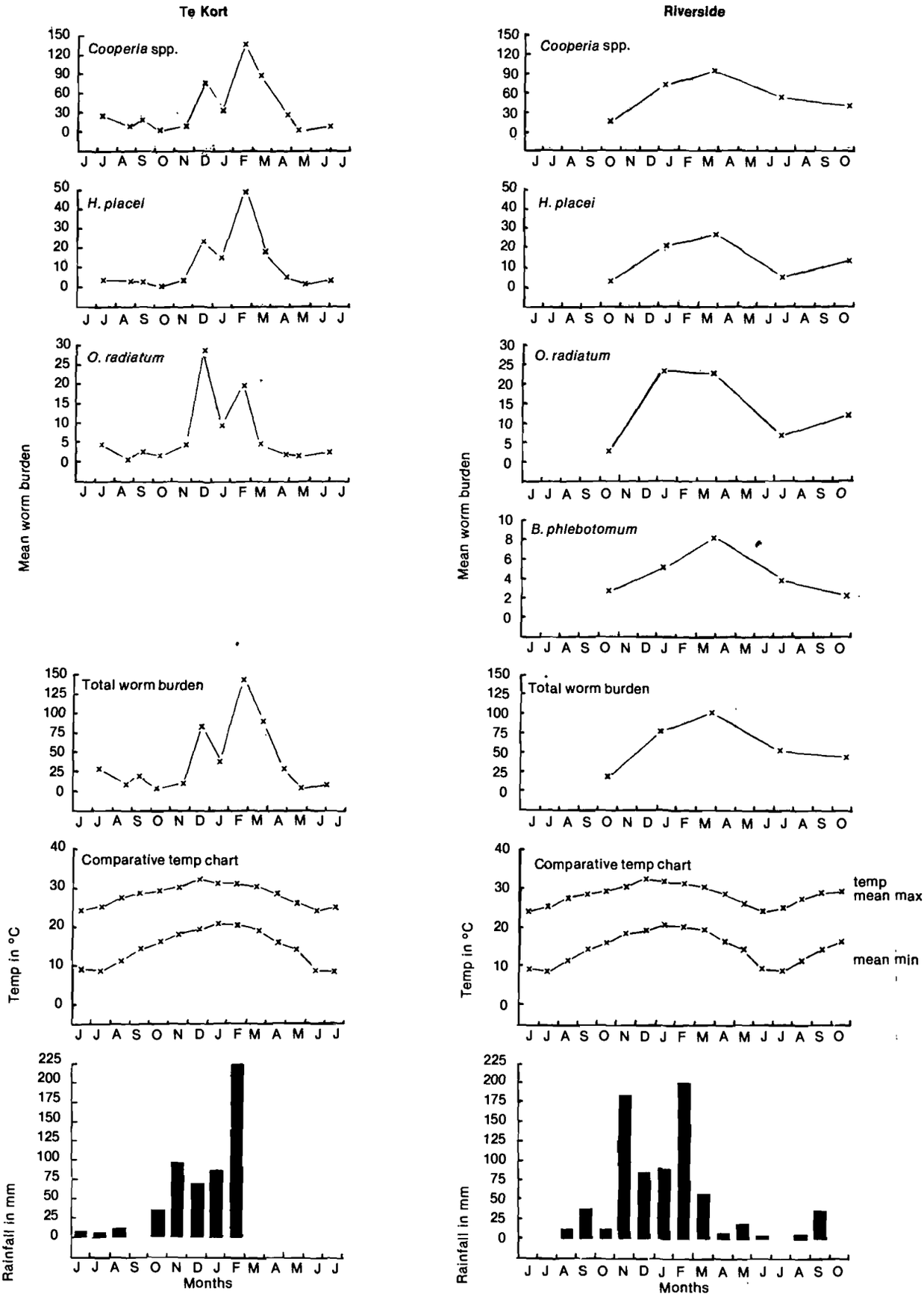


**Fig. 3** Variations in mean worm burdens, temperature and rainfall at Te Kort and Riverside

*O. radiatum*

Te Kort: From a low level in winter this species rose suddenly to a peak in November and December, falling in January, rising to a secondary peak in February and falling steadily thereafter throughout the autumn.

Riverside: Worm burdens in October were low, rose to a peak in January remaining high until March to fall in July, rising again in October.

*B. phlebotomum*

Te Kort: This worm was absent possibly due to the short exposure of the tracers to infestation.

Riverside: Low worm burdens in October followed by a steady rise through the summer to a peak in March and a marked fall thereafter to the lowest level the following October.

*T. falculatus*

Te Kort: Only 4 calves were infested with low numbers: 2 in November and December and 2 during the period February and March.

Riverside: In only one animal 36 worms were recovered in the period March to July.

*N. helvetianus*

Te Kort: This was present in only 2 calves, one of which had 3 worms and the other 13.

Riverside: In one calf only one worm was recovered.

*Trichuris* spp.

Te Kort: Absent.

Riverside: Four out of 10 calves were negative and in the others worms burdens ranged from 1 to 18. Seasonal incidence could not be determined with these low worm burdens but both calves slaughtered in July had 7 and 18 worms respectively.

On Riverside one calf had *M. expansa*, one calf *Stilesia hepatica*, 4 calves adult *Paramphistomum* spp. and in 3 calves *P. bovicola* were present. On Te Kort only 4 calves had *M. expansa*.

Rainfall figures are illustrated in Fig. 3. On Te Kort rainfall exceeding 25 mm fell from October 1979, to exceed 200 mm in February 1980 but was absent from March to May. No rain fell on Riverside in July 1981, but was recorded every month from August 1980 to the following June, exceeding 175 mm in November 1980 and February 1981.

## DISCUSSION

Schröder[6] stated that the use of tracer calves determined the availability of infective larvae on pastures. In the present trial the summer rains were the most important factor in the seasonal fluctuation of infestation on the herbage. At Te Kort worm burdens in tracers reached a maximum in January and February under the influence of good rains from November to February. The sharp fall in autumn was possible due to the complete lack of rainfall from March to May.

Careful scrutiny of the actual worm burdens summarised in Table 1 show that the apparent peak in January and February was due to the massive worm burdens of *Cooperia* spp. of 26 477 and 4 026 *H. placei* in Calf 15 which artificially boosted the mean worm burdens of the 2 calves slaughtered at that time. *Cooperia* spp. was at a high level in most calves slaughtered between December and March.

The data of the grazing calves on Riverside was influenced by rainfall in spring and summer. Peak worm burdens were recorded for *O. radiatum* in January but were delayed until March for the other major species. On this farm good rains were recorded from November to February which were probably translated to increase worm burdens in March (Fig. 3).

Decreased worm burdens in winter and the following spring were probably largely due to the very low rainfall. Mean monthly maximum temperatures were 24° to 25°C and mean minimum ranged from 8° to 9°C in June and July which should be adequate for most eggs to hatch and develop to the infective stage in the dung pad. Lack of rainfall however means that the infective larvae were trapped in the dung and could not migrate onto the grazing to infest the pasture[4].

Both the tracers at Te Kort and the grazing calves at Riverside confirm the observations of Horak[3] and Schröder[6] that the winter is unsuitable for cattle nematodes but differ from their observations in that the free living stages are present on the pastures in September and October in the Lowveld if early spring rains occur.

The Lowveld's climate is sub-tropical, the mean minimum temperatures being 2,5°C and the mean maximum is 37,5°C, with rainfall ranging from 360-600 mm in the most arid, rising to 720-1 500 mm in the most humid areas (Fig. 1). Data of our trials can be applied to most of the Lowveld.

Control measures must be a combination of veld management and efficient anthelmintics. Despite the fact that on Te Kort there were 20 camps and the cattle grazed for 2 weeks per camp, and on Riverside there were 16 camps with a short term grazing period of 3 days per camp, neither system was effective in controlling the worms.

On Riverside the most effective time of treatment was shown to be in June. The egg counts at Te Kort show that calves and weaners should not run with heifers, this probably also applies to weaned steers as they are the main source of contamination of the grazing.

Cows with calves at foot and recently weaned animals should be provided with 'safe grazing'. A safe pasture is one in which infestation is limited to the minimum. On a ranch this can be provided by first treating cows or oxen more than 2,5 years of age with anthelmintics and then allowing them to graze in at least 2 camps before placing young susceptible animals in them. Weaning takes place between April and July.

Treat heifers and steers at weaning and place heifers in one safe pasture and the steers in the other. This will extend the period of low infestation on the grazing but it may be wise to treat them again in January or February. This is probably not necessary for older cows and oxen.

ACKNOWLEDGEMENTS

We wish to thank Messrs Herman Kotze, Morgan Broughill-Dowling and Karino Farms Riverside (Pty) Ltd for making their farms available for this trial; the Underberg cattle study group for donating tracer calves; Mrs Nicky Nurse and Mr G. Shabangu for their assistance with the trial; Mrs Helen Viljoen for typing the manuscript and Mrs Ria van Zyl for preparing the graphs.

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ABSTRACT: Pitchford, R.J. & Visser P.S., 1981. ***Schistosoma* Wienland, 1858 from *Hippopotamus amphibius* Linnaeus, 1758 in the Kruger National Park.** *Onderstepoort Journal of Veterinary Research*, **48**, 181-184 (1981).

Adults of *Schistosoma edwardiense* Thurston, 1964, were recovered from *Hippopotamus amphibius* in the Kruger National Park. Small round to oval *Schistosoma margreboweie*-like eggs, presumed to be those of *S. edwardiense*, were found fairly frequently in the faeces of infected hippopotami together with a few *Schistosoma haematobium*-like eggs the identity of which remains uncertain.

*Biomphalaria* sp., exposed to the droppings of infected hippopotami, shed cercariae thought to be those of *S. edwardiense*. No evidence of schistosoma adults was found at necropsy in rodents exposed to these cercariae. The parasite appears to be host specific to the hippopotamus.

Arguments, based on biological and anatomical characteristics are put forward regarding *Schistosoma hippopotami* Thurston, 1963 as synonymous with *Schistosoma mansoni*.

ABSTRACT: Boomker, J., 1981 **The haemocytology and histology of the haemopoietic organs of South African freshwater fish. III. The leucocytes, plasma cells and macrophages of *Clarias gariepinus* and *Sarotherodon mossambicus*.** *Onderstepoort Journal of Veterinary Research*, **48**, 185-193 (1981).

The various leucocytes, plasma cells and macrophages are described and illustrated. Eosinophils and basophils are lacking in *Clarias gariepinus* but present in *Sarotherodon mossambicus*. The leucocytes of *C. gariepinus* resemble those found in mammals, as do the plasma cells and macrophages of both species. A possible mechanism for their formation is postulated.

ABSTRACT: Boomker, J., 1981. **The haemocytology and histology of the haemopoietic organs of South African freshwater fish. IV. Ultrastructure of some cells of *Clarias gariepinus* and *Sarotherodon mossambicus*.** *Onderstepoort Journal of Veterinary Research*, **48**, 195-205 (1981).

This paper describes the ultrastructure of several cells found in the blood and haemopoietic tissues of the catfish (*Clarias gariepinus*) and the Mozambique bream (*Sarotherodon mossambicus*). The cells are haemocytoblasts, small lymphoid haemoblasts, thrombocytes, monocytes, lymphocytes, developing and mature neutrophilic granulocytes, plasma cells and macrophages. The various organelles normally found in mammalian haemocytes, plasma cells and macrophages were observed in those of fish. With the exception of the mature erythrocytes, which resemble the normoblasts of mammals, the various cells show distinct similarities of those of other fish species as well as of mammals.

ABSTRACT: Nevill, E.M., 1981 **The development of *Parafilaria bovicola* in *Musca xanthomelas* and *Musca lusoria*.** *Onderstepoort Journal of Veterinary Research*, **49**, 207-213 (1981).

Artificially infected adult flies were used in this study. In both *Musca* species, *P. bovicola* developed in the fat-body cells, mainly of the abdomen. Escape from the midgut and penetration of the fat-body cell was possibly achieved by use of the cephalic hook of the microfilaria. At 27°C, development to the 3rd larval stage took 9 days, but maximum length was only reached after 11 days. Sharp increases in larval length took place on Days 6-7 and on Day 9. These sudden increases possibly indicate moults to the 2nd and 3rd larval stages respectively.

During larval development the fat-body cell increased markedly in size to form a thin-walled capsule around the larva. On reaching the 3rd stage, larvae escaped from the capsules and migrated to the head cavity and proboscis.

ABSTRACT: Gray, J.S. & De Vos, A.J., 1981. **Studies on a bovine *Babesia* transmitted by *Hyalomma marginatum rufipes* Koch, 1844.** *Onderstepoort Journal of Veterinary Research*, **48**, 215-223 (1981).

A *Babesia* sp. was recently observed in *Hyalomma marginatum rufipes* and found to be transmissible to bovines. Further observations were carried out on this parasite and a study made of the morphology of stages in both erythrocytes and tick haemolymph. Apart from *Babesia divergens*, intra-erythrocytic parasites were not readily distinguishable from bovine *Babesia* spp. Merozoites in tick haemolymph morphologically resembled those of *Babesia bigemina*, but they were significantly larger. This *Babesia* sp. proved to be highly infective for adult *H. m. rufipes*, with transmission taking place transovarially and next generation nymphae and adults transmitting the infection.

Features of the infection were its vey low pathogenicity, even in splenectomized animals, and the tendency of parasitized erythrocytes to accumulate in capillaries.

Serologically, this species could be differentiated from *Babesia bigemina*, *B. divergens*, *B. bovis* and *B. major*. A serological survey of 25 farms showed a wide distribution of this species in South Africa and its high rate of transmission on most properties. It was concluded that this is a true but hitherto undescribed bovine *Babesia* sp. and the name *Babesia occultans* n. sp. is proposed.

# ANTHELMINTIC EFFICACY OF FENBENDAZOLE IN DONKEYS ASSESSED BY THE MODIFIED NON-PARAMETRIC METHOD

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ABSTRACT: Malan, F.S.; Reinecke, R.K.; Scialdo-Krecek, R.C. **Anthelmintic efficacy of fenbendazole in donkeys assessed by the modified non-parametric method.** *Journal of the South African Veterinary Association* (1982) 53 No. 3 185-188 (En) Hoechst Research Station P.O. Box 124, 1320 Malelane, Republic of South Africa.

Eleven donkeys were each given a single oral dose of fenbendazole (FBZ) at 30 mg/kg body mass and a further 7 were left as untreated controls. Controls were killed 3 to 11 days later and treated donkeys 28-38 days after treatment. The modified non-parametric method (NPM) was used to assess anthelmintic efficacy. Efficacy against adult *Habronema* spp., *Strongylus vulgaris* and *Triodontophorus* spp. was more than 80 % in more than 80 % of animals (Class A). Against the sub-family Cyathostominae efficacy was only Class B, ie > 60 % effective in > 60 % of animals, because there was a delay of 28 to 38 days between treatment and slaughter compared with th controls killed from 3-11 days after treatment. More than 95 % of the Cyathostominae, however, were 5th stage worms and not mature adults. Reasons for these findings are discussed.

Key words: Fenbendazole, anthelmintic, equine nematodes, *Strongylus vulgaris*, Cyathostominae.

## INTRODUCTION

Differential faecal worm egg counts in which egg counts are combined with larval cultures give a rapid, economical assessment of anthelmintic efficacy and were used by Taylor & Sanderson[16] in anthelmintic trials with phenothiazine in horses. McBeath et al.[12] treated naturally infested horses with fenbendazole (FBZ) at doses ranging from 7,5-60 mg/kg live mass and compared their faecal worm egg counts with those in undosed controls every week for 18 weeks. Egg counts were negative in treated horses for 6-8 weeks after treatment. Urch & Allen[17] treated ponies, donkeys and donkey foals with FBZ at 7,5 mg/kg and carried out examinations on the faeces 14 and 28 days after treatment for eggs and for first stage larvae of *Dictyocaulus arnfield* from 7-28 days after treatment. They concluded that although FBZ at 7,5 mg/kg was effective against strongyles, *Parascaris equorum* and *Strongyloides westeri*, it had no effect against *D. arnfieldi*, even at doses as high as 30 mg/kg.

Malan et al.[11] treated horses with FBZ paste at 7,5 mg/kg and then carried out differential faecal egg counts every 7 days for 9 weeks. In addition, all faeces were collected for 7 days after treatment and aliquots examined for expelled worms. This test, which is referred to as the faecal worm count method, confirms that worms are expelled when egg counts become negative. Although fourth stage larvae ($L_4$) of Cyathostominae (*Trichonema*) were present in the faeces, this method cannot be used to assess anthelmintic efficacy against these $L_4$ because the numbers of larvae still retained in the gut wall of the host are unknown.

In the critical test developed by Hall & Foster[7], the entire faecal output of treated animals is collected daily for 4 days and the expelled worms counted. The animals are then killed, the remaining worms counted and the efficacy expressed as follows:

$$\text{Percentage efficacy} = \frac{\text{the number of worms expelled}}{\text{the total number of worms remaining}} \times 100$$

The principal advantage of this method is that each animal acts as its own control and untreated controls are unnecessary. It is generally conceded that the critical test is the best anthelmintic test for horses. In critical tests carried out by Drudge et al.[1] with FBZ at 5 mg/kg in 7 naturally infested horses, adult worm burdens of common nematode parasites were reduced by 86-100 %.

Malan & Reinecke[8] dosed 10 donkeys with FBZ at 10 mg/kg and subsequently Malan et al.[11] gave 5 horses 7,5 mg/kg of FBZ paste; their results confirmed those of Drudge et al[1].

Since Malan et al.[11] were only able to demonstrate an efficacy of 5-38 % against $L_4$ Cyathostominae with FBZ paste at 7,5 mg/kg they considered that no reliance could be placed on critical tests against larval stages.

Moskey & Harwood[13] described the controlled anthelmintic test in which one group of artificially infested animals are treated and another group remain as untreated controls. Both groups are killed 2 weeks after treatment. The worms remaining in the controls are regarded as the probable number of worms that would have remained without treatment while those remaining in the treated groups are worms unaffected by the compound. From these data anthelmintic efficacy is estimated as follows:

$$\text{Percentage efficacy} = \frac{\text{Mean number of worms in controls} - \text{mean number in treated animals}}{\text{mean number of worms in controls}} \times 100$$

This is the best anthelmintic test for larval stages in the gastro-intestinal tract, even those whose habitat is the caecum and colon, regardless of whether they are situated in the gut wall or in the lumen[9 10 14]. Worms such as *Strongylus vulgaris* in the arteries and *Strongylus edentatus* in the subperitoneal tissues of the abdominal wall can only be evaluated by a test of this nature.

Duncan et al.[3] treated 3 groups of 6-12 month old naturally infested ponies with FBZ at either 15, 30, or 60 mg/kg and compared them with 4 undosed controls. This controlled test with 30 mg/kg or higher removed 93 % $L_4$ of Cyathostominae, while 60 mg/kg were necessary to remove 83 % and 89 % $L_4$ of *S. vulgaris* and *S. edentatus*, respectively. Subsequently Duncan et al.[4] separated 8 naturally infested ponies into a group of 4 animals treated with 7,5 mg/kg of FBZ daily for 5 days and a control group of 4 untreated ponies. The percentage reduction in $L_4$ Cyathostominae was 94,6 %, was 80 % against larvae of *S. vulgaris* but 100 % against larvae of *S. edentatus*.

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The markedly skew distribution of the results of Duncan at al.[3] suggested that a non-parametric method be used and we analyzed their results by the Mann-Whitney U test as described by Siegel[15]. When FBZ was dosed at 30 mg/kg the significance by the Mann-Whitney test was:

$L_4$ *S. vulgaris* $p = 0,029 = 97,1$ %
$L_4$ *S. edentatus* $p = 0,057 = 94,3$ %
$L_4$ Cyathostominae $0,100 > p > 0,171 = 82,9 - 90$ %
(*Trichonema*)

In other words the levels of significance are 97,1 %, 94,3 % and 82,9-90 % for $L_4$ of *S. vulgaris*, *S. edentatus* and Cyathostominae, respectively. This paper reports the results of a trial with FBZ at 30 mg/kg in naturally infested donkeys and illustrates the value of a non-parametric test (NPM) to assess anthelmintic efficacy.

## MATERIALS AND METHODS

Eighteen donkeys were bought in Sekukuniland, Transvaal. They were kept on a concrete floor, each in a separate pen which was scrubbed daily. Hay free of equine strongyle larvae and water were supplied *ad libitum*. The body mass of each donkey was determined before treatment. Eleven donkeys were treated with 10 % FBZ solution at a dosage rate of 30 mg/kg body mass *per os* with a stomach tube and 7 animals were left as untreated controls.

Controls were slaughtered between Day +3 and Day +11; treated animals were slaughtered from Day +28 to Day +38. The methods described by Malan et al.[9 10] were used for worm recovery.

### Post Mortem Examination

All the helminths and *Gasterophilus* larvae in the stomach and small intestinal ingesta were counted, whereas only ¼ by mass of the ingesta of the caecum, ventral and dorsal colon were collected for worm counts.

The stomach wall was examined and palpated for nodules caused by *Draschia megastoma*. When present, the nodules were incised and the worms were removed. The glandular mucosa and muscularis layers of the stomach were scraped from the serosa and digested for 1,5 h in pepsin/HCl[15]. The walls of the small intestine, caecum, ventral and dorsal colon were processed similarly but only ¼ by mass of each part of each organ was digested for 2 h.

In addition the aorta and its major branches were opened and the fourth stage larvae ($L_4$) of *S. vulgaris* removed. Particular attention was paid to the cranial mesenteric artery and its branches. Fourth moults were included in $L_4$ counts.

The efficacy was assessed by the NPM (C.J. Clark, 1969, Imperial Chemical Industries, Macclesfield, Cheshire, personal communication cited by Reinecke[14]). Clark's modification can be summarized as follows:

1. The median rather than the mean is used to indicate the worm burdens of the controls.
2. Simulation studies have shown that if this median is reduced by 75 % after treatment there is no chance that compounds which produce an 80 % reduction in worm burdens (or less) in 80 % of the treated group will be graded Class A.
3. At the 90 % confidence limit when 11 animals are treated the gradings are as follows:

**Class A:** more than 80 % effective in more than 80 % of the treated group. This is estimated by multiplying the control median by 0,25 and only one of 11 treated animals may exceed this figure.

**Class B:** more than 60 % effective in more than 60 % of the treated group, which is estimated by multiplying the control median by 0,4. Three out of 11 treated animals may exceed this figure.

**Class C:** more than 50 % effective in more than 50 % of the treated group, which is estimated by multiplying the control median by 0,5 and 4 out of 11 treated animals may exceed this figure.

**Class X:** Ineffective.

## RESULTS

The numbers of worms recovered are summarized in Tables 1, 2 and 3.

### Cyathostominae

$L_4$: Worm recoveries from the controls varied from 596 to 16 324 and from the treated animals from 1 211 to 19 460. The 5th stage and adult worms in the controls varied from 7 509 to 93 734 and in treated animals ranged from 60 to 19 699.

*Anthelmintic efficacy (Table 1)*

On statistical analyses of $L_4$ of Cyathostominae, all 11 donkeys had more worms than the reduced median for Class C and the compound was ineffective (Class X), but the efficacy rose to Class B (>60 % effective in >60 % of treated animals) against the 5th and adult stages.

### *Strongylus vulgaris*

$L_4$ (intravascular): The median burden of the 7 control donkeys was 18 (range 0-43). The burdens recovered from the treated animals ranged from 0-277.

Table 1: ANTHELMINTIC EFFICACY AGAINST CYATHOSTOMINAE ASSESSED BY THE NPM

| $L_4$ Cyathostominae | | Fifth stage and Adult Cyathostominae | |
|---|---|---|---|
| Control | Treated | Control | Treated |
| 596 | 1 211 | 7 509 | 60 |
| 601 | 1 777 | 12 899 | 543 |
| 1 202 | 1 782 | 18 901 | 1 044 |
| **1 444** | 1 966 | **30 889** | 2 633 |
| 3 128 | 2 454 | 36 855 | 4 536 |
| 4 068 | 4 786 | 60 657 | 6 942 |
| 16 324 | 6 397 | 93 734 | 9 091 |
| | *10 297* | | *10 013* |
| | 12 076 | | 11 511 |
| | 16 251 | | 12 510 |
| | *19 460* | | 19 699 |
| Mean 3 909 | 7 132,5 | 37 349 | 6 949,1 |
| Median 1 444 | — | 30 889 | — |
| 1 444 × 0,5 = 722 | 11/11 exceed 722 | 30 889 × 0,25 = 7 222,25<br>30 889 × 0,4 = 12 355,6 | 2/11 exceed 12 355,6 |
| | Class X | | Class B |

Table 2: ANTHELMINTIC EFFICACY ASSESSED AGAINST $L_4$ AND 5th STAGES OF *S. VULGARIS* IN THE ARTERIES AND ADULTS IN THE INGESTA OF THE CAECUM AND COLON

| $L_4$ intravascular | | 5th stages intravascular | | Adults in the ingesta | |
|---|---|---|---|---|---|
| Controls | Treated | Controls | Treated | Controls | Treated |
| 0 | 0 | 0 | 5 | 0 | 0 |
| 0 | 0 | 0 | 8 | 31 | 0 |
| 14 | 0 | 2 | 9 | 53 | 0 |
| 18 | 18 | 7 | 11 | 56 | 0 |
| 25 | 26 | 8 | 12 | 89 | 1 |
| 37 | 32 | 24 | 17 | 92 | 1 |
| 43 | 38 | 52 | 20 | 134 | 2 |
| | 50 | | 24 | | 4 |
| | 88 | | 49 | | 6 |
| | 150 | | 68 | | 7 |
| | 277 | | 91 | | 28 |
| Mean 19,6 | 61,7 | 13,3 | 28,5 | 65 | 4,5 |
| Median 18 | — | 7 | — | 56 | — |
| 18 x 0,5 = 9 | 8/11 exceed 9 Class X | 7 x 0,5 = 3,5 | 11/11 exceed 3,5 Class X | 56 x 0,25 = 14 | 1/11 exceed 14 Class A |

Table 3: ANTHELMINTIC EFFICACY AGAINST ADULT *HABRONEMA* SPP.

| *Habronema* spp. Adult | |
|---|---|
| *Controls* | *Treated* |
| 0 | 0 |
| 0 | 0 |
| 21 | 0 |
| 134 | 0 |
| 290 | 0 |
| 406 | 0 |
| 409 | 0 |
| | 0 |
| | 0 |
| | 0 |
| | 3 |
| Mean 180 | 0,3 |
| Median 134 | — |
| 134 x 0,25 = 33,5 | 0/11 exceed 33,5 Class A |

5th Stage (intravascular): Worm recoveries from the controls ranged from 0-52 (median 7) and from the treated animals from 5-91.

Adults (ingesta): The median worm burden of the controls was 56 (variation 0-134) and the range in the treated animals was 0-28.

*Anthelmintic efficacy (Table 2)*

FBZ was ineffective against $L_4$ and 5th stage of *S. vulgaris* in the arteries but attained Class A (>80 % effective in >80 % of donkeys) against adults in the caecal and colonic ingesta.

### *Habronema* spp.

Adults: A median burden of 134 (range 0-409) was found in the controls. One donkey in the treated group had 3 worms and the rest none.

Four of 7 controls had from 6-188 $L_4$ *H. majus* while the others had O. The numbers of positive donkeys was less than 5 and the worm burdens in them too low to use for the NPM. Only 2 treated animals had 1 and 26 $L_4$ and the rest were negative.

*Statistical efficacy (Table 3).*

FBZ attained Class A against adults.

## DISCUSSION

The number of worms in a group of animals do not have a normal distribution. Egerton et al.[5] used transformations to overcome this problem but this is not acceptable because the distribution in treated animals differs from that in the controls. Groeneveld & Reinecke[6] developed a non-parametric method of evaluating anthelmintics which makes full allowance for this markedly skew distribution in both treated and control animals. Moreover, the effect of the anthelmintic can be analyzed simultaneously in two different ways:

(a) The percentage reduction of worm burdens and
(b) the percentage of the treated group in which the anthelmintic is effective.

This method was subsequently modified and is known as the modified non-parametric method (NPM). It required a minimum of 5 control and 11 treated animals[14] and is described in the materials and methods.

Drudge[2] stated that a single dose of FBZ of 60 mg/kg body mass or 2 daily doses of 50 mg/kg body mass was not effective in yearling horses that were naturally infested with $L_4$ and 5th stages of *S. vulgaris* in the arterial system. We have confirmed his observations and not those of Duncan et al[3] with the NPM in donkeys for these stages in the arterial system.

In the present trial FBZ at 30 mg/kg was ineffective (Class X) against $L_4$ and it only rose to Class B against 5th and adult stages of Cyathostominae. In critical tests with donkeys and horses, using only 5 animals we showed that FBZ at dosage rates of 10 mg or 7,5 mg/kg live mass was ineffective against $L_4$ but rose to >95 % effective against 5th and adult Cyathostominae. The reasons for the poor results in the present trial (30 mg/kg) compared with previous trials at much lower doses, may be due to various factors.

The basic requirements for tests against larval stages are: accurate knowledge of the parasite life cycle, pure infective larvae of the species under test, and treatment aimed against one larval stage and the subsequent moult only[14].

In equines, unfortunately it is not possible to fulfil the basic requirements of the NPM and use had to be made of naturally infested animals, in which neither the range

of parasites nor the number and stage of their development nor the total worm burden are known.

Whether we slaughtered the controls on the day of treatment or 3-11 days after treatment their worm burdens would not be altered, particularly if the hay that was fed was worm-free and the pen cleaned daily.

The reasons for delaying slaughter in treated donkeys was that we had to allow:

(i) sufficient time for the complete disintegration of the $L_4$ and 5th stage *S. vulgaris* killed by FBZ and
(ii) the disappearance of larvae in the caecal and colonic wall affected by treatment with FBZ.

Moreover it took us a full day to perform a complete necropsy on 1 donkey and 18 donkeys therefore required 18 working days. As a result it was impossible to kill all the donkeys on the same day.

It was difficult to assess the efficacy of FBZ against Cyathostominae in the gut wall. If the donkeys were killed within 5-10 days of treatment only $L_4$ would have been present in the gut wall of most of them. Doses as low as 5 mg, 7,5 mg and 15 mg/kg respectively removed 86-100 % of 5th and adult Cyathostominae from the gut lumen (Drudge et al.[1], Malan et al.[11], Duncan et al.[3]).

We therefore postulate that the 5th and adult stages that survived treatment were probably $L_4$ (in the gut wall) on the day of treatment. When naturally infested donkeys are used, therefore, the treated animals should be killed within 7-10 days of treatment in order to assess the efficacy of the drug against larval stages.

It must be emphasized that the present results were obtained in donkeys; horses may react differently.

In the present trial we digested the caecal and colonic wall to release the larval stages of the Cyathostominae and in the process probably destroyed many of them. Malan et al.[10] counted larvae in the gut wall with the aid of an illuminated magnifying glass (diamond sorting glass). Subsequently we scraped the same mucosa and digested it in pepsin and 3 % 10 N HCl at 37°C in a waterbath for 2 hours. We then sieved the digest on a fine sieve (apertures 38 $\mu$m), recovered the larvae from the sieve's surface and examined the digest with a stereoscopic microscope. When we compared the counts by these 2 methods we had 40 % more worms in the undigested gut wall than in the digests. In retrospect the data on larvae released from the gut wall after digestion should be treated with reserve.

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# OXFENDAZOLE: ANTHELMINTIC ACTIVITY IN CALVES ARTIFICIALLY INFECTED WITH SIX SPECIES OF NEMATODES AND IN CALVES NATURALLY INFESTED WITH TAPEWORMS

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ABSTRACT: Berger J.; Tema B.O. **Oxfendazole: Anthelmintic activity in calves artificially infected with six species of nematodes and in calves naturally infested with tapeworms.** *Journal of the South African Veterinary Association* (1982) **53** No. 3, 189-194 (En) Kwanyanga Research Station, Coopers (South Africa) (Pty) Ltd., Greenfields, 5208 East London, Republic of South Africa.

In a series of 5 trials, in which 115 artificially infected calves were used, larval anthelmintic tests were done to assess the activity of oxfendazole at 4,5 mg/kg against all 3 parasitic stages of 6 species of namatodes. An efficacy of over 98 % was demonstrated against the 4th stage larvae ($L_4$), 5th and adult stages of *Haemonchus placei, Ostertagia ostertagi, Cooperia pectinata, Bunostomum phlebotomum* and *Oesophagostomum radiatum* and the 3rd larval stage ($L_3$) of *H. placei* and *C. pectinata*. Against the $L_3$ of *O. ostertagi, B. phlebotomum* and *O. radiatum* efficacies of 81,7 %, 92,4 % and 97,3 % respectively were shown. Against *Dictyocaulus viviparus* adult, 5th, $L_4$ and $L_3$ efficacies were recorded of 100 %, 96,7 %, 95,4 % and 89,1 % respectively. A test for ovicidal activity confirmed that cattle can be safely moved to clean pasture 8 hours after dosing. Complete removal of strobila of *Moniezia benedeni* from 25 naturally infested calves followed a dose rate of oxfendazole at 4,5 mg/kg, but of these each of 4 calves retained one scolex.

Keywords: oxfendazole, anthelmintic, calf, nematode, cestode.

## INTRODUCTION

The very high degree of anthelmintic activity exerted by oxfendazole at a dose rate of 5 mg/kg against 8 of the sheep nematode species of special importance in the Republic of South Africa has already been recorded[1].

The trials reported here were designed to record, in larval anthelmintic tests[4], the efficacy of a standard dose of oxfendazole at 4,5 mg/kg against all 3 parasitic stages of 6 important nematode species of cattle. To confirm the satisfactory efficacy results reported elsewhere[3] a critical trial was also included to record the results of treatment, with the same dose rate, of calves naturally infested with *Moniezia spp.*

## MATERIALS AND METHODS

### Experimental calves.

(a) *nematode trials*

In 5 trials a total of 115 Friesian calves were used aged 2,5 – 4,5 months at time of artificial infection and 4-6 months at slaughter. All calves, having been housed and reared in crates since birth, free from helminth infestation, were also dosed parbendazole at 60 mg/kg one week prior to artificial infection. During the course of the trials they were kept on slatted floors and were fed a lucerne chop and pellet concentrate ration.

Only male calves were used in Trials 1, 2 and 3 and of the 52 calves used in Trials 4 and 5 only 6 were female.

(b) *cestode trial*

Twenty eight calves, 3-6 months old, carrying natural infestations of *Moniezia benedini* were brought to the laboratory from 4 local farms. Apart from 3 Jersey calves all others were cross-bred beef cattle and only 7 were females. They were restrained in yokes and fed lucerne chop until slaughtered.

### Experimental infections

Donor calves carrying fully susceptible laboratory strains of *Haemonchus placei, Ostertagia ostertagi, Cooperia pectinata, Bunostomum phlebotomum, Oesophagostomum radiatum* and *Dictyocaulus viviparus* were the source of infective larvae. The *Cooperia* spp. infection carried for many years in donor calves originally was mixed *C. pectinata* and *C. punctata* but the latter species has died out. The larval doses and dosing regimens used in the larval anthelmintic tests are included in Table 1. Larvae suspended in water were given by tube into the oesophagus, each total dose comprising divided daily doses within the prescribed period for the particular larval stage of the species concerned. The total dose of *B. phlebotomum* infective larvae was applied dermally on one day to 2 areas of clipped skin as previously described[5].

### Calf groups

(a) *nematode trails*

The results of the larval anthelmintic test were assessed against each stage of each nematode species using 11 treated and 9 untreated control calves, except in the case of Trial 5 in which there was a common control group of 7 calves to the 2 groups each of 11 treated calves. In addition one larval indicator control (LIC) was slaughtered on the day of treatment of each group to show the distrubution and degree of development of the various parasitic stages at that time.

Calves were selectively allocated to give a comparable live mass range in treated and control groups.

(b) *cestode trial*

In the first stage of this trial 12 treated and 3 untreated calves were slaughtered and when the result had been recorded a further 13 treated calves were slaughtered. Calves (with the exception of No. 923) were only included in the trial if *Moniezia* spp. eggs were found in faeces samples taken on the farm and again at the laboratory.

### Dosing procedure

Each calf in the treatment group was dosed with a 9,06 % m/v aqueous suspension of oxfendazole at 4,5 mg/kg, given from small volume dosing gun calibrated to the nearest 0,2 ml.

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## Autopsy and worm recovery

(a) *nematode trials*

Treated and control calves were slaughtered when infections should have reached maturity or near maturity.

Procedures adopted conformed to those described for larval anthelmintic tests in ruminants suitable for analysis by a modified non-parametric method of evaluation[2]. Whenever appropriate, peptic digests of viscus wall were carried out to ensure recovery of both migrating or inhibited larvae. Lungs were processed in the first 4 trials for the recovery of immature or adult *D. viviparus* and in trial 5 LIC calf for immature *B. phlebotomum*. Where there was a possibility of the presence of $L_3$ or $L_4$ *D. viviparus* the gastric, mesenteric and lung lymph nodes were also processed and additional search for $L_3$ was made in the lymph nodes of the head, pancreas, liver, kidney, spleen and blood of the LIC in Trial 2.

In recording worm burdens recovered at autopsy any worms still in the previous larval stage to that under scrutiny were not included in the total. Hence in Trial 5 the total number of *O. ostertagi* recorded in the same group of control calves are marginally less for the 5th stage/adult assessment than in the case of the $L_4$ stage.

(b) *cestode trial*

Within 3 h of treatment faecal bags were attached, the contents were removed twice daily for 3 d and the entire mass of faeces washed in a 250 μm mesh sieve prior to preservation with formalin. The total volume of evacuated segments, recovered in black trays, was recorded.

All calves were slaughtered 5 to 9 d after treatment. In processing the small intestine the mucosa was washed vigorously in physiological saline 3 times using finger pressure and the contents were cleaned in a 75 μm mesh sieve and preserved with formalin. To ensure recovery of any residual scolices only very small samples of gut content were scrutinised at a time in black trays.

## Assessment of ovicidal activity

Immediately prior to treatment (T) faeces samples from the 11 treated and 3 of the control calves in Trial 3 were taken for egg count and culture and again at T + 1, 3, 5, 8, 10 and 24 h post-treatment. From each sample an estimate was made of the total number of infective larvae recovered from 30 g of faeces and a percentage differential count carried out.

## RESULTS

The results of the larval anthelmintic tests have been summarised in Table 1, which also records the worm recoveries from the LIC calves and the anthelmintic efficacy classification evaluated by the modified non-parametric method[2].

Oxfendazole at 4,5 mg/kg had an efficacy of over 98 % against $L_4$, 5th and adult stages of all 5 gastrointestinal species and against $L_3$ *H. placei* and *C. pectinata*. Against $L_3$ *O. radiatum, O. ostertagi* and *B. phlebotomum* efficacy rates of 97,3 %, 81,7 % and 92,4 % respectively were recorded. The lung species *D. viviparus* also proved highly susceptible to this dose rate, efficacies of 100 %, 96,7 % 95,4 % and 89,1 % being recorded against adult, 5th, $L_4$ and $L_3$ stages respectively.

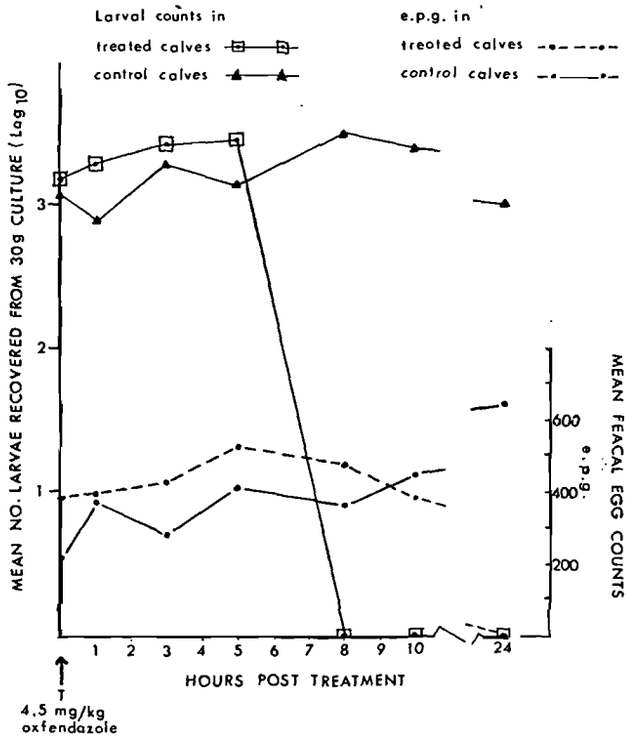


**Fig. 1:** Mean egg counts and counts of infective larvae recovered from cultures made at time of treatment and at 1 to 24 hours post-treatment.

The results of the test of ovicidal acitivity are illustrated in Fig. 1. Up to 5 h after dosing the viability of eggs of all 3 species proved unaffected. By 8 h after dosing 8 of the 10 samples cultured yielded no larvae, a total of only 6 larvae being recovered from the remaining 2, indicating that at this point faeces no longer carried significant numbers of viable eggs. Although egg counts were still high 10 h post-treatment all cultures proved negative and 14 h later egg counts had also become negative.

The result of the cestode trial is summarised in Table 2. There was no evidence that infestations were other than *M. benedeni*. The character of the eggs seen in all faeces samples prior to treatment and those released from evacuated segments after treatment, and the worms recovered from untreated controls, all conformed to that of this species. The 3 untreated calves carried a mean number of 3 tapeworms having a mean volume of 57 ml of strobila. In the treated calves expulsion of segments was largely completed within 48 h of dosing. Twenty-one of the 25 treated calves were completely cleared of tapeworms and a single scolex was found in each of the other 4, indicating complete destrobilisation in 100 % and total clearance of cestodes in 84 % of the calves.

## DISCUSSION

In all the nematode efficacy trials satisfactory mean takes allowed valid assessment. Oxfendazole at 4,5 mg/kg proved to have a high degree of activity against all stages of cattle nematodes comparable to that shown by 5 mg/kg against sheep nematodes[1]. The 5th stage/adult worms of all 6 species tested were completely or nearly completely eliminated.

During the course of the sheep trials previously

Table 1: LARVAL INFECTION RATES, LARVAL AGE AT TIME OF TREATMENT AND SLAUGHTER, RANGE IN NUMBER OF WORMS RECOVERED FROM 9 UNTREATED CALVES AND 11 CALVES DOSED OXFENDAZOLE AT 4,5 mg/kg. PERCENTAGE REDUCTION IN WORM BURDENS AND EFFICACY CLASSIFICATION.

(a) *Haemonchus placei*

| | $L_3$ (Trial 2) | | $L_4$ (Trial 1) | | 5th/Adult (Trial 3) | |
|---|---|---|---|---|---|---|
| Larval dose[1] | 4830 (2 × 2415) | | 4950 (12 × 412) | | 4960 (16 × 310) | |
| Larval age[2] (days) | 1 – 2 | | 3-14 | | 15 – 30 | |
| LIC No. worms<br>calf stage % | 905<br>$L_3$94 % | | 2970<br>$L_3$1 % $L_4$70 % 5th 29 % | | 2911<br>5th/Ad 100 % | |
| | CONTROL | TREATED | CONTROL | TREATED | CONTROL | TREATED |
| Slaughter age[3] (days) | 41 – 44 | 40 – 42 | 29 – 46 | 35 – 48 | 27 – 43 | 25 – 41 |
| Range of worm burdens<br>Median | 1188 – 2098<br>1674 | 0 – 5 | 1832 – 3477<br>2891 | 0 – 3 | 1615 – 4128<br>3276 | 0 – 2 |
| Group mean<br>Mean take | 1653<br>34 % | 0 – 54 | 2768,1<br>56 % | 0,45 | 3031,2<br>66 % | 0,27 |
| Group mean reduction | | 99,9 % | | 99,9 % | | 99,9 % |
| Control median × 0,25 | 418,5 | 0/11<br>>418,5 | 722,75 | 0/11<br>>722,75 | 819 | 0/11<br>>819 |
| Efficacy classification | | A | | A | | A |

(b) *Cooperia pectinata*

| | $L_3$ (Trial 2) | | $L_4$ (Trial 1) | | 5th/Adult (Trial 3) | |
|---|---|---|---|---|---|---|
| Larval dose<br>Larval age (days) | 6000 (3 × 2000)<br>1 – 3 | | 7590 (5 × 1518)<br>4 – 8 | | 6110 (3 × 1013 + 6 × 512)<br>9 – 20 | |
| LIC No. worms<br>calf stage % | 1340<br>$L_3$84 % $L_4$16 % | | 3700<br>$L_4$99 % 5th 1 % | | 5166<br>$L_4$4 % 5th/Ad 96 % | |
| | CONTROL | TREATED | CONTROL | TREATED | CONTROL | TREATED |
| Slaughter age (days) | 41 – 44 | 40 – 42 | 30 – 40 | 36 – 42 | 20 – 31 | 19 – 31 |
| Range of worm burdens<br>Median | 4317 – 5174<br>4487 | 0 – 60 | 4985 – 6236<br>5671 | 0 – 126 | 4397 – 5731<br>4708 | 0 – 25 |
| Group mean<br>Mean take | 4608<br>77 % | 16,1 | 5685,5<br>75 % | 18,45 | 4888,5<br>80 % | 4,7 |
| Group mean reduction | | 99,65 % | | 99,7 % | | 99,9 % |
| Control median × 0,25 | 1121,75 | 0/11<br>>1121,75 | 1417,75 | 0/11<br>>1417,75 | 1177 | 0/11<br>>1177 |
| Efficacy classification | | A | | A | | A |

1. Total number (rounded off) of infective larvae given and approximate daily infection rates in parenthesis.
2. Range of days of infection prior to day of treatment.
3. Range of days between infection and slaughter.

reported[1] it was found that the great majority of $L_4$ and 5th stage *O. circumcincta* were recovered from the abomasal filtrate of the LIC lambs. In contrast in the LIC calves in Trial 5 most of the$gL_4$, 5th stage and even adult stage of *O. ostertagi* were found in the abomasal wall digest, indicating that the developing stages of the latter parasite have a more intimate contact with the abomasal mucosa than do the equivalent stages of *O. circumcincta*. In spite of this it was gratifying to note that 98 % of the $L_4$ *O. ostertagi* were removed by treatment.

Although the result of treatment against $L_3$ *O. ostertagi* (Table 1) at first sight indicates a valid claim for an A rating within the limit set out in the larval anthelmintic test[4], a B rating was granted by the Registering Officer (Act 36/1947) following statistical analysis of the individual worm recoveries recorded.

Although the distribution of *D. viviparus* is not widespread in the Republic of South Africa in some areas of the Eastern Cape it can prove a serious problem to individual farmers, to whom an anthelmintic with such high efficacy against all stages of this parasite would be welcome. The larval infection regimes for the trials against *D. viviparus* $L_3$ and $L_4$ stages conformed to advice kindly given by R.K. Reinecke (1981 Faculty of Veterinary Science, University of Pretoria personal

Table 1 (continued)
(c) *Oesophagostomum radiatum*

| | $L_3$ (Trial 2) | | $L_4$ (Trial 1) | | 5th/Adult (Trial 3) | |
|---|---|---|---|---|---|---|
| Larval dose[1]<br>Larval age[2] (days) | 2520 (10 x 252)<br>1 – 10 | | 2530 (10 x 253)<br>11 – 20 | | 2540 (2 x 255 + 16 x 127)<br>21 – 40 | |
| LIC No. worms<br>calf stage % | 355<br>$L_3$78 % $L_4$22 % | | 683<br>$L_4$100 % | | 1604<br>$L_4$9 % 5th/Ad 91 % | |
| | CONTROL | TREATED | CONTROL | TREATED | CONTROL | TREATED |
| Slaughter age[3] (days) | 41 – 52 | 40 – 50 | 37 – 52 | 43 – 54 | 32 – 51 | 31 – 51 |
| Range of worm burdens<br>Median | 448 – 679<br>534 | 1 – 62 | 207 – 1355<br>773 | 0 – 7 | 772 – 1886<br>1620 | 0 – 0 |
| Group mean<br>Mean take | 537,3<br>21 % | 14,4 | 716,3<br>28 % | 2,36 | 1541,2<br>61 % | 0 |
| Group mean *reduction* | | 97,3 % | | 99,7 % | | 100 % |
| Control median x 0,25 | 133,5 | 0/11<br>>133,5 | 193,25 | 0/11<br>>193,25 | 405 | 0/11<br>>405 |
| Efficacy classification | | A | | A | | A |

(d) *Ostertagia ostertagi*

| | $L_3$ (Trial 4) | | $L_4$ (Trial 5) | | 5th/Adult (Trial 5) | |
|---|---|---|---|---|---|---|
| Larval dose<br>Larval age (days) | 4050 (3 x 1350)<br>1 – 3 | | 4050 (10 x 405)<br>4 – 13 | | 4050 (10 x 405)<br>14 – 23 | |
| LIC No. worms<br>calf stage % | 1145<br>$L_3$100 % | | 1563<br>$L_3$8 % $L_4$69 % 5th 23 % | | 3424<br>$L_4$1 % 5th/Ad 99 % | |
| | CONTROL | TREATED | CONTROL | TREATED | CONTROL | TREATED |
| Slaughter age (days) | 22 – 24 | 22 – 25 | 39 – 48 | 38 – 48 | 39 – 48 | 38 – 48 |
| Range of worm burdens<br>Median | 1943 – 2968<br>2451 | 228 – 830 | 1082 – 3556<br>3058 | 8 – 121 | 1073 – 3538<br>3051 | 5 – 52 |
| Group mean<br>Mean take | 2479,1<br>61 % | 452,4 | 2452,7<br>60,5 | 48,1 | 2446,3 | 20,7 |
| Group mean reduction | | 81,7 % | | 98,0 % | | 99,15 % |
| Control median x 0,25 | 613 | 1/11<br>>613 | 764,5 | 0/11<br>>764,5 | 762,75 | 0/11<br>>762,75 |
| Efficacy classification | | A | | A | | A |

(e) *Bunostomum phlebotomum*

| | $L_3$ (Trial 5) | | $L_4$ (Trial 5) | | 5th/Adult (Trial 4) | |
|---|---|---|---|---|---|---|
| Larval dose<br>Larval age (days) | 3040<br>7 | | 3040<br>17 | | 2010<br>40 | |
| LIC No. worms<br>calf stage % | 21<br>$L_3$100 % | | 722<br>$L_3$1 % $L_4$99 % | | 330<br>5th 100 % | |
| | CONTROL | TREATED | CONTROL | TREATED | CONTROL | TREATED |
| Slaughter age (days) | 42 | 41 – 42 | 42 | 41 – 42 | 61 | 62 – 62 |
| Range of worm burdens<br>Median | 226 – 810<br>327 | 13 – 67 | 226 – 810<br>327 | 0 – 0 | 198 – 686<br>628 | 0 – 0 |
| Group mean<br>Mean take | 409,7<br>13,5 % | 31,2 | 409,7 | 0 | 487,8<br>24 % | 0 |
| Group mean reduction | | 92,4 % | | 100 % | | 100 % |
| Control median x 0,25 | 81,75 | 0/11<br>>81,75 | | 0/11<br>>81,75 | 127 | 0/11<br>>127 |
| Efficacy classification | | A | | A | | A |

1. Total number (rounded off) of infective larvae given and approximate daily infection rates in parenthesis.
2. Range of days of infection prior to day of treatment.
3. Range of days between infection and slaughter.

Table 1 (continued)
(f) *Dictyocaulus viviparus*

| | $L_3$ (Trial 2) | | $L_4$ (Trial 4) | | 5th stage (Trial 1) | | Adult (Trial 3) | |
|---|---|---|---|---|---|---|---|---|
| Larval dose[1]<br>Larval age[2] (days) | 2020 (2 × 1008)<br>1 – 2 | | 1015 (5 × 203)<br>3 – 7 | | 1005<br>8 | | 990<br>25 | |
| LIC No. worms<br>calf stage % | 10<br>$L_3$ 100 % | | 6<br>$L_4$ 100 % | | 180<br>5th 100 % | | 341<br>5th 1 % Ad 99% | |
| | CONTROL | TREATED | CONTROL | TREATED | CONTROL | TREATED | CONTROL | TREATED |
| Slaughter age[3] (days) | 41 – 44 | 40 – 42 | 24 – 28 | 25 – 29 | 34 – 40 | 40 – 42 | 36 – 38 | 35-36 |
| Range of worm burdens<br>Median | 30 – 660<br>443 | 2 – 128 | 100 – 304<br>184 | 0 – 21 | 136 – 355<br>295 | 0 – 33 | 180 – 497<br>328 | 0 – 0 |
| Group mean<br>Mean take | 391,5<br>19 % | 42,7 | 186,3<br>18 % | 8,6 | 291,4<br>29 % | 9,64 | 334,5<br>34 % | 0 |
| Group mean reduction | | 89,1 % | | 95,4 % | | 96,7 % | | 100 % |
| Control median × 0,25 | 110,75 | 1/11<br>>110,75 | 46 | 0/11<br>>46 | 73,75 | 0/11<br>>73,75 | 82 | 0/11<br>>82 |
| Efficacy classification | | A | | A | | A | | A |

1. Total number (rounded off) of infective larvae given and approximate daily infection rates in parenthesis.
2. Range of days of infection prior to day of treatment.
3. Range of days between infection and slaughter.

Table 2: PRE-DOSING FAECAL EXAMINATIONS, DOSING RECORDS, VOLUME OF *MONIEZIA BENEDENI* PROGLOTTIDES EXPELLED AND WORMS RECOVERED AT AUTOPSY

| Calf No. | Pre-dosing eggs in faeces | | Live-mass kg | Dose 9,06 % oxfendazole ml | Vol. segments expelled ml | Recoveries at autopsy | |
|---|---|---|---|---|---|---|---|
| | | | | | | No. of scolices | Vol. strobila ml |
| Controls | | | | | | | |
| 145 | + | + + | 135 | – | – | 4 | 150 |
| 202 | + | + | 133 | – | – | 4 | 20 |
| 936 | + | + | 78 | – | – | 1 | 2 |
| Treated | | | | | | | |
| 150 | + + | + + | 105 | 5,2 | 10 | 0 | 0 |
| 200 | + | + | 122 | 6,05 | 9 | 0 | 0 |
| 201 | + | + | 96 | 4,8 | 54 | 0 | 0 |
| 400 | + | + | 70 | 3,5 | 4 | 0 | 0 |
| 507 | + + | + + | 128 | 6,35 | 30 | 0 | 0 |
| 887 | + | + | 125 | 6,2 | 6 | 1 | 0* |
| 919 | + | + | 68 | 3,4 | 10 | 0 | 0 |
| 920 | + | + | 84 | 4,2 | 85 | 0 | 0 |
| 921 | + | + | 116 | 5,8 | 6 | 0 | 0 |
| 923 | + | – | 134 | 6,65 | 1 | 0 | 0 |
| 926 | + | + | 106 | 5,3 | 83 | 0 | 0 |
| 928 | + | + | 100 | 5,0 | 0,25 | 0 | 0 |
| 9 | + + | + + | 102 | 5,1 | 6 | 0 | 0 |
| 10 | + | + + | 134 | 6,65 | 23 | 0 | 0 |
| 11 | + + | + + | 101 | 5,0 | 15,5 | 1 | <0,1** |
| 14 | + + | + + | 85 | 4,2 | 8,5 | 0 | 0 |
| 21 | + + | + + | 90 | 4,5 | 8 | 0 | 0 |
| 26 | + + | + + | 100 | 5,0 | 26 | 0 | 0 |
| 30 | + + | + + | 125 | 6,2 | 13,5 | 1 | <0,1** |
| 294 | + | + + | 100 | 5,0 | 54 | 0 | 0 |
| 296 | + | + + | 83 | 4,1 | 50 | 0 | 0 |
| 297 | + | + | 155 | 7,7 | 0,3 | 1 | <0,1** |
| 309 | + + | + | 85 | 4,2 | 5 | 0 | 0 |
| 310 | + | + | 75 | 3,7 | 5 | 0 | 0 |
| 312 | + + | + + | 97 | 4,8 | 24 | 0 | 0 |

*Scolex only.

**Scolex with 35 to 37 mm neck

communication). Trickle infection was not attempted in the case of the 5th stage/adult trials as it was feared that an early immune response might interfere with the take. Recovery of the $L_3$ worms from the LIC calf proved unrewarding as meticulous search of the variety of tissues processed yielded only 2 larvae, one from the lung filtrate and one from the mesenteric lymph nodes.

If the eggs of other cattle nematode species show the same susceptibility to oxfendazole as did the 3 species concerned in the ovicidal trial, it can be clearly seen that after a holding period of 8 h dosed cattle can be safely moved to clean pasture.

Although the efficacy rating against *Moniezia* spp. is Class 2, according to the statistical evaluation required for registration in the Republic of South Africa, the results of treatment were very satisfactory in that complete removal of strobila occurred in all 25 treated calves and in the 4 still positive each carried only a single very immature parasite. The absence of lengths of strobila and the small volume of evacuated segments recovered from treated calves probably indicated that there is a more rapid disintegration of *M. benedeni* than occurs in *M. expansa*.

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## BOOK REVIEW

## BOEKRESENSIE

# THE VETERINARY ANNUAL

Editors: G.S.G. GRUNSEL and F.W.G. HILL

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In this the twenty-second issue of the well-known Veterinary Annual, there is something of interest for most veterinarians in many different fields of the profession in the RSA, but perhaps of special significance to us because of the current interest being shown in them, that is if one may single out 2 of the articles, are those on "Farm animal disease trends" and "DAISY in veterinary practice – planned animal health and production services and small computers". In the former article, the diseases which are diagnosed at 33 investigation centres in the UK are recorded in a computer in order to give an indication of trends of disease prevalence. The availability of such an animal disease surveillance system would be of great assistance to us in RSA. In the latter article, "DAISY" is an acronym for "Dairy Information System". This is an integrated suite of computer programmes for small computers, and is designed to assist the farmer, his veterinarian and other advisers in the planning and control of dairy herd health and production.

The basic purpose of the Veterinary Annual is briefly to review recent trends and advances in veterinary science. This edition includes, amongst others, articles on: Reproduction and infertility; Problems of antibiotic resistance in animals and their public health significance; Immune recognition and regulation; Control of foot and mouth disease by vaccination; The recumbent cow: differential diagnosis and differential therapy; Recumbency in cattle in the slaughterhouse; Laminitis in cattle; Teat surgery in cattle; Embryo transfer in cattle; Maedi/visna virus in sheep; Swine vesicular disease; Coccidiosis in pigs; Prevention and treatment of equine strongylosis; Some aspects of equine obstretrics; Crib-biting and windsucking; Lateral fenestration of thoracolumbar protrusions in the dog; Disorders of the canine temporomandibular joint; Radiological investigation of congenital urinary incontinence in the bitch; Neurological emergencies in small animals; Some emergency disorders in the dog; Advances in canine gastroenterology; The Ataxic cat; Neoplasia of the canine oropharynx; Feline glomerulonephritis; and finally, several articles on diseases of the eyes of small animals.

**R.C. Tustin**

# CYATHOSTOMINAE AND OTHER STRONGYLES OF HORSES IN THE FEDERAL REPUBLIC OF GERMANY

IRMGARD G. ANDERSON* and M.A. HASSLINGER**

ABSTRACT: Anderson Irmgard G.; Hasslinger M.A. **Cyathostominae and other strongyles of horses in the Federal Republic of Germany.** *Journal of the South African Veterinary Association* (1982) **53** No. 3, 195-197 (En) Department of Zoology, University of Zululand, 3886 Kwa-Dlangezwa, Republic of South Africa.

The faeces of 34 horses of the "Bavarian main and state studfarm Schwaiganger" (Group I) and 10 ponies (Group II) from various regions of the Federal Republic of Germany, were examined and 4 834 small strongyles were identified. Three species *Cyathostomum catinatum*, *Cylicostephanus longibursatus* and *Cylicocyclus nassatus*, were found to be widely distributed. Although various modern anthelmintics with different effective components were used over the past 20 years, no remarkable change has been brought about in the composition of the parasitic species of the horses in the Federal Republic of Germany.

Key words: *Cyathostomum* spp., strongyles, equine verminosis.

## INTRODUCTION

Microscopical examination of faecal samples from equines gives a limited indication of the presence of parasitic strongyle species. Even the differentiation between large and small strongyles is only reliable if accompanied by larval cultures. In our experience, the large adult strongyles (*Strongylus vulgaris*, *Strongylus equinus* and *Strongylus edentatus*), that take 6½ to 11 months to develop, and which are 4-8 % of the total worm burdens have little effect, whereas the other 40 or more species of cyathostomes[18] have a considerable effect on the host. The smaller species have a relatively short prepatent period of about 5-12 weeks[21] and apart from seasonal fluctuations[16], as many as 3 generations can develop in one season[8] in the climatic conditions of Bavaria in Germany. Various anthelmintics have been used for the control of strongyle nematode parasites of the horse[9]. In the past there was no interest in the efficacy of chemotherapy against the different species of small strongyles. Treatment was considered to have been successful if a few days after therapy many worms were expelled in the faeces and microscopic examination of faeces showed that egg production had stopped.

More recently attempts are made to evaluate the independent effect of new anthelmintics on the different species[15]. Adults or fourth-stage larvae ($L_4$) of *Cyathostomum*, *Cylicocyclus* and *Cylicostephanus* are less susceptible to some modern anthelmintics[4 5 13]. These data, however, have been analysed eratically. The main aim of the present work was to collect and identify the species of strongyle nematodes from the large intestine of horses in the Federal Republic of Germany (FRG).

## MATERIALS AND METHODS

Material from two separate investigations (referred to as Group I and Group II) was available for study.

Group I: Specimens were collected from 34 horses used in an experimental trial with mebendazole (Telmin plus Jansen) on about 300 horses of the "Bavarian main and state studfarm Schwaiganger" breed. The worms eliminated after treatment were collected from the host faeces 2 to 4 days after treatment. One hundred worms were collected from each of 29 horses and from 21 to 83 parasites from each of 5 horses and all the worms were identified microscopically. The 34 horses were closely related and did not leave the studfarm for any great length of time.

Group II: In an anthelmintic experiment with Avermectin $B_{1a}$ (Ivermectin, Merck Sharp & Dohme) nematodes were collected during a critical test using 5 series of 2 ponies each[10]. These animals were bought from various regions of the FRG. Faeces were collected daily over a period of 10 days and examined macroscopically for the larger parasites (strongyles, oxyurids, ascarids, gasterophilids) and a sample was taken for microscopic examination for the presence of smaller strongyles. After this period, the 5 treated and 5 untreated animals were slaughtered to obtain the remaining worms for identification and to assess parasitic status in the control animals. The aliquots used for the identification of worms in this experiment varied as follows: controls (Table 2) 1/100-1/10 and treated ponies 1/100-7/100.

Identifications were made using the descriptions of Popowa[20], Barus[3], Hasslinger[7], Lichtenfels[11] and Georgi[6].

## RESULTS

A total of 3 133 small strongyles, comprising 18 species from 5 genera were collected in the faecal samples passed by 34 horses (Table 1). The hosts harboured between 3 and 11 species and *Cylicocyclus nassatus* was the most common species (Table 1).

The larger part of the worm burdens were 2 398 females and 735 males. Fourth-stage larvae and 5th stages constituted only 1,5 % of the total sample.

A total of 207 000 small strongyles were collected from the 10 ponies of Group II, but only 1 701 of these were identified. *Cyathostomum catinatum* and *Cylicostephanus longibursatus* were found in all the hosts of Group II (Table 2). *Cylicocyclus nassatus* were present in 7, *Cyathostomum coronatum* and *Cylicocyclus radiatus* in 6 out of 10 specimens examined respectively. The other species were only present in 2-4 out of 10 specimens examined in Group II.

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Table 1: SPECIES RECOVERED FROM FAECES OF 34 HORSES IN GROUP I AFTER TREATMENT WITH MEBENDAZOLE

| Species | Horses in which species were present | % of horses in which species were present | Males | Females | Total No. of worms identified | % of total worm count |
|---|---|---|---|---|---|---|
| *Cyathostomum pateratum* | 26 | 76,5 | 83 | 180 | 263 | 8,4 |
| *Cyathostomum catinatum* | 22 | 64,7 | 39 | 110 | 149 | 4,8 |
| *Cyathostomum coronatum* | 11 | 32,4 | 99 | 33 | 42 | 1,3 |
| *Cyathostomum labiatum* | 23 | 67,7 | 29 | 74 | 103 | 3,3 |
| *Cyathostomum labratum* | 1 | 2,9 | 0 | 2 | 2 | 0,1 |
| *Cylicostephanus calicatus* | 26 | 76,5 | 12 | 76 | 88 | 2,8 |
| *Cylicostephanus longibursatus* | 20 | 58,8 | 32 | 102 | 134 | 4,3 |
| *Cylicostephanus minutus* | 5 | 14,7 | 3 | 8 | 11 | 0,4 |
| *Cylicostephanus poculatus* | 3 | 8,8 | 3 | 1 | 4 | 0,1 |
| *Cylicocyclus nassatus* | 34 | 100 | 304 | 1 204 | 1 508 | 48,1 |
| *Cylicocyclus radiatus* | 30 | 88,2 | 141 | 305 | 446 | 14,2 |
| *Cylicocyclus leptostomus* | 28 | 82,4 | 49 | 193 | 242 | 7,7 |
| *Cylicocyclus elongatus* | 7 | 20,6 | 12 | 29 | 41 | 1,3 |
| *Cylicocyclus insigne* | 2 | 5,9 | 3 | 2 | 5 | 0,2 |
| *Triodontophorus serratus* | 8 | 23,5 | 7 | 8 | 15 | 0,5 |
| *Triodontophorus brevicauda* | 3 | 8,8 | 2 | 2 | 4 | 0,1 |
| *Poteriostomum imparidentatum* | 8 | 23,5 | 2 | 11 | 13 | 0,4 |
| *Poteriostomum ratzii* | 6 | 17,7 | 2 | 14 | 16 | 0,5 |
| $L_4$* & 5th stage | 14 | 41,2 | 3 | 44 | 47 | 1,5 |

*$L_4$ = fourth stage larvae

Table 2: SPECIES RECOVERED FROM FAECES OF 10 HORSES IN GROUP II AFTER TREATMENT WITH AVERMECTIN $B_{1a}$

| Species | Horse number | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 644 | 647 | 648 | 649 | 655 | 645[c] | 646[c] | 650[c] | 651[c] | 652[c] | ... times recovered |
| *Cyathostomum catinatum* | 15 957 | 6 800 | 900 | 700 | 1 300 | 4 509 | 320 | 700 | 21 800 | 10 | 10 |
| *Cyathostomum coronatum* | | 5 500 | 1 280 | 1 900 | 400 | | 280 | 2 200 | | | 6 |
| *Cyathostomum labiatum* | 300 | | | | 500 | 3 | | 100 | | | 4 |
| *Cyathostomum labratum* | | | | | 200 | | | | | 20 | 2 |
| *Cylicostephanus longibursatus* | 1 971 | 29 200 | 5 360 | 3 500 | 3 800 | 500 | 120 | 7 000 | 15 950 | 700 | 10 |
| *Cylicostephanus calicatus* | | 1 900 | | | 3 000 | | | | 2 300 | | 3 |
| *Cylicostephanus minutus* | | 6 550 | | | 500 | | | | | | 2 |
| *Cylicocyclus nassatus* | 26 643 | 1 300 | 2 000 | | 1 900 | 5 410 | | 5 800 | 1 700 | | 7 |
| *Cylicocyclus radiatus* | 2 910 | 850 | | | 1 | 1 102 | | 100 | 100 | | 6 |
| *Cylicocyclus leptostomus* | 1 743 | 3 050 | | | | 200 | | 100 | | | 4 |
| *Triodontophorus serratus* | | 100 | | | 6 | 218 | 120 | | | | 4 |
| *Triodontophorus brevicauda* | | 1 | | | | 104 | 60 | | | | 3 |
| $L_4$ & 5th stage | 1 314 | | 400 | | | 500 | | | | | 3 |
| Total | 50 838 | 55 851 | 9 940 | 6 100 | 11 607 | 12 542 | 900 | 16 000 | 41 850 | 830 | — |

c = control

## DISCUSSION

The role of the horse in Germany is now more one of sport and pleasure rather than work and transport and the horses have become even more financially valuable. Many reports on strongyles were primarily concerned with the biology, pathogenicity, prophylaxis and/or therapy and therefore only the total worm burdens were mentioned with little effort made to differentiate the parasite as far as species is concerned. There is little knowledge of the pathogenesis of individual species of cyathostome[18].

The present study puts more emphasis on the importance of the individual species of parasites than on the total worm burden of the host. The prevalence of cyathostome in horses from South Germany (Allgäu, Baden) according to Hasslinger[7] was *Cyathostomum catinatum* 68 %, *Cylicocyclus nassatus 65 %, Cyathostomum coronatum 37 % and Cylicostephanus longibursatus* 33 % respectively. The additional 7 species of cyathostomes occurred in only 2-23 % of horses. Subsequent to the work of Hasslinger[7] many anthelmintics have been synthesised and therefore it became interesting to establish whether there was any

change or variation in the dominant species. For example, in a trial involving 6 benzimidazoles Drudge et al.[5] observed considerable fluctuations (between 34 and 84 %) in the effect of the drug on *Cylicocyclus nassatus*, *Cyathostomum catinatum* and *Cylicostephanus longibursatus*. These species were frequently seen during the current investigation. In all 34 horses of Group I *Cylicocyclus nassatus* was collected and this species is widely distributed in England[22] and Belgium[19]. *Cylicocyclus nassatus* also constituted almost half the total material from the horses of Group I. From the results of Group II it can be seen that the species, *Cylicocyclus nassatus*, *Cyathostomum catinatum* and *Cylicostephanus longibursatus* are conspicuous by their high numbers. This observation agrees with that made by Ogbourne[17] from horses in Britain with a prevalence of 93,1 %, 94,2 % and 98,9 % for the same 3 species respectively. *Cylicocyclus radiatus* which was described from Spain for the first time in 1978[2] and in Holland in 1980[14] was found in relative abundance (Table 1 and 2) in the stud horses (Group I) and the ponies (Group II). New records for this species have been given by Barus[3] for Czechoslovakia and Sobieszewski[23] for Poland.

With the exception of *Triodontophorus brevicauda*, *Cylicostephanus poculatus* and *Cylicocyclus insigne*, each of which was present in very small numbers, all other species of strongyles from these horses showed a female : male ratio greater than 1, as has been observed recently by Anderson[1] with nematodes from the impala (*Aepyceros melampus*) in South Africa.

In conclusion, the difference in the abundance of species should still be emphasised. Whilst Lyons et al.[12] in Kentucky found up to 29 species, the earlier[7] and later (see Group II, this paper) observations from various parts of Germany with only 14 and 12 species, respectively, show a reduced number of species present. The results in Group I had a total of 18 species. From the latter results in Group I it can be concluded that in Germany, on pastures with only one animal species, the infestation risk and distribution of parasite species are particularly enhanced.

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**SHORT COMMUNICATION** **KORT BERIG**

# STANDING HEAT AFTER SYNCHRONIZATION WITH CLOPROSTENOL IN CATTLE

S. HERR*

ABSTRACT: Herr S. **Standing heat after synchronization with cloprostenol in cattle.** *Journal of the South African Veterinary Association* (1982) **53** No. 3, 198 (En) Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa.

Following 2 injections of cloprostenol spaced 10 days apart, 8/17 (47 %) of the heifers showed standing heat at 48 h. Where fixed time insemination is practised it is therefore suggested that the group of animals synchronized be observed for the first signs of heat as early as 48 h and that the double insemination be done 12 and 36 hours later rather than at a set time such as 72 and 96 hours after the 2nd injection.

Key words: Oestrus synchronization, cloprostenol.

## INTRODUCTION

Following double treatments with natural or synthetic prostaglandins spaced 10-12 days apart, oestrus occurs between 2-5 days later in a large percentage of cattle[1]. Fixed time inseminations after such double treatments are generally recommended for 72 and 96 h after the 2nd injection[2 3 4]. In a group of heifers synchronized by double injection of cloprostenol spaced 10 days apart the times of natural service and therefore of standing heat were investigated, and the optimal time for insemination derived from this data.

## MATERIALS AND METHODS

Seventeen virgin, Friesland heifers (2-3 years old), were each injected twice with 2 ml (500 $\mu$g) cloprostenol (Estrumate, Milvet Ethicals) intramuscularly, 10 days apart. Starting 48 h after the 2nd injection, 3 Friesland-type bulls (3-4 years old) were introduced for 30 min intervals at 08h00, 12h00 and 17h00 for 3 days. Immediately after a single service the heifers were removed from the group.

## RESULTS

In the presence of the bulls, 8/17 (47 %) of the heifers showed standing heat and were served at 48 h, 1/17 (6 %) at 56 h, 6/17 (35 %) at 72 h and 1/17 (6 %) at 76 h. One heifer failed to show standing heat during the period of observation.

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## DISCUSSION

Although the occurrence of oestrus is known to vary from 2-5 days after administration of prostaglandins and the synchrony is sometimes advanced by up to 24 h, the general recommendation for fixed time insemination is 72 and 96 h[1 2 3 4]. In our trial the high proportion (47 %) of heifers showing standing heat at 48 h was significant. Unless a large proportion of these heifers remained on heat for a further 12 h or more it is doubtful whether a fixed time insemination scheduled for 72 h would have much chance for success. We therefore suggest that where fixed time insemination is to be practised, the group nevertheless be observed for signs of oestrus after 48 h and the timing of the insemination be flexibly, instead of rigidly, applied. Thus, if signs of oestrus were seen in the group at 48 h, the inseminations should be done 12 and 36 h later. In this trial 16/17 (94 %) of the heifers, it is felt, would have had a reasonable chance for successful insemination at these times. This is calculated to be 60 and 84 h after the 2nd prostaglandin injection. For as many as 8/17 (47%) of the heifers insemination at 72 h may well have been too late. This may account for some of the low conception rates reported in various synchronization trials.

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RESEARCH NOTE NAVORSINGSNOTA

# SERUM AND MILK CONCENTRATIONS OF OXYTETRACYCLINE AFTER THE ADMINISTRATION OF A LONG-ACTING FORMULATION TO SHEEP

A. IMMELMAN* and G. ZIV**

ABSTRACT: Immelman A.; Ziv G. **Serum and milk concentrations of oxytetracycline after the administration of a lovg-acting formulation to sheep.** *Journal of the South African Veterinary Association* (1982) 53 No. 3, 199-200 (En) Department of Physiology, Pharmacology and Toxicology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

A long-acting formulation of oxytetracycline was administered intramuscularly to lactating Awassi ewes. Serum and milk samples were collected and the concentration determined using a microbiological technique. The peak serum and milk levels were very similar but the milk concentration was attained 4 h later than the serum level. A concentration of 0,5 μg/ml was maintained in serum and milk for 56 h.

Key words: Sheep, milk, oxytetracycline.

## INTRODUCTION

The excretion of antibiotics in milk is of great importance because of the adverse effects it may have on human health and the manufacturing of various cultured dairy products. It is the duty of the veterinary profession to avoid this contamination of the milk supply. This can only be done if the veterinarian knows the excretion times of the various antibiotics that are used for parenteral administration.

The excretion times of various tetracycline analogues have been studied in cows and ewes[3]. The formulations used during these studies were products commercially available. The formulation of oxytetracycline was that intended for daily administration, i.e. a short-acting formulation. After a single intravenous administration of 20 mg/kg body mass a detectable blood level was maintained for 8 h. To maintain relatively constant serum levels for 10-14 h, the drug was initially injected intravenously at a dosage of 10 mg/kg body mass. This was followed by a maintenance dose, equal to one-half the priming dose, given intramuscularly at intervals equal to the biological half-live of the drug. This resulted in the serum concentrations of oxytetracycline being maintained between 2,25-2,8 μg/ml. At the same time the ratio of milk ultrafiltrate concentration to serum ultrafiltrate concentration was found to be 0,75 ± 0,13. This indicates that the drug penetrated milk poorly[3].

The purpose of this present study was to compare the serum levels of exytetracycline with the concentration in milk after the intramuscular administration of a long-acting formulation of the drug.

## MATERIALS AND METHODS

As experimental animals 8 adult lactating Awassi ewes were used. Their lambs were approximately 4 weeks old. The live mass of these ewes varied between 55 and 65 kg. They were kept outdoors and fed roughage twice a day. The lambs stayed with the ewes during the trial.

The formulation employs a solvent system based on 2-pyrrolidone, the lactam of gamma-amino-butyric acid. This produces a high and extended antibiotic level for 3-4 days after a single intramuscular injection. The dosage recommended to achieve this is 20 mg/kg body mass[1].

Blood specimens were collected from the *Vena jugularis* 0,5, 1, 2, 3, 4, 6, 8, 10, 24, 30, 48, 56 and 72 h after injection. After allowing the blood to clot the serum was collected and frozen at minus 20°C until the analyses were done. With each blood specimen a milk specimen was collected and treated similarly.

The method of assay was a standard agar plate microbiological technique for the determination of antibiotics with *B. subtilis* as test organism. Standards were prepared using sheep's serum and milk. The lower sensitivity limits for this assay method was 0,5 μg/ml in milk and 0,3 μg/ml in serum.

## RESULTS AND DISCUSSION

The results as presented in Fig. 1 shows a sharp rise in serum concentrations of oxytetracyclines. At the time the first specimen were collected the serum values were 1,5 μg/ml but nothing could be detected in the milk. The first positive finding of 1,4 μg/ml in milk was only achieved 2 h after administration. The serum level reached a peak of 3,1 μg/ml at that time. The highest concentration of antibiotic in the milk was after 6 h and the level was almost the same as the peak concentration in the serum. The decline in milk and serum concentrations were very similar with the milk concentration slightly higher. After 56 h both concentrations dropped below the detectable levels of the assay method. In one ewe oxytetracycline could be detected in the milk 72 h after administration and in another ewe the serum sample was still positive for oxytetracycline after 72 h.

It has been suggested that the distribution of tetracycline antibiotics from blood to milk is due to passive diffusion and that it could be explained on the basis of the pH-partition hypothesis[3].

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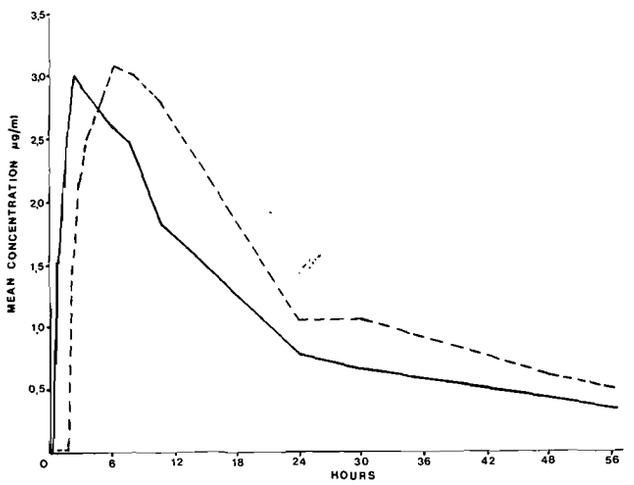


**Fig. 1:** The concentration of oxytetracycline in serum and milk of ewes after intramuscular injection of a long-acting formulation.

In a previous study using a short acting formulation, tetracycline was injected intramuscularly at a dosage of 20 mg/kg body mass in ewes with mastitis in the one half of the udder[2]. In that study a peak serum concentration of approximately 4 µg/ml was reached after 2 h and the concentration dropped below 0,5 µg/ml after 8 h. In milk the peak concentration was half that of the serum value, was reached 4 h after administration and the concentration of 0,5 µg/ml was maintained for 12 h. These authors calculated that to maintain a minimum inhibitory concentration of 0,5 µg/ml tetracycline in their circumstances the drug must be administered at a dosage of 20 mg/kg intramuscularly. In normal ewes the dose must be repeated 6 hourly and in cases of mastitis every 10 h.

In the ewes used in this study the blood levels were not as high as that attained in Merino sheep (Immelman – unpublished data). The minimum inhibitory concentration in the serum was maintained for 56 h. The peak level in milk was much higher than those recorded in a previous study using a short acting formulation of oxytetracycline[2]. Minimum inhibitory concentration in milk was maintained for the same period as in the serum.

ACKNOWLEDGEMENTS

We wish to express thanks to the South African Medical Research Council for financial assistance and the Director of the Kimron Veterinary Research Institute of Israel for the facilities made available to us.

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## BOOK REVIEW — BOEKRESENSIE

# THE SCIENCE OF ANIMAL HUSBANDRY
## 2nd Edition 1979

J. BLAKELY and D.H. BADE.
Illustrations by T. STALLMAN

Reston Publishing Company Inc., Reston Virginia 22090. Introductory XI Text 516. Figures 276. Tables 38.
Publ. Price R26,95

The book is about animal husbandry in the United States of America but the information given will be found useful elsewhere. It is designed for use by students and teachers. The print is clear and easy to read. There is a list of study questions with the answers at the end of each chapter which makes revision easy. There are numerous illustrations and useful tables. The list of contents is well presented enabling any information required to be found easily. Sub-titles under the main chapter headings give the page on which each subject appears. Appendix "a" gives the composition of feeds and appendix "b" the Canadian beef and pork grading systems.

The first chapter describes the beginnings of animal husbandry and the introduction of animals into the United States of America. Then there are 8 chapters on beef cattle, 4 on dairy cattle, 5 on sheep, 7 on swine, 5 on poultry and 4 on horses. There is no chapter on goats but it is not known if the goat industry in the United States of America justifies a section on them.

A standard pattern is followed in dealing with each industry. The main aspects dealt with are the breeds, reproduction, feeding, criteria for selection, judging, management, breeding, handling and diseases. The procedures involved in the production of beef, milk, mutton, wool, pork, broilers and eggs are described.

The book contains essential basic information for anyone who is studying or teaching animal husbandry and will be found useful as a reference book.

G.D. Sutton

REVIEW OORSIG

# URIC ACID METABOLISM IN THE DALMATIAN COACH HOUND

O.M. BRIGGS* and O. SPERLING**

ABSTRACT: Briggs O.M.; Sperling O. **Uric acid metabolism in the Dalmation coach hound.** *Journal of the South African Veterinary Association* (1982) 53 No. 3, 201-204 (En) 32 Blue Route Centre, 7966 Tokai, Republic of South Africa.

The Dalmation coach hound differs in its uric acid metabolism from all other breeds of dogs in that it is hyperuricaemic and hyperuricosuric and exhibits increased renal urate clearance. The decreased oxidation of uric acid by the liver uricase has been shown to result from defective uric acid transport into the hepatocytes. The increased renal urate clearance has been shown to result either from a similar tranport defect in the kidney or due to the presence of an abnormal liver metabolite which interfers with renal urate handling.

Key words: Dalmatian, uric acid metabolism, urolithiasis.

## INTRODUCTION

The Dalmatian coach hound, as compared to other dog breeds, exhibits three characteristic abnormalities of uric acid metabolism namely hyperuricaemia[16 17 29 31], hyperuricosuria [2 6 12 21 31] and increased renal uric acid clearance[10 16 17 27 31]. These properties are associated with hypoallantoinaemia[6 11 30] and hypoallantoinuria[6 11 21 30]. As result of these abnormalities a high incidence of urate urolithiasis occurs in this breed[4 26]. Other diseases such as recurrent dermatitis, chronic cystitis and deafness are possibly related[5 20]. In excreting uric acid the Dalmation dog resembles man more closely than the other breeds. On the other hand, in its high renal urate clearance, this breed of dogs differs from man whose renal clearance values are lower and therefore closer to those of the non-Dalmatian. In this respect the Dalmation resembles man affected with the inborn renal urate transport defect connoted renal hypouricaemia[4]. The uniqueness of uric acid metabolism in the Dalmatian has attracted many investigators in search of the underlying metabolic defect(s). In addition these features render the Dalmatian suitable for study in relation to pathological conditions of purine metabolism in man, mainly in connection with hyperuricosuria, uric acid urolithiasis and hereditary renal hypouricaemia. An outline of purine degradation is given in Figure 1.

## THE INITIAL REPORT

The greater degree of uric acid excretion in the urine of the Dalmatian than of other dogs was first described by Benedict in 1915[2]. It was a chance discovery which sparked off the interest in this breed. Addition of hydrochloric acid to the urine of a Dalmatian immediately produced a precipitate which was later identified as uric acid crystals. Benedict found that this same animal on a purine-free diet excreted almost as much uric acid per day as an average-sized man on a similar diet.

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## THE MODE OF INHERITANCE

Trimble & Keeler[24] crossed Dalmatians with Collies to study the inheritance of this anomaly. They found that "high uric acid excretion" is inherited as an "almost completely recessive, non-sex-linked unit character, depending for its expression upon the presence of a single pair of Mendelizing genes". Keeler pointed out that the breed must have been almost pure-bred for this characteristic over a long time, since all the Dalmatians tested in all parts of the world possess this unique characteristic with no exception[9].

## THE RENAL AND HEPATIC HANDLING OF URIC ACID

The conversion of uric acid to allantoin (Fig. 1) occurs in the liver and this conversion ceases in hepatectomized dogs[13]. Therefore researchers concentrated on the liver as the site of the metabolic defect which resulted in the discovery of hyperuricosuria and hypoallantoinuria in the Dalmatian. In 1918, Wells[25] showed that Dalmatians possessed uricase in the hepatocytes and ought therefore to be able to convert uric acid to allantoin in the same way as all other dogs. This was confirmed later by Klempere et al.[11] who found in 1938, that the livers of all breeds of dogs, including Dalmatian, were rich in uricase.

In view of these findings, it became evident that the defect in uric acid conversion to allontoin is due not to absence of uricase but in the mechanism rendering uric acid available to the hepatocyte enzyme. As to the nature of this liver abnormality, Klemperer et al.[11] suggested that the Dalmatian liver may have a defective urate transport system.

An entirely different suggestion to explain the nature of the liver defect, was brought forward in association with the finding of another abnormality in uric acid metabolism in the Dalmatian, namely the increased renal urate clearance. Very early in the study of uric acid metabolism in the Dalmatian, evidence was obtained indicating the presence of an abnormality in this dog in the renal handling of uric acid also. Young et al.[28] and later Myers & Hanzal[19] noted that the daily excretion of creatinine and of uric acid in the Dalmatian dog was approximately equal. This finding was confirmed by Friedman & Byers[6], who were also the first to


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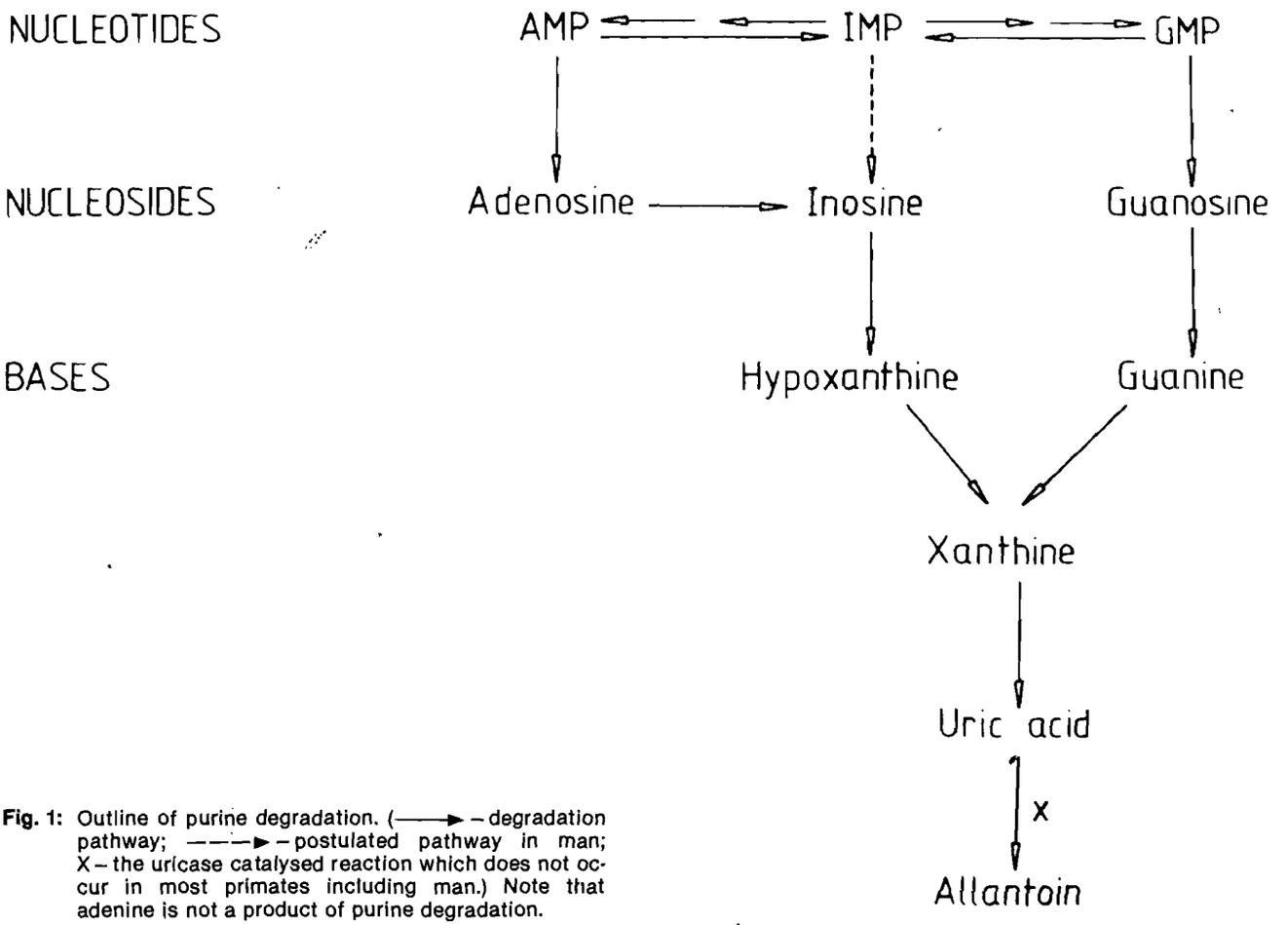


**Fig. 1:** Outline of purine degradation. (——► – degradation pathway; ——►– postulated pathway in man; X – the uricase catalysed reaction which does not occur in most primates including man.) Note that adenine is not a product of purine degradation.

demonstrate in the Dalmatian a renal tubular abnormality in urate handling, manifest in decreased reabsorption of uric acid. These investigators suggested the renal abnormality to be the primary and only defect underlying both liver and renal abnormalities. They suggested that the decreased oxidation of uric acid in the liver is due to more uric acid being excreted and less being available at the hepatocytes for conversion to allantoin. Although the latter suggestion was not adopted by other investigators, the presence of the renal abnormality was confirmed in many other studies. Woolfson et al.[27] found that the renal clearance of urate in the Dalmatian dog exceeded the glomerular filtrHtion rate, indicating that its excretion is the result of both glomerular filtration and active tubular secretion. Kessler et al.[10] found that whereas uric acid underwent net reabsorption in the non-Dalmatian kidney, it underwent net secretion in the Dalmatian. They found that the proximal tubule was the site of both urate reabsorption in the non-Dalmatian and urate secretion in the Dalmatian dog. Further work by Zins & Weiner[31] and by Mudge et al.[16] led to the realization that uric acid is both reabsorbed and secreted by the renal tubule[17] and that the relative magnitude of reabsorption and secretion varies from species to species.

From the studies conducted up to this point it became evident that the Dalmatian may have 2 abnormalities concerning uric acid metabolism. One in the liver, in not rendering uric acid available to the hepatocyte uricase and the other in the kidney, in decreased renal tubular urate reabsorption. In order to establish the relationship between these two organ defects and the relative effect of each on the high uric acid excretion and on the high renal urate clearance, organ transplants between Dalmatians and non-Dalmatians were performed. Cohn et al.[3] demonstrated that transplantation of a non-Dalmatian kidney to a Dalmatian, caused an increase in allantoin excretion, whereas transplantation of a Dalmatian kidney to a non-Dalmatian caused a decrease in allantoin excretion. They concluded that the Dalmatian kidney differed from that of the non-Dalmatian. They also suggested that the major defect in purine metabolism, that underlying the hyperuricaemia and hyperuricosuria, did not lie entirely within the kidney. Support for the latter conclusion, is evident from the results of the study of Appleman, Hallenbeck & Shorter[1], who found that a Dalmatian, following receipt of a non-Dalmatian kidney, continued to excrete large amounts of uric acid. However, since in their experiments the effect of transplantation on renal urate clearance was not recorded, their results do not relate at all to the question of the presence or absence of a uric acid transport defect in the Dalmatian kidney.

A second and more plausible explanation than that suggested by Friedman & Byers[6] for the dual existence in the Dalmatian of abnormalities in uric acid metabolism in both liver and kidney, was furnished by Harvey & Christensen[8]. They demonstrated that a specific transport system for uric acid, present in the erythrocytes of man and non-Dalmatians, appeared to be absent in Dalmatians. In view of this finding, they suggested a generalized defect in the transport process of uric acid in this dog, explaining both the unavailability of uric acid to liver cell uricase as well as the defective

reabsorption of urate in the renal tubules. The suggestion of a defective urate transport in the Dalmatian hepatocytes is compatible with the suggestion made as early as 1938 by Klemperer et al.[11]. Moreover, studies done more recently by Yu et al.[30] support the evidence for such a defect. They caused non-Dalmatian dogs to mimic Dalmatians in decreased uricase activity by administration of oxonic acid – an uricase inhibitor. The fact that the oxonate-induced increase in the excretion of urate was not associated with increased renal urate clearance, indicates that the increased renal urate clearance in the Dalmatian is not due to the high excretion of urate.

Whereas the presence of a urate tranport defect in the Dalmatian liver seems very likely, the presence in this dog of a generalized urate transport defect as suggested by Harvey & Christensen[8], or of a renal transport defect is still questionable; the former because of the inability of Duncan & Curtiss[5] to confirm the erythrocyte transport defect and the latter, because of the results of the liver transplantation experiments performed by Kuster et al.[12 13]. These investigators found that when non-Dalmatian dogs received Dalmatian livers, the renal clearance and excretion of uric acid increased to values typical of Dalmatians and that when Dalmatians received non-Dalmatian livers, the above parameters diminished to those typical for non-Dalmatians. These results were taken to indicate that the Dalmatian liver is responsible for both the increased amount of excreted urate, as well as the increased renal urate clearance. According to the results of this study, the increased renal urate clearance in the Dalmatian is caused by an abnormal metabolite produced in the liver. Obviously, such results refute the possibility of a true reabsorption defect in the Dalmatian renal tubules. The role of the Dalmatian liver in the pathogenesis of the hyperuricaemia and hyperuricosuria, as reflected in the liver transplantation experiments, is probably correct. However, in view of the conflicting data concerning the renal defect, the suggested role of the liver in the pathogenesis of the renal abnormality needs further support.

## STONE FORMATION

The hyperuricosuria of the Dalmatian is associated with urate urolithiasis. Porter[21 22 23], examined the chemistry involved in the formation of stones by Dalmatians. He found this to differ from man in that most, if not all, Dalmatian stones include ammonium urate crystals as opposed to human stones which include mostly uric acid. As in man, high urinary pH is beneficial in preventing stone formation in the Dalmatian but rather because it decreases the concentration of the ammonium ion than because it increases uric acid solubility.

## HEREDITARY RENAL HYPOURICAEMIA IN MAN

In 1972, Greene et al.[7] reported the first case of hereditary renal hypouricaemia in man. They emphasized the similarity between the renal abnormality in their patent and that in the Dalmatian. Since then several additional families affected with renal hypouricaemia have been found[5]. However, whether in these patients there is a true renal transport defect for uric acid or whether there is in them, as suggested for the Dalmatians, an abnormal metabolite produced in the liver which affects renal urate handling, is still to be resolved.

## FACTORS RELATIVE TO VETERINARY MEDICINE

The Dalmatian breed of dogs have several biological aberrations in addition to uric acid metabolism. Duncan & Curtiss[4] listed some which include congenital deafness, cardiac arrythmias, ocular abnormalities, urinary tract disease and recurrent dermatitis. In 1969, the Dalmatian Research Foundation was started by J.C. Lowrey of Pennsylvania. He speculated on the connection between the unique uric acid metabolism and some of above diseases, such as recurrent cystitis and dermatitis found in Dalmatians[14 20]. Individual cases treated with allopurinol (Zyloprim, Calmic), a xanthine oxidase inhibitor which prevents the degradation of hypozanthine and xanthine to uric acid (Fig. 1) showed improvement. However, the role of the other concurrent treatment was not made clear. Lowrey et al.[15] in 1973 and Muller & Kirk[18] in 1976 used the term "bronzing" to describe a specific Dalmatian skin condition. They linked it with hyperuricosuria, uric acid uroliths and urinary tract infections. Although they advocated allopurinol therapy, there is as yet no sound biochemical basis for its use in this condition. Veterinarians have reported satisfactory results using the uricosuric agent, Benzbromarone (Minuric, Reckitt & Colman) in acral lick dermatitis and other non-specific dermatitis in Dalmatians (G.J. Futter, 32 Blue Route Centre, Tokai, Personal communication). However further investigations are necessary to evaluate the role of this drug in veterinary medicine.

### ACKNOWLEDGEMENTS

The authors would like to thank Professor E.H. Harley of the Medical School, University of Cape Town, for his enthusiasm, encouragement and guidance.

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## BOOK REVIEW — BOEKRESENSIE

# THE ANATOMY OF THE DOMESTIC ANIMALS
## Vol. 3: The Circulatory System, the Skin, and the Cutaneous Organs of the Domestic Mammals

A. SCHUMMER, H. WILKENS, B. VOLLMERHAUS and K.-H. HABERMEHL

Translated by W.G. Siller and P.A.L. Wight. Paul Parey, Berlin and Hamburg & Springer, New York 1981 pp XV + 610 Figs 437 (173 colour) ISBN 3 – 489 – 55618 – 6 (Paul Parey, Berlin & Hamburg) ISBN 0 – 387 – 91193 – 6 (Springer, New York). Price: DM 186

This is an unrevised, unabridged translation which faithfully follows the German text. The nomenclature is in accord with the nomina Anatomica Veterinaria. References up to 1975 are included.

The introductory chapter discusses the cardiovascular system in a general way. The formation, composition, function and destruction of blood are dealt with and this is followed by a discourse on the structure and function of the blood vessels, including their nervous and hormonal control. The description of the heart follows the same scheme: the first part deals with fundamental cardiac anatomy applicable to all mammals while subsequently consideration is given to species peculiarities and the comparative characteristics of the organ.

In the chapters dealing with the origin, course and distribution of the blood vessels, all pertinent detail is included by a comparative presentation of the vessels of all species, region by region. This method emphasizes the basically similar pattern in all species, thus allowing rules of nomenclature to be established for individual blood vessels. This principle is used throughout except in the blood vessels of the foot where, because of the obvious differences in the structure of the extremities, it is necessary to give a detailed description for each species.

The chapter dealing with the Lymphatic System comprises pp 169. All available literature up to 1975 is included, as well as the classical descriptions of Baum. Numerous valuable illustrations from older monographs, long out of print, are reproduced. In accord with the general approach of the authors, the systematic description of the lymphatic organs in the various species, has been compiled so as to provide both basic knowledge and information of practical value in veterinary practice. The translators decided to retain the references to meat inspection and the German legal requirements for the examination of lymph nodes in the abattoir, because they may be found useful as a general guide.

In the chapter on the Skin and Cutaneous Organs (pp 114) the carnivores, pig and ruminants are dealt with more thoroughly than in previous textbooks of anatomy. Particular stress is placed on the description of special skin glands which are of importance as marker – and signal glands.

As a textbook this volume is in a class of its own. The quality of the illustrations, the thoroughness of the text and the technical perfection of the publication deserve the highest praise. As a reference book for anatomists, surgeons, health inspectors and practitioners who strive for excellence, this book is totally satisfying.

M.M.S. Smuts

**CASE REPORT** **GEVALVERSLAG**

# URIC ACID UROLITHIASIS IN A DALMATIAN COACH HOUND

O.M. BRIGGS*, A.L. RODGERS** and E.H. HARLEY**

ABSTRACT: Briggs O.M; Rodgers A.L.; Harley E.H. **Uric acid urolithiasis in a Dalmatian coach hound.** *Journal of the South African Veterinary Association* (1982) 53 No. 3, 205-208 (En) 32 Blue Route Centre, 7966 Tokai, Republic of South Africa.

A case of urethral obstruction in a Dalmatian coach hound is presented. The calculi were surgically removed and identification of the chemical constituents revealed them to consist of sodium acid urate monohydrate together with uric acid, but without ammonium acid urate. Pre- and postoperative serum and urinary uric acid concentrations, urinary pH and specific gravities were determined to monitor the response to allopurinol therapy, which resulted in relief of symptoms accompanied by a moderate reduction in serum urate levels.

Key words: Dalmatian, urolithiasis, uric acid metabolism.

## INTRODUCTION

Many reports describe a defect in the transport of uric acid across liver cells in the Dalmatian coach hound[4 6 9 10]. This appears to result in uric acid failing to reach hepatic uricase, thereby resulting in the hyperuricaemia and hyperuricosuria typical of this breed. In non-Dalmation dogs, uric acid is converted by uricase in the liver to allantoin, which relative to uric acid is a more soluble end product of purine metablism. It is not surprising therefore to find that of all the dog breeds, urate stones are most commonly encountered in Dalmatians[9 16]. Porter found that Dalmatians which form calculi do not excrete more uric acid than do non-stone-forming Dalmatians[10] and he concluded that hyperuriosuria was not the only determinant in the aetiology of urate lithiasis[11]. All the Dalmatian stones examined by Porter were reported to have as their major component ammonium urate[10]. This is contrary to the situation in man where uric acid lithiasis is more common[9]. Porter found the concentrations of urate in normal and stone-forming urines of Dalmatians to be far in excess of the solubility limit for urate in aqueous solution[11]. He demonstrated that the stability was due to the formation in urine of lyophobic, urate-containing colloids[12]. The ammonium ion is a strong precipitator of these colloidal urates and high levels of the former tend therefore to result in stone formation. A high urinary pH results in reduced ammonium ion concentration and hence is beneficial in preventing stone formation. From Porter's investigations it is clear that control of urinary urate concentration and alkalinization of the urine are two pertinent approaches to the treatment of ammonium urate stone-forming Dalmatians.

## MATERIALS AND METHODS

An 11 year old male Dalmatian coach hound with a body mass of 21 kg presented with tenesmus and anuria of 6 hours duration. Previously the dog had been a regular, healthy patient. Blood and urine had been collected 120 days before for uric acid determination (Table 1).

On examination, the dog appeared depressed and in discomfort. It had a firm, distended bladder, normal temperature and mild dehydration. A plain lateral radiograph failed to reveal calculi. Anaesthesia was induced using thiopentone sodium and maintained on a halothane/oxygen mixture. A 5 % dextrose solution was administered intravenously to correct the dehydration. A catheter passed up the urethra met with considerable obstruction. Flushing with saline produced some small calculi but was unable to relieve the obstruction. A prepubic urethrotomy was performed and further retrograde flushing attempted. However, the blockage was so extensive that a perineal urethrostomy and eventually a cystotomy were necessary to remove all the calculi.

Procaine penicillin G (Novocillin Vet, Novo) was administered subcutaneously at 900 000 u daily. Five days postoperatively, this was changed to chloramphenicol (Troymycetin, SCS Pharmalab) lg per os divided twice daily. The administration of intravenous fluids was continued for 3 days postoperatively. Two litres of 5 % dextrose were used on Day 1 and 1ℓ each on Days 2 and 3. Increased uptake of fluids were encouraged by salting the food. The prepubic urethrotomy had not been sutured closed and the dog voided urine from this wound and the perineal urethrostomy. Persistent discomfort, haematuria and a purulent discharge indicated continued urolith formation.

Allopurinol treatment (Zyloprim, Calmic) was started on the 12th day post-operatively at 600 mg divided twice daily. The haematuria and infection cleared and on the 19th day postoperatively the allopurinol was reduced to 300 mg daily. After a further 7 days this was reduced to a maintenance dose of 100 mg daily. Recovery was uneventful and there has been no recurrence in 4 months.

Blood and urine were collected on the following postoperative days: 0, 3, 6, 13, 19, 29, 38 and 43. The serum and urinary uric acid concentrations were determined by the uricase method (Test-combination, Boehringer, Catalogue number 124737) (Table 1). The specific gravity (SG) of each urine sample was measured with an SG refractometer (Uricon, Atago) and is listed together with urinary pH (measured with a Taueber and Gorssen Scientific pH meter model 800) in Table 1.

## STONE ANALYSIS

### X-ray diffraction (XRD).

One of the calculi was cleaved with a sharp blade and

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samples were obtained from the central and outer regions of the stone's exposed surface. These were ground to a fine powder in an agate mortar and pestle and x-ray diffraction patterns were recorded on Kodak NS-392T film by the Debye-Scherrer[1] method using a Philips powder camera (radius 28,65 mm) mounted on a Philips 1008 x-ray generator with nickel filtered $CuK_\alpha$ radiation. The constituents giving rise to the reflections were identified by comparison with published reference standards of Sutor and Scheidt[14].

### Scanning electron microscopy (SEM).

Stone samples were mounted on aluminium stubs, their cleaved surfaces uppermost, and were coated with approximately 80 nm of carbon at a pressure of about 1,3 m Pa in a Balzers vacuum coater equipped with a planetary sample rotator. Specimens tilted at 35° to the collector were examined using a Cambridge S180 Scanning Electron Mircroscope operating in the secondary electron collection mode at a nominal beam potential of 15 kV and beam current of 250 $\mu\alpha$. Micrographs were recorded on Ilford FP4 roll film at 120 second frame period and 800 lines per frame. An x-ray energy dispersive analyzer system, linked to the microscope, permitted the qualitative determination of sodium and heavier elements.

### Chemical analysis.

Two tests for uric acid were performed. In the murexide test[7], 3 drops of concentrated nitric acid were added to approximately 50 mg of the powdered stone in a porcelain evaporating dish and evaporated to dryness over a small flame. On cooling, a drop of $NH_4OH$ (2 mol/ℓ) was added. In the second test[2], one drop of $Na_2CO_3$ (1,9 mol/ℓ) and 2 drops of Folin's reagent were added to approximately 20 mg powdered sample and the colour noted. To test for the presence of ammonia, 50 mg of the powdered stone was heated with KOH (1,8 mol/ℓ) and a piece of damp red litmus paper was held at the mouth of the test tube[7].

Table 1: CHEMICAL ANALYSIS ON SERUM AND URINE

| DAY | SERUM | URINE | | |
|---|---|---|---|---|
| | uric acid (mmol/l) | uric acid (mmol/l) | SG | pH |
| – 120 | 0,11 | 2,1 | — | — |
| 0 | 0,34 | 2,0 | 1,025 | 7,33 |
| 3 | 0,30 | 2,3 | 1,023 | 7,32 |
| 6 | 0,33 | — | 1,022 | 6,25 |
| 13 | 0,27 | 2,1 | 1,020 | 6,27 |
| 19 | 0,27 | — | 1,005 | 6,18 |
| 29 | 0,18 | — | 1,010 | 6,29 |
| 38 | 0,28 | 2,2 | 1,012 | 6,55 |
| 43 | 0,23 | 4,0 | 1,016 | 6,49 |

## RESULTS OF THERAPY

Initiation of allopurinol therapy on the 12th day produced an immediate decrease in serum uric acid (Table 1) and an improvement in the patient's condition. The uric acid concentration of the serum taken 120 days earlier was less than half the concentration at time of surgery. This could not be related to any dietary or other change. The urinary uric acid concentrations remained unaltered but although included here, such figures are relatively uninformative without determination of the daily urine volume, measurement of which would have required a metabolic cage. A urinary SG of 1,025 or less has been proposed to minimise stone formation[16]. The SG remained below 1,025 throughout the period of therapy which indicates that adequate measures were taken to dilute the urine. An optimal pH of 6,5 has been advocated for treatment of urate lithiasis, and when pH drops below 5,75, administration of sodium bicarbonate is recommended[9 12 16]. The pH values remained at acceptable levels. The initially high pH of 7,33 was probably a consequence of the urinary infection. This was brought under control by antibiotic therapy and the pH subsequently dropped. The dog remained clinically healthy from the time of initiation of allopurinol therapy.

## RESULTS OF STONE ANALYSIS

### XRD.

The x-ray diffraction patterns recorded for both the inner and outer regions of the stone were identical. One such pattern is shown in Fig. 1. The relative intensities, I, and spacings, d, of the various reflections are presented in Table 2 together with the reference standards for sodium acid urate monohydrate, uric acid and ammonium urate[14]. The constituents of the stone were accordingly assigned as sodium acid urate monohydrate and uric acid.

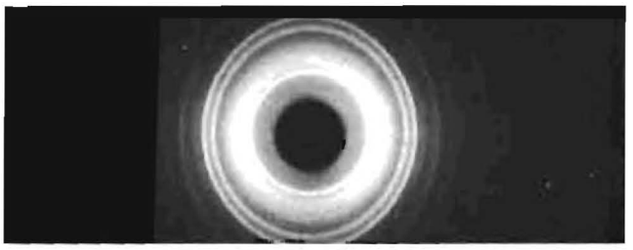

**Fig. 1:** X-ray powder diffraction pattern of stone sample.

Table 2: RELATIVE INTENSITIES AND SPACINGS

| Dalmatian | Sodium acid urate monohydrate | Uric acid | Ammonium acid urate | Assignment |
|---|---|---|---|---|
| d(A) I | d(A) I | d(A) I | d(A) I | |
| 7,80 VW | 7,5 W | | | NaUM |
| 5,54 M | | 5,63 W | 5,64 M | UA |
| 3,90 W | | | | |
| 3,44 M | 3,45 W | | 3,46 S | NaUM |
| 3,36 W | 3,36 W | | | NaUM |
| 3,11 S | 3,12 S | 3,09 S | 3,01 M | NaUM + UA |
| 2,68 VW | 2,69 W | | | NaUM |
| 2,54 W | | 2,57 W | | UA |

W: Weak, M: Medium, S: Strong
NaUM: sodium acid urate monohydrate, UA: uric acid

### SEM.

Two different crystalline morphologies were observed. Sodium acid urate monohydrate, characterized on the basis of energy dispersive analysis with respect to Na, was observed as rod-like crystals (Fig. 2) while uric acid was present in the form of fine, hair-like deposits (Fig. 3).

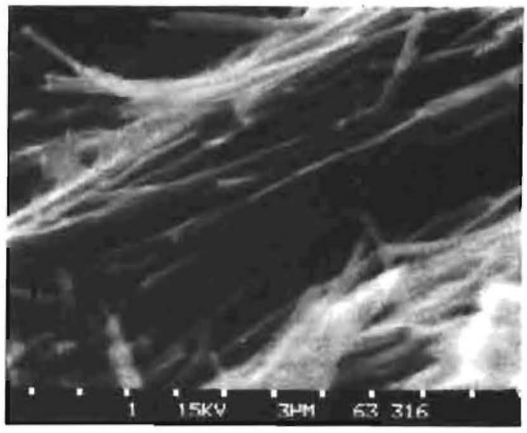


**Fig. 2:** Scanning electron micrography of rod-like sodium acid urate crystals6

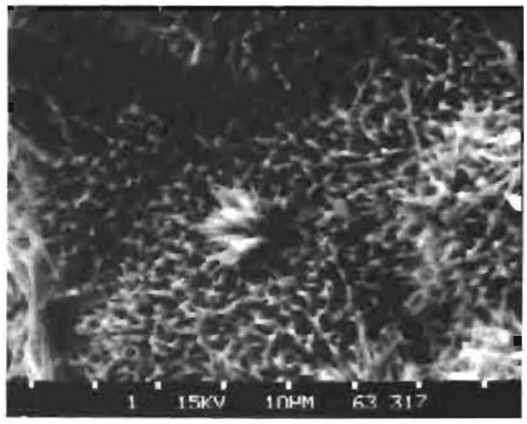


**Fig. 3:** Scanning electron micrograph of fine, hair-like uric acid crystals.

### Chemical analysis.

On evaporating to dryness in the murexide test, a yellow residue, characteristic of uric acid (or its sodium of ammonium salt), remained. The appearance of a purplish red colour on the addition of $NH_4OH$ together with the deep blue colour obtained with Folin's reagent further confirmed its presence. Ammonia was not detected by smell or by any action on the litmus paper indicating the absence of ammonium urate from the calculus.

## DISCUSSION

### Prophylaxis

Reduction of urinary uric acid concentration is the most pertinent approach to prophylaxis of urate calculus disease following surgical removal of the calculi[9 12 16]. Allopurinol inhibits the enzyme xanthine oxidase, thus decreasing the conversion of xanthine and hypoxanthine to uric acid. A possible complication of allopurinol therapy would be xanthine stones. However the drug has been used by Osbaldiston and Lowrey[8] and two of the authors (Briggs and Harley, Dept. of Chem. Path., U.C.T., 1980 Unpublished observations) at high doses and for long periods with no side-effects. The induction of polyuria dilutes the urine and hence reduces the concentration of urinary acid[9]. This was achieved in the present instance by intravenous fluids in the postoperative phase and thereafter by addition of sodium chloride to the food[9 16]. Apart from salting the food, dietary treatment of urate urolithiasis is of questionable value. Low purine diets have been proposed by some[8 16] but have not been found by others[3 19] to be effective in reducing urinary uric acid concentrations. In the present instance the effectiveness of the therapy was reflected better by the serum urate levels than by urinary urate concentration measurements, the latter being of necessity variable in random urine specimens. It is somewhat puzzling however, that the observed decreases in urinary SG during the period of therapy are not accompanied by a corresponding fall in urinary urate concentrations.

Alkalinization of the urine by the addition of sodium bicarbonate in the food has been suggested as a way of reducing the concentration of ammonium ions and thus preventing ammonium urate stone formation[12]. Alkalinization of the urine is also recommended in the prevention of uric acid stone in man[9] and one would assume, although it has yet to be shown, that it would assist in preventing uric acid stones in the Dalmatian. However some authors have questioned the value of urine alkalinisation since urate stones in Dalmatians have been found in alkaline urine[9 10 16]. This will not be resolved until the pH of urine can be measured at the time of initiation of stone formation and before infection secondary to established calculi influences urinary pH.

### Stone analysis

The occurrence of sodium acid urate and uric acid rather than ammonium acid urate is somewhat surprising since it is the latter component which has been reported by other workers. None of these however used x-ray powder diffraction (XRD) as a means of analysis. White et al.[17] reported the incidence of urate in 9 out of 9 Dalmatian calculi after analysis by microchemical, chromatographic and spectroscopic procedures while in a later paper the same author does not indicate which techniques he used in establishing the occurrence of urate in 12 out of 12 stones[18]. Brown[5] found 13 out of 17 Dalmatian calculi to contain urate and used an analysis kit to establish this. Only Sutor and Wooley[15] used XRD and found UA as a major component with ammonium acid urate in one calculus. XRD is today regarded as the procedure of choice in stone analysis and we have already used it successfully for the identification of ammonium acid urate calculi in a cross-bred Yorkshire terriet[13]. It is therefore conceivable that the dearth of reports of sodium acid urate and uric acid in Dalmatian calculi is due to the failure of earlier workers to detect the presence of these components.

### ACKNOWLEDGEMENTS

We would like to thank the Medical Research Council and the University of Cape Town for the award of research grants and Drs Davidson and Futter for performing the surgery.

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## ANIMAL FEED CONTAMINATION: THE VETERINARIAN'S RESPONSIBILITY

Animal feed contamination with chemicals, mycotoxins, and heavy metals has become a major problem in the United States. Of great importance is the rendering and utilization of carcasses of animals that have died from such contaminant-caused toxicoses. The rapid distribution and incorporation of contaminated animal materials rendered and processed into animal feeds make it likely that the effects of such contamination will become widespread in the food chain.

In the past there have been veterinarians who were aware of, or at least suspicious of, a toxic contamination problem and did not attempt to prevent the rendering of suspect dead or dying animals until after a positive diagnosis was made. To prevent the rendering and distribution of rendered by-products prior to diagnosis, veterinary practitioners must assume the responsibility of advising producers of the potential danger by:

1) Notifying appropriate state of federal regulatory officials immediately when multiple deaths or other animal losses occur which suggest toxicosis. If the carcasses have reached the renderer, state officials should inform the renderer of the potential danger of the products and indicate what actions are necessary before further processing can be permitted.
2) Informing the producer that toxicosis is suspected and that the rendering process must be controlled. The veterinarian should:
   (a) Notify the producer in writing that the carcass should not be shipped to a renderer until a diagnosis is confirmed or the shipment is approved by regulatory officials.
   (b) Recommend that the owner bury or incinerate the carcass in a manner approved by state regulatory officials.

*–AVMA Council on Public Health and Regulatory Veterinary Medicine, Sept 18, 1981.*

**CASE REPORT** **GEVALVERSLAG**

# ORAL VERRUCOUS CARCINOMA IN TWO DOGS

I.B.J. VAN RENSBURG*

ABSTRACT: Van Rensburg I.B.J. **Oral verrucous carcinoma in two dogs.** *Journal of the South African Veterinary Association* (1982) 53 No. 3, 209-210 (En) Department of Pathology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

Verrucous carcinoma was diagnosed in the oral cavity of 2 unrelated dogs. The neoplasm in both cases recurred after surgical excision. Autopsy of one of these did not reveal any evidence of metastasis.

Key words: Verrucous carcinoma, oral neoplasia, canine.

## INTRODUCTION

Although verrucous carcinoma is not very rare in man, the first case in a domestic animal was reported only in 1981 by Lownie et al.[3] who described a case in the maxilla of an 18-week-old Pyrenean Mountain dog. This report concerns 2 further canine cases of oral verrucous carcinoma, one in a middle-aged male crossbred and the other in a 12-year old male Beagle.

## CASE DESCRIPTIONS

The middle-aged crossbred dog was presented with an oral tumour approximately 10 mm in diameter in the gum just behind one of the upper canine teeth. The tumour had a smooth appearance, was covered by intact oral mucosa and projected beyond the normal surrounding tissue. It clinically resembled an epulis. Upon surgical excision it was found to be very infiltrative and had invaded the maxillary bone. The surface where the tumour had been removed was extensively hyfrecated. Within 4 weeks after its removal it regrew to about twice its original size and then had a dark red mottled rough surface somewhat resembling that of a cauliflower. At this stage euthanasia was performed at the owner's request and an autopsy was performed to determine whether metastasis had taken place – with negative results. Both the original and regrown tumours were examined histopathologically and were diagnosed as verrucous carcinoma.

In the Beagle an oral tumour had been present in the gum ventral to the lower incisors for approximately 3 months before veterinary advice was sought. Macroscopically the tumour was approximately 15 × 8 mm in size, had a rough surface and revealed evidence of secondary bacterial infection. On surgical removal the neoplasm was found to be very infiltrative and it proved impossible to excise all neoplastic tissue. An attempt to destroy the remaining tissue by electrical cautery was, however, made. Verrucous carcinoma was diagnosed on histopathological examination. The tumour recurred within 2 months. The owner requested euthanasia but no autopsy was performed.

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### Microscopical appearance

Histopathological examination showed that the tumour consisted of folds of neoplastic epithelial cells originating from the surface epithelium. The original tumour from the cross-breed revealed broad sheets of epithelial cells with scant fibrous stroma in between and no evidence of keratinisation (Fig. 1) while the recurrec version consisted of narrower rete with a frond-like appearance (Fig. 3 & 4) and did show a tendency towards keratinisation in the folds (Fig. 2). The stroma in this case contained fairly large numbers of a mixed leukocytic infiltration. Mitotic figures were fairly common in the cells of the stratum spinosum of the neoplasm. No keratin pearls as in typical squamous cell carcinoma were noticed. The appearance of the neoplasm from the Beagle was similar except that in some areas the peripheral cells were arranged in pallisades thus resembling those present in some solid basal cell tumours.

## DISCUSSION

In man the occurrence of the tumour is associated with the habit of chewing tobacco or keeping snuff in the mouth[1 2]. In dogs a similar association obviously cannot play a role and no theories on the aetiology can be postulated. The neoplasm is more common in elderly people and the cases described here were also older animals, whereas the case reported by Lownie et al.[3] occurred in a very young dog.

In humans recurrence after surgical removal is common (about 75 %)[1]. The rapid recurrence of the neoplasms in the 2 cases reported in this paper as well as the very infiltrative nature of the neoplasm observed in these 2 cases and in the case reported by Lownie et al.[3], suggests that the incidence of recurrence in dogs after surgical excision will probably be very similar to that in humans.

Verrucous carcinoma in man is described as a rare variant of squamous cell carcinoma of elderly patients which grows slowly, deeply invades the jaws but rarely metastasizes[2]. According to Anderson the tumour is well differentiated but very destructive[1].

From a differential diagnostic point of view verrucous carcinoma could possibly be confused with epulis and squamous cell carcinoma. To differentiate it


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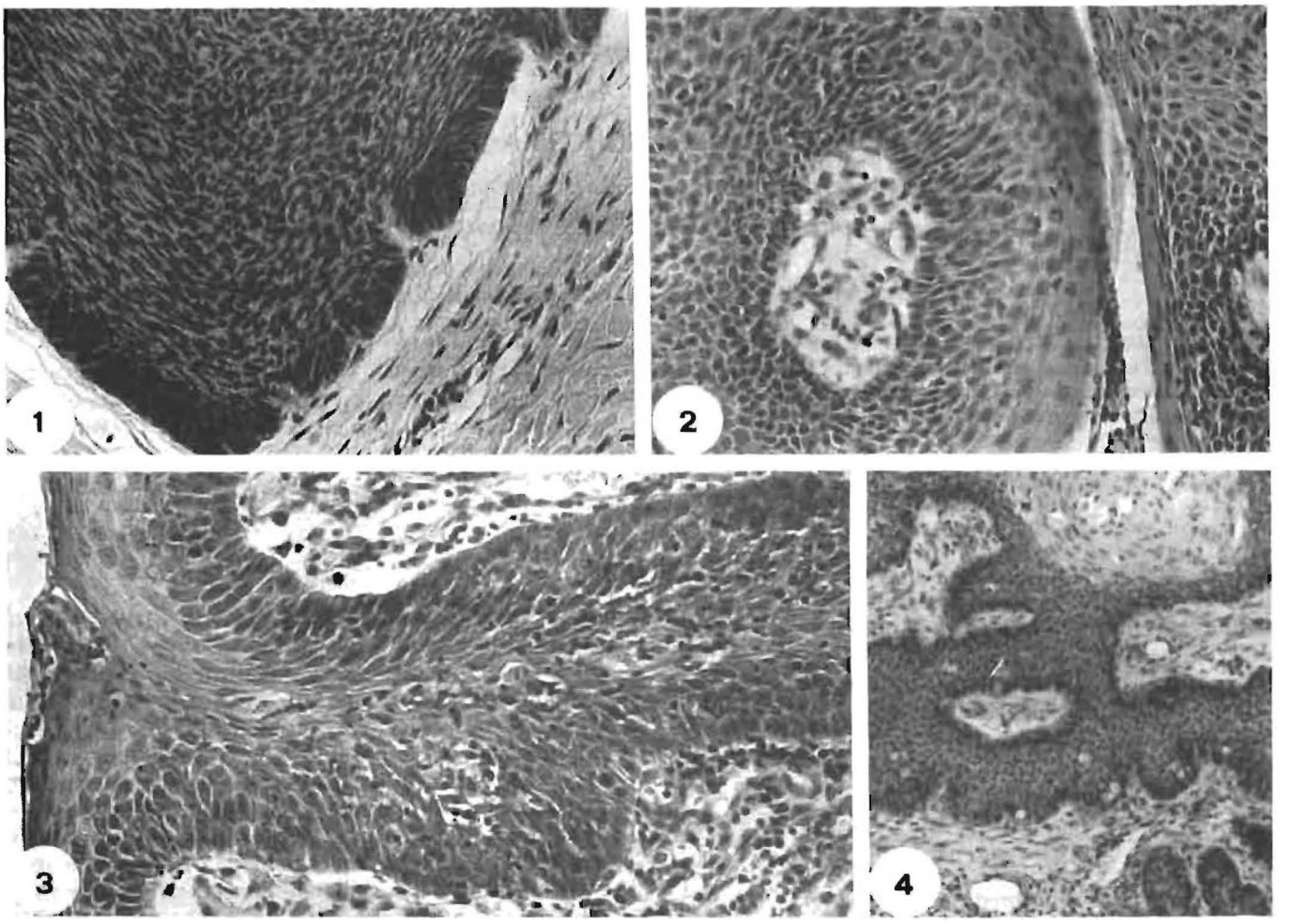


**Fig. 1:** Broad sheet of squamous epithelium growing down into the underlying fibrous tissue.

**Fig. 2:** Tendency towards keratinisation in the folds of the recurred tumour.

**Fig. 3:** Band of invasive squamous cells keeping contact with the surface epithelium. Note the infiltration of leukocytes into the stroma.

**Fig. 4:** Sheets of neoplastic cells showing pallisading of peripheral cells.

from the pseudoepitheliomatous hyperplasia of an epulis, the cells in the former are usually more dysplastic and the mitotic index is higher. Verrucous carcinoma is in addition more invasive. In differentiating it from squamous cell carcinoma the basement membrane of the rete in verrucous carcinoma remains intact and the rete always retains contact with the surface epithelium, whereas nests of epithelial cells break away and become isolated in squamous cell carcinoma. The cells in the squamous cell variety also have a more anaplastic appearance, while the presence of keratin pearls is, of course, typical.

As far as surgical treatment is concerned excision should be very wide. According to Lownie et al.[3] radiotherapy is contraindicated because of the danger of transformation to anaplastic carcinoma.

ACKNOWLEDGEMENTS

Drs C. Irvine-Smith of Westville and C.B. McDonald of Fishhoek are thanked for supplying clinical data and histories. Prof. R.C. Tustin is thanked for criticism of the manuscript, Mrs V. Käber for the typing thereof and Mr P.L. Meyer for photography.

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CASE REPORT GEVALVERSLAG

# CLOSTRIDIAL MYOSITIS IN A HORSE

J. VAN HEERDEN* and W.S. BOTHA**

ABSTRACT: Van Heerden J.; Botha W.S. **Clostridial myositis in a horse.** *Journal of the South African Veterinary Association* (1982) **53** No. 3, 211 (En) Department of Medicine, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

*Clostridium septicum* infection in a horse resulted in severe gangrenous myositis. The infection was characterized clinically by crepitating, deep subcutaneous swellings and signs of severe pain.

Key words: *Clostridium septicum*, equine, myositis.

## DESCRIPTION OF CASE

An approximately 10-year-old nondescript grey gelding was admitted to the Department of Medicine, Faculty of Veterinary Science, University of Pretoria, for further investigation into the cause of recurrent colic. It had been treated twice during the 24 hours before admittance by the intramuscular injection of an analgesic (Analate, Hoechst) into the muscles of the neck.

On admittance the horse showed no signs of pain and, apart from being slightly depressed, no clinical abnormalities were detected. Eighteen hours later the animal was found prostrate, shivering, shaking and sweating profusely. Well circumscribed crepitating swellings were observed on the lateral aspect of the neck as well as on the lateral aspect of the left fore leg, over the region of the distal humerus. When the horse was encouraged to stand up, it could only do so with difficulty and without supporting itself fully with its left fore leg.

No favourable response was observed to the intravenous administration of high doses of pethidine (Centaur Laboratories), Analate (Hoechst) and xylazine (Rompun, Bayer).

The animal's condition rapidly deteriorated until it was euthanased approximately 4 h later in a state of severe shock and general collapse.

The most outstanding lesion on necropsy was multifocal areas (100-200 mm in diameter) of a gangrenous myositis on the left side of the neck. These were black in colour and porous due to gas bubbles which separated the muscle bundles and localized in the musculature of the left side of the neck. A marked stenosis of the left dorsal colon was also found.

Examination of smears from the affected muscle showed numerous clostridial organisms and these were demonstrated to be *Clostridium septicum* by specific fluorescence with fluorescein labelled antiserum (Clostridial diagnostic antisera fluorescein labelled; Wellcome Diagnostics, P.O. Box 654, Kempton Park).

## DISCUSSION

Relatively little has been published on clostridial myositis in horses[1-3]. In a case described by Westman et al.[3] a non-fatal myositis of the neck region resulted from a mixed infection of *Cl. septicum* and *Cl. chauvoei*. Successful treatment was achieved by the use of a penicillin-dihydrostreptomycin combination. McLaughlin et al.[2] reported on a fatal case which was initially treated for mild colic. In this case there was no external evidence of infection.

Clostridial organisms normally occur in the soil or in the intestinal tract of animals. They or their toxins most commonly enter the body through breaks in the skin or via the digesive tract. In the case described here it is possible that the organism was introduced into the neck muscles by means of the intramuscular injections. The grave prognosis of a clostridial infection in the horse as well as the possibility of introducing the infection by injection underlines the necessity of always maintaining aseptic injection techniques.

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**TO THE EDITOR** **AAN DIE REDAKSIE**

# HEMOLITIESE ANEMIE IN 'N MINIATUUR DACHSHUND VEROORSAAK DEUR INNAME VAN GROOT HOEVEELHEDE UIE (*ALLIUM CEPA*)

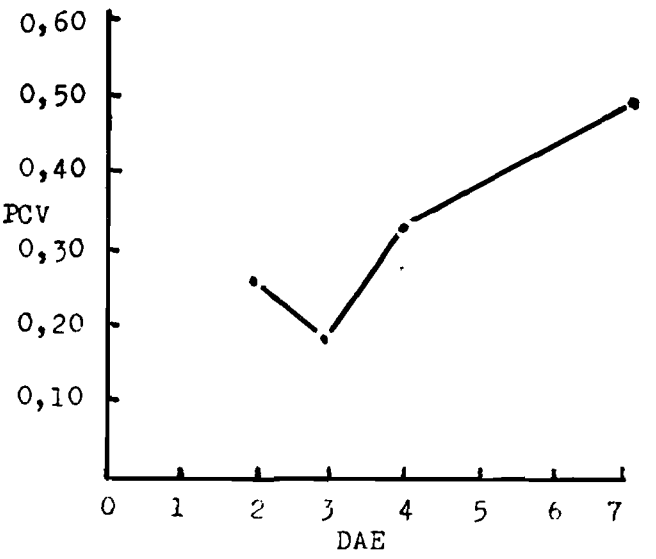


**Fig. 1:** Hematokritwaardes gedurende 7 dae nadat uie weggelaat is uit die diëet. Tussen dag 3 en dag 4 is 150 ml. bloed toegedien. Let op die goeie regeneratiewe respons teen dag 7.

'n Twee-jaar-oud miniatuur Dachshund reun is na my gebring weens anoreksie en depressie. Kliniese ondersoek het hemoglobienurie, geel stoelgang en 'n temperatuur van 40 °C getoon. Daar was geen ikterus nie. Galkoors is vermoed, maar geen bloedparasiete kon gevind word op bloedsmeer gekleur met Stevenol's kleur nie. Die hond is toe simptomaties behandel met ampicillin (Penbritin, Beecham) en tioktiese suur (Tioctan-Vet, Panvet).

Die volgende dag was die hond se temperatuur laer, nl. 39 °C, maar die habitus en simptome was onveranderd. 'n Bloedsmeer is vervolgens gestuur na 'n laboratorium vir dierlike kliniese patologie. Die smeer is gekleur met Cam's Quick-Stain en daar is gevind dat ongeveer 92% rooibloedselle Heinz liggaampies bevat. Hierdie liggaampies kom voor in die bloed van honde in gevalle van hemolitiese anemie waar 'n oksideermiddel die hemoglobien denatureer[1].

Navraag is toe gedoen na die hond se diëet. Daar is vasgestel dat die hond gewoonlik gekookte maalvleis en wortels kry. 'n Nuwe bediende het egter sowat 2½ maande tevore die kos begin voorberei, en sy het groot hoeveelhede uie bygevoeg. Die hond het dus in werklikheid 'n bredie bestaande uit vleis, wortels en uie gekry. Uie bevat 'n oksideermiddel wat hemolitiese anemie kan veroorsaak[2,3]. Die eienaar is toe aangeraai om die uie weg te laat uit die kos.

Omdat daar so baie Heinz liggaampies in die bloed was, is daar verwag dat die hond nog baie anemies sou word. Twee dae later het die oogmukosa sigbaar bleek geword, en die hematokrit is bepaal. Dit was 0,25. Toe die hematokrit die volgende dag na 0,18 gedaal het, is 150 ml. bloed binne-aars toegedien. Van hierdie oomblik af het die hond se habitus en aptyt verbeter. 'n Week na die uie uit die kos weggelaat is, was die hematokrit 0,50. Sien Fig. 1.

Die eienaar het ook 'n 6-maande-oud miniatuur Dachshund teef. Sy het geen kliniese simptome van hemolitiese anemie getoon nie: haar hematokrit was 0,42 en daar was slegs 8 % rooibloedselle wat Heinz liggaampies bevat het. Die rede waarom sy nie siek geword het nie, is waarskynlik omdat sy elke oggend pap gevreet het en nie net uitsluitlik uie bredie nie. Die reun het geweier om pap te vreet.

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0181 Pretoria.

## VERWYSINGS

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## TO THE EDITOR — AAN DIE REDAKSIE

# OXYTETRACYCLINE PLASMA LEVELS IN DOGS

I would like to deliver comment on the paper 'Oxytetracycline Plasma Levels in Dogs after Instramuscular Administration of Two Formulations' by A. Immelman and Gillian Dreyer in the Journal of the SAVA 52 No. 3 September 1981: 191-193.

There are 2 salient points:

Table 1 is wrongly headed – as the table itself shows the Terramycin 100 is a polyvinyl – pyrrolidone formulation and not propylene glycol.

Pfizer Laboratories definitely recommends that polyvinylpyrrolidone as well as Terramycin Long Acting not be used in canines. The allergic response to the PVP described by Professor Immelman bears out this viewpoint.

C.W. Moore
Veterinary Technical Advisor
Pfizer (South Africa)
P.O. Box 783720
2146 Sandton.

## AUTHORS' REPLY

Regarding the 2 salient points mentioned by Dr C.W. Moore: He is quite correct as far as the error in the heading of Table 1 is concerned, although in the table itself PVP is indicated. We apologise for this error but are sure that your readers had no problem in understanding the contents of the table.

We did not comment on the recommendations of the specific company. We must point out that other manufacturers using PVP formulations do recommend their products for small animal use and it has come to our notice that Terramycin LA is used in dogs.

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## BOOK REVIEW — BOEKRESENSIE

# DISEASES OF THE REPTILIA

Edited by: J.E. COOPER and O.F. JACKSON

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This two-volume set is certainly the most comprehensive and authoritative work yet produced on the diseases of reptiles, and offers something for researchers in every discipline of veterinary science. The first volume initially describes the anatomy and physiology of these animals and proceeds to describe the various techniques commonly employed in histopathology, microbiology and parasitology. The diseases caused by virusses, bacteria, fungi, actinomycetes, protozoa, and ecto- and endoparasites are described in some detail. The second volume deals with traumatic conditions, nutritional factors, neoplasms, and lists many congenital and developmental disorders, while clinical aspects, anaesthesia, surgery and numerous drugs and their doses are also dealt with.

It is impossible to provide detailed descriptions of all the diseases of reptiles, and the editors also state that these books are intended as a guide to the diseases particularly, but not exclusively, of captive reptiles. The various chapters are written by specialists in their field and being a parasitologist I was impressed with the thoroughness of the sections in which parasites were dealt with. Numerous references are provided at the end of each chapter. Some sections may be difficult for non-veterinarians to interpret and understand, since they are written in technical language, which assumes previous knowledge of the subject.

The editors and authors are to be congratulated on providing a work that is easy to read, logically arranged and amply illustrated. For veterinarians who wish to add to their knowledge, zoo veterinarians, herpetologists and zoologists this is an invaluable work and is highly recommended.

J.D.F. Boomker

**TO THE EDITOR** **AAN DIE REDAKSIE**

# HEPARIN DEGRADATION BY *EUBACTERIUM* AND *PEPTOSTREPTOCOCCUS* SPECIES FROM BOVINE ENDOMETRITIS

The possible role of obligate anaerobic bacteria in the aetiology of bovine endometritis has not been well studied. This may be partly due to the strict requirements of these organisms for anaerobiosis, especially during sampling and primary isolation and the fact that the specialized techniques and equipment required for successful isolation of strict anaerobes are not routinely available.

We have recently studied 6 cows which contracted purulent endometritis shortly (± 3 weeks) after calving. They all belonged to a large commercial Friesland dairy herd (± 400 cows) maintained on the Transvaal highveld. Intra-uterine pus was aspirated by means of a sterile disposable insemination pipette inserted aseptically through the cervix with the help of a speculum. Pus was massaged from the uterus via the pipette and a mid-stream sample was then collected by attachment of a disposable syringe to the pipette. The pipettes and syringes were preflushed with anaerobic $CO_2$. Incubation, isolation and identification of anaerobes were done according to procedures previously described[2,4].

*Escherichia coli* and *Corynebacterium pyogenes* were isolated from all 6 cows. Four of the 6 (66 %) cows studied yielded one or more species of anaerobic bacteria (Table 1).

Table 1: HEPARINASE PRODUCING ANAEROBES FROM BOVINE ENDOMETRITIS*

| Cow | Anaerobes | Heparinase Activity |
|---|---|---|
| 1 | Negative | |
| 2 | *Eubacterium saburreum* | + |
| | *Eubacterium lentum* | − |
| 3 | *Eubacterium alactolyticum* | + |
| 4 | *Eubacterium* sp. | − |
| | *Peptostreptococcus* sp. | + |
| 5 | Negative | |
| 6 | *Veillonella parvula* | ND |
| | *Megasphaera elsdenii* | ND |

*Preliminary specification according to Holdeman et al.[4]
ND = Not examined

In view of their possible importance in both intra-uterine infections and in bovine mastitis[2] we are at present investigating virulence factors of asporogenous obligate anaerobic bacteria. Routine scanning of these organisms for their ability to degrade heparin[3] has revealed that 3 of the 6 (50 %) cows were infected with heparinase producing anaerobes (Table 1). The significance of this characteristic needs to be further studied. As far as we can ascertain, heparinase has only been reported for certain Gram negative bacteria viz: *Flavobacterium heparinum*[6] (a soil saprophyte) and a few species belonging to the pathogenic genus *Bacteroides*[7].

Because of the marked power of heparin to inhibit blood coagulation by activating the $\alpha_2$-globulin proteinase inhibitor AT-III, which in turn progressively inactivates both thrombin and factor Xa, key enzymes in the blood coagulation cascade[5], its inactivation by inter alia bacterial heparinase may have serious consequences. An aetiological relationship between anaerobic heparinase producing Gram negative bacteria and thromboembolic disease has been reported in humans[1].

To summarise we have found:

1. Various obligate anaerobic bacteria not previously reported in a possible aetiological role from purulent bovine endometritis.
2. Heparinase activity in Gram positive anaerobic bacteria not previously reported in the genera *Eubacterium* and *Peptostreptococcus*. Heparinase activity in these organisms may perhaps be a virulence factor in the aetiology of certain anaerobic infections.

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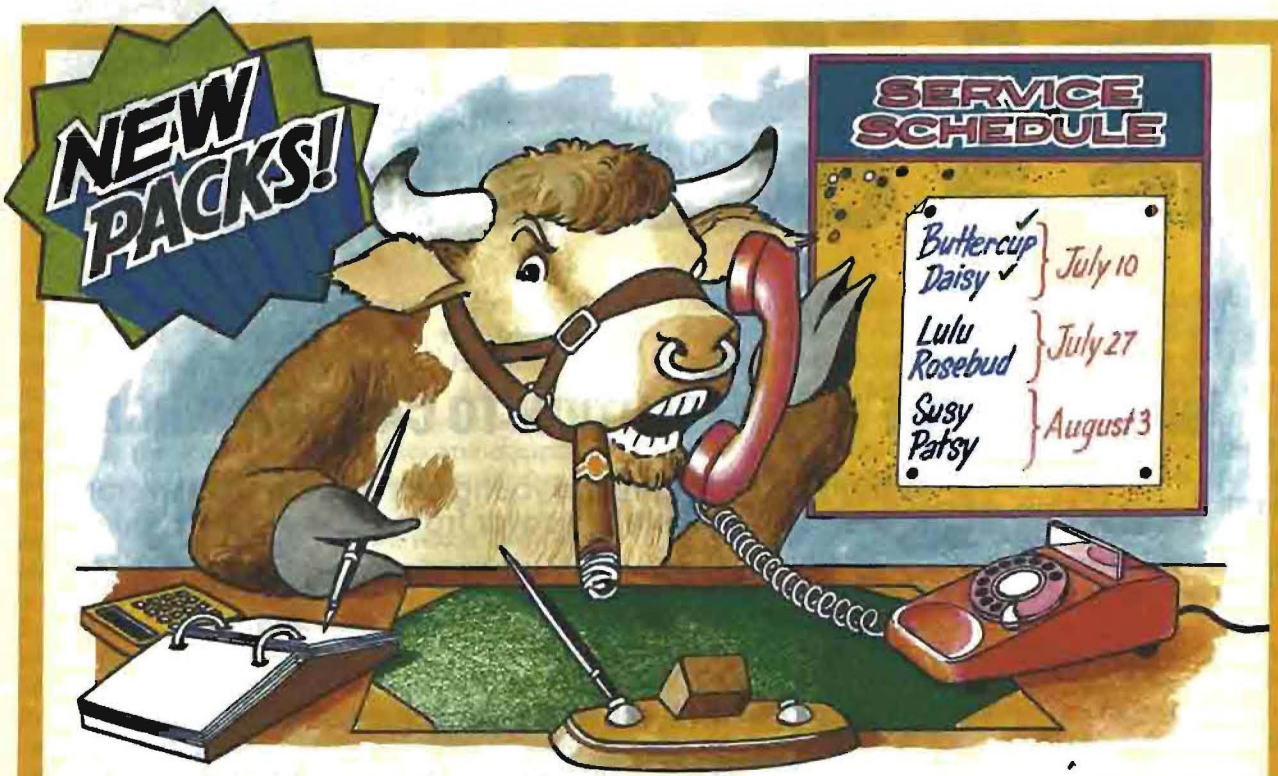


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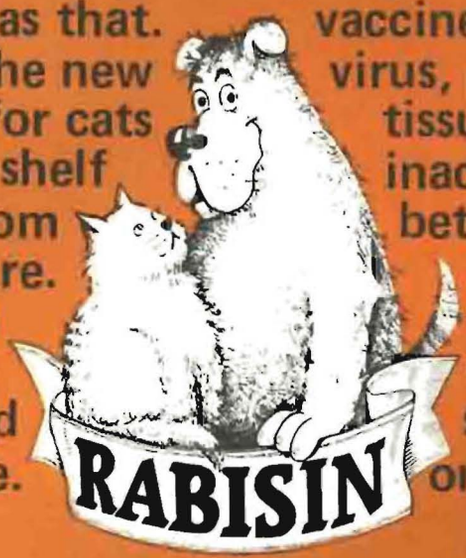


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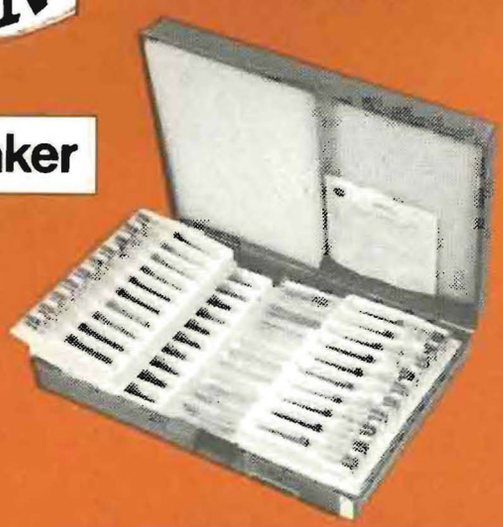

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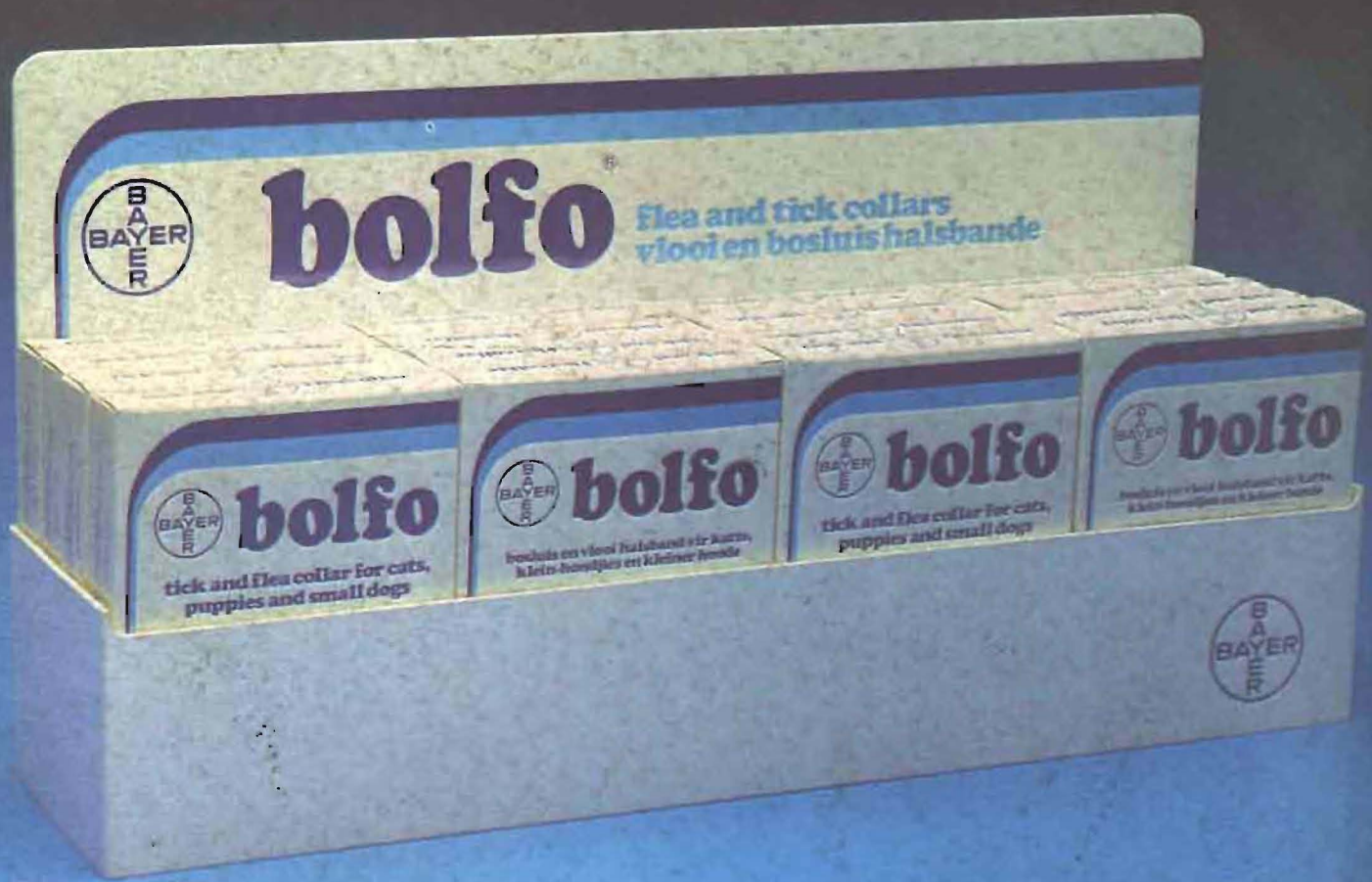


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