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ADDRESS **VOORDRAG**

DIE ROL VAN DIE APTEKER IN DIE MODERNE SAMELEWING*

P.R. VAN DER MERWE

INLEIDING

As aanvangspunt moet dit aanvaar word dat die tradisionele rol van die apteker as menger en samesteller van medisyne, in die moderne samelewing totaal verander het tot die van bewaarder of beheerder en raadgewer oor medisyne.

Die professie het verander van die van 'n vakmanskap na die van 'n wetenskap. In die tye waarin ons vandag lewe, met die geweldige tekort aan opgeleide mannekrag en die strewe na die beter benutting van opgeleide mannekrag en met inagneming van die hoë kostes hieraan verbonde, is dit ook duidelik dat die apteker en sy kennis onder-waardeer word en dat hy ook nie tot sy volle potensiaal in die totale gesondheidsdiens van die gesondheidspan van die land en sy bevolking benut word nie.

Teenstrydig met populêre opvattings het die belangrikheid van die apteker en die dienste wat hy kan lewer as gevolg van hoogs ontwikkelde en gevaarlike middels wat hanteer word en moderne gesofistikeerde wetgewing wat die beheer oor medisyne uitoefen, eerder toegeneem as verminder.

Ter inleiding, wil ek graag statistiek aanhaal oor die verspreiding van aptekers en apteke, en kortliks die apteker se opleiding en sy regsaamspreeklikheid behandel.

(i) Statistiek

Van 'n totaal van 5 333 aptekers in S.A. Afrika is
3 3164 in die kleinhandel in net oor 2 050 apteke,
126 in die groothandel,
354 in die vervaardingsbedryf,
57 in staatshospitale,
528 in provinsiale Hospitale.
58 in privaat hospitale,
14 in sendinghospitale.
72 in S.A.S. & H Siekefonds Afdelings.
13 in ander siekefondse.
96 in akademiese opleiding.
91 in administrasie,
304 is nie-praktiserend,
246 is afgetree en
210 tree op ander gebiede op.

Van die aptekers is 60% in die kleinhandel.

(ii) Opleiding

Die syllabus vir die kursus wat die apteker moet voltooi word neergelê deur die Aptekersraad en die kursus mag gevolg word aan 'n erkende Universiteit of Kollege vir Gevorderde Tegniese Onderwys waarvan daar 10 altesaam in die land is. Dit neem vier jaar voltydse studie in beslag en een jaar ingeskrewe kwekelingskap by 'n praktiserende kleinhandelapteker, of in 'n hospitaal, laboratorium, vervaardiger of in die groothandelbedryf. Vakke sluit onder andere in Chemie, Fisika, Farmakologie, Fisiologie, Gesondheidsvoorligting, Farmakognosie en bestuurs- en kommunikasie wetenskappe.

(iii) Legal Responsibility

The parmacist, after qualifying, has to register with the S.A. Pharmacy Board. This is a statutory body and he immediately falls under the discipline of the Board and the Ethical Rules laid down by that body which functions and controls pharmacists in terms of the Pharmacy Act, Act No. 52/1974.

A pharmacist found guilty of improper or disgraceful conduct faces severe penalties which could lead to suspension or removal of his name from the register – in which case he can no longer practice as a pharmacist.

In terms of the Medicines and Related Substances Control Act, Act 101/1965 only certain persons may sell, supply, import or manufacture medicine under certain conditions.

The pharmacist is the main supplier and dispenser of medicine, the custodian of medicine. His premises has to be registered with the S.A. Pharmacy Board and is inspected regularly by inspectors from the Department of Health. While the pharmacy is open for business the premises have to be under the continuous personal supervision of a pharmacist.

To sell or supply medicine involves grave legal responsibilities and the pharmacist has to be an expert in all the laws governing the sale of medicine and the records that have to be kept, e.g. a prescription book and the Schedule 7 Substances Register. Act 101/1965 is very sophistacted legislation and the pharmacist must know the Act and the regulations in its entirety, specifically:

(1) the conditions under which he may supply and sell medicine;
(2) the labelling requirements in respect of all medicines supplied;
(3) the classification under nine schedules of all the prescription medicines available in South Africa and the regulations governing the sale of each medicine according to its particular classification.
These conditions become stricter the higher the schedule number goes. Scheduled medicines comprise thousands of items and combinations of chemically active substances. These schedules are subject to amendments which occur at least twice a year and with this the pharmacist has to keep up to date.
(4) The regulations under the Act which state clearly that records must be kept of all scheduled medicines supplied. To illustrate I refer to certain court rulings in America on the pharmacis's responsibilities. (This would equally apply in South Africa).

In compounding medicines and filling prescriptions a

*Gelewer tydens SAVV-kongres te Grahamstad 1977.

pharmacist requires a degree of vigilance and prudence commensurate with the dangers involved. In the case Troppi v Scarf, where a tranquillizer was dispensed instead of a prescribed oral contraceptive, the Court ruled that "A pharmacist is held to a very high standard of care in filling prescriptions. When he negligently supplies a drug other than the drug requested, he is liable for resulting harm to the purchaser."

In the case McLeod v M.S. Merrell Company, where the dispensing pharmacist of the company did not share in the liability for harmful side-effects, the Court established precisely what a pharmacist warrants when he sells a precription:

(1) he has compounded the drug prescribed;
(2) he has used due and proper care in filling the prescription; including giving the correct directions for use;
(3) that the proper methods were used in the compounding process; and
(4) that the drug has not been affected with some adulterating foreign substance.

To reiterate, a pharmacist may be liable if he misinterprets a physician's order, or if he errs by filling the prescription with the wrong drug or the wrong strength. Likewise, he is responsible for the quality and purity of the drugs dispensed. In addition, in all jurisdictions, to one degree or another, there are regulations regarding drug storage, prescription labelling, and the use of proper containers. Finally, anti-substitution legislation is designed to prevent the pharmacist from determining which brand to select.

DIE ROL VAN DIE APTEKER

Gebaseer op al die huidige beskikbare dokumentasie is daar 4 belangrike rolle wat gereeld met aptekers geassosieer word of wat aptekers reeds in die praktyk behartig of voel dat hulle moet behartig en wat hulle kollegas in die geassosieerde professies graag sou wou sien dat hulle behartig.

(i) Eerstens die rol as beheerder van alle medisynebronne en inligting oor medisyne

Hierdie rol vereis voortdurende aanspreeklikheid vir al die fasette van die medisynehanteringsproses. Dit sluit die aanskaffing, berging, kontrole, keuse, voorbereiding, reseptering en verspreiding van medisyne en verwante gesondheidsitems in, waar gesondheidsdienste ookal gelewer mag word. Die apteker beskik in hierdie rol die vermoë om geïntegreerde verspreidingsisteme vir medisyne te ontwerp, binne sowel as buite die inrigtingsopset. Hy is vaardig in die doeltreffende en ekonomiese bestuur van hierdie sisteme. Hy is hierby ook in staat om gepaste kosteberekenings te maak en kontrole aan te wend vir die verbetering van beide pasientesorg en monetêre bestuur.

Hy is in staat om die mannekrag te werf, op te lei, personeel te voorsien en om toesig te hou oor die werkspan wat nodig is om farmaseutiese dienste te verskaf in enige van die verskillende dienste wat gelewer word, bv mediese venootskappe, dienste vir gesondheidsinstandhouding, hospitaalorganisasies ens.

As beheerder van medisynebronne is hy in staat om tegnologiese programme wat benodig mag word te ontwerp en te implementeer soos bv intraveneuse oplossings, toevoegingsdienste en ander farmaseutiese dienste wat deskundige bekwaamheid vereis. Hy kan ten volle outomatiese tegnieke ten opsigte van pasiënterekordssisteme toepas en dit in sy voortgesette inligtingsprogramme integreer.

(ii) Tweedens, die rol van Gesondheidsopvoeder

Dwarsdeur die geskiedenis tree die apteker as die opvoeder van die verbruiker op, alhoewel hy hierdie rol nie so benut soos wat hy miskien kon nie. Hier stel hy aktief belang in raadgewing en advies oor medisyne en verwante gesondheidsaangeleenthede aan die gemeenskap. Dit sluit die volledige spektrum van voorskrifmiddels, nievoorskrifmiddels en die misbruik van verdowingsmiddels in. Aangesien hy gewoonlik die lid van die gesondheidsprofessies in sy gemeenskap is wat die maklikste bereikbaar is, is die apteker die logiese persoon om die belangrike verantwoordelikheid van die gesondheidsopvoeding van die gemeenskap te aanvaar. 'n Verantwoordelikheid wat hy met geneeshere, verpleegsters, openbare sowel as staatsamptenare deel.

Met sy kennis van persoonlike sowel as openbare gesondheidsake as basis, is die apteker op bekwame wyse in staat om te praat oor prosedures vir beheerde medisynegebruik in die huis, medisyne gebruik en misbruik, voorkoming van vergiftiging, die vernietiging van ongebruikte medisyne, algemene higiëne en preventiewe geneeskunde, siektebespeuring, die behandeling van minder belangrike kwale, noodbehandeling, pediatriese immuniseringsroetines, beheer van veneriese siektes, gesinsbeplanning, gemeenskapsgesondheidsprogramme en burgerlike beskerming.

Die apteker kan hierdie inligting verskaf deur direkte persoonlike kommunikasie met pasiënte, deur die verspreiding van gedrukte inligting en pakkate, deur deelname aan openbare forums en deur verwysing.

Hierdie twee rolle – dié van beheerder van medisynebronne en dié van gesondheidsopvoeder – kan waarskynlik die beste beskryf word as die tradisionele rol van die apteker, aangepas by die veranderde sosiale behoeftes en eise wat op die oomblik op die gesondheidsversorgingsisteem gemaak word.

Die huidige kurrikulum vir die baccalareusgraad in farmasie, wat gewoonlik vier jaar professionele opleiding vereis, konsentreer skynbaar op hierdie twee rolle.

(iii) Derdens, die apteker in die rol van medisynekonsultant

Die Apteker word oor die algemeen erken as die kenner van medisyne en die werking daarvan. Tradisioneel behels die farmasie kurrikulum ook 'n sterk komponent van farmaseutiese chemie en farmakologiese kursusse met laboratoriumwerk, beperk tot dierestudies. Die rol van medisynekonsultant is vandag egter meer pasiëntgeoriënteerd en vereis 'n nuwe vlak van opleiding en onderrig wat menslike kliniese farmakologie en ondervinding in die gebruik van medisyne in 'n kliniese omgewing bv hospitale, en inrigtingsapteke sal insluit.

In so 'n rol beskik die apteker oor kennis van siekteprosesse en die betrokke behandeling daarvoor, administrasie, wisselwerkings, newe-effekte en die korrekte dosisaanpassings van medisyne.

So 'n apteker praktiseer gewoonlik in 'n omgewing waar hy tot die beskikking van geneeshere, verpleeg-

sters en ander gesondheidspersoneel is vir raadpleging oor aangeleenthede wat betrekking het op medisyne. In sommige hospitale doen hy mee tydens mediese rondtes, hy voer toelatingsonderhoude om die medisynegeskiedenis te bepaal en 'n pasiënte verslagkaart vir elke pasiënt op te stel, hy kontrolleer die medisyne inname van die pasiënt.

Gesentraliseerde medisyneinligtingsprogramme word al hoe meer in die inrigtingsopset geïmplementeer om 'n ondersteunende diens vir die konsultantrol te lewer. Raadpleging mag die vorm van aanbevelings aan die farmaseutiese en terapeutiese komitees van die mediese personeel aanneem met betrekking tot die aanskaffing van nuwe middels in die formuleboek en behels baie keer groepseminare en publikasies oor medisyneterapie.

(iv) Vierdens 'n rol in primêre versorging

Die apteker het tradisioneel baie van die elemente van primêre versorging voorsien, alhoewel dit nie in dié konteks erken is nie. Te danke aan sy medisynekennis word die apteker baie keer direk deur individue genader vir 'n "diagnose" van minder belangrike klagtes en siektes; hulle vra hom om 'n medisynepreparaat aan te beveel d.w.s. 'n "voorskrywende" funksie.

Dit was nog altyd aanvaarbaar dat 'n apteker beide diens en medisyne vir minder belangrike kwale sal voorsien, dit is toe te skrywe aan sy bereikbaarheid en aan die aard van sy praktyk.

Omdat die apteek as 'n in-stap fasiliteit oor lang periodes elke dag meer beskikbaar is vir die publiek as ander gemeenskapsdienssentra, is dit die logiese plek vir mense om inligting en voorligting te soek oor plaaslike gesondheidsversorginghulpbronne soos noodversorging en geneeskundige, tandheelkundige en veterinêre dienste.

Hierdie laaste twee rolle naamlik medisyneterapie konsultant en voorsiener van primêre versorging het een aspek in gemeen naamlik 'n kliniese of pasiënteoriëntering.

Aangesien die leerplan vir die baccalareusgraad nie ruimte of tyd toelaat vir hierdie hoë graad van kliniese beklemtoning nie moet die kurrikulum van die doktor van farmasie beskou word as die minimum opvoedingsvereiste wat benodig sal word vir hierdie kliniese rolle.

MODERN CONCEPTS ON THE ADDITIONAL ROLE OF THE PHARMACIST IN AMERICA

(i) Clinical pharmacy practice

The first is the growth of what has come to be called clinical pharmacy practice.

Clinical pharmacy is a label without adhesive; it will not adhere to any one concept. Literally, the term clinical implies bedside treatment, and undoubtedly, the first application of the term involved hospital pharmacy practice. The modern dictionary definition is "of or pertaining to direct observation and treatment of patients". Most modern pharmacy leaders will argue that the direct contact between pharmacist and patient over the prescription counter is sufficient to establish a clinical relationship. At the centre of either approach is the element of communication regarding medications. In the hospital it is the pharmacist's consultation with the physician and in the store it is the pharmacist's consulation with the patient.

Before we can look at the implications of clinical practice in community pharmacies, it would serve us well to consider it as a functional part of hospital pharmacy service. In a hospital, clinical practice includes activities such as taking medication histories, monitoring drug use, contributing to drug therapy selection and administration, and surveillance for adverse reactions and interactions. Viable programmes exist in most teaching institutions, and despite initial resistance by some members of medical staff, they appear to be growing in both scope and quality.

Although the programmes vary among institutions, most begin with the taking of a drug history by a clinical pharmacist as to any drug sensitivities or allergies, what drugs the patient was taking prior to admission, and what drugs the patient may have taken in the past. This interview concerns itself not only with prescription drugs, but with over-the-counter products as well.

Ordinarily, the next step is to review the updated patient chart, consider diagnostic work-ups and to attend the physician's diagnosis. A consultation form is then prepared with recommended courses of drug therapy and is attached to the chart. The physician may choose one of the alternatives, none of them, or may request more information. Another possibility is that the physician will order whatever drugs he wants to use, and the services of the clinical pharmacist will be limited to those specific areas of inquiry about which the physician may have some doubt.

Regardless of the approach, one of the primary functions of the pharmacist is to seek to avoid adverse drug reactions or interactions. This is accomplished by his particularised knowledge of the patient's drug history and his specialised training and experience in the field. It has been suggested that there are basically six types of possible drug interactions.
They are:

(1) Straightforward potentiation, either by additive effect or synergistic action;
(2) Enzymatic inhibition, whereby one drug prevents the normal metabolism of another drug with cumulative effect and enhanced toxicity;
(3) Stimulation of enzyme systems, whereby one drug increases the rate of metabolism of another;
(4) Displacement of one drug from plasma or tissue protein by another drug which has a greater affinity for the protein binding site, causing the unbound drug to reach toxic levels;
(5) Electrolyte imbalance, such as the loss of potassium induced by thiazide diuretics, causing increased toxicity of digoxin or muscle relaxants;
(6) Interference with urinary excretion.

The purpose of listing these possibilities is to indicate the relative impossibility of monitoring drug therapy without a comprehensive background and the necessary time. These two ingredients, argue the proponents of clinical pharmacy, are the ones most often absent in standard medical practice. Indeed, properly staffed, a functional clinical pharmacy programme can contribute significantly to improved patient care.

(ii) Patient profiles

With regard to community pharmacy practice, a whole new approach to the role of the pharmacist is emerging.

A duty to communicate lies at the heart of the issue. Community pharmacists could provide useful information to either the prescribing physician or the customer patient.

Traditionally, community pharmacists have been doing it to one degree or another for quite some time, but it may be useful to examine a relatively new facet of pharmacy practice which will aid a pharmacist in developing a warning mechanism. This new concept is called the patient profile.

While it is true that pharmacists have always kept records of patients medication in one form or another, it has been only recently that some of them have been made useful for other functions than mere reference.

These more sophisticated record systems called patient profiles have become a valuable consumer service. Ordinarily, they contain patient and family records on medicines which have been dispensed to each patient, allergic responses, and adverse reactions. From these a pharmacist is in a much better position to advise both the physician and the patient on possible problems.

What is important is that by keeping records of all purchases, staying abreast of the latest reports on toxicity, and noting previous allergic and adverse reactions – the pharmacist can become one of the most important members of the health team.

(iii) Dispensing information

Another modern trend towards greater pharmacist involvement, is a fairly recent regulation promulgated by the Pharmacy Board of the State of Washington. It states: "With each new prescription dispensed after January 1, 1974, the pharmacist, in addition to labelling the prescription in accordance with present requirements, must orally explain to the patient or the patient's agent the directions for use and any additional information, in writing if necessary, to assure the proper utilisation of the medication prescribed. This was made possible by the subsequent publication of United States Pharmacopoeia XIX on July 1, 1975. The significance of this event can be grasped only by an understanding of the nature of the United States Pharmacopoeia. The answer lies in the unique legal status of the compendium. Under the Federal Food and Drug Act, the United States Pharmacopoeia and its companion volume, the National Formulary, which has now been incorporated into the former, are given the authority to establish the legal standards for the strength, quality, purity, packaging and labelling of drugs. It is unique in that standards become enforceable by law, without having to be approved by any governmental body. In addition to its national recognition, the U.S.P. is considered official in State Food, Drug and Cosmetic Acts, and most State Boards of Pharmacy require each store to have a copy on hand before it can be licensed.

Prior to the new edition, the standards established were rigid and enforceable, but now a new dimension was added, when the authors decided to add a feature called dispensing information. The intent was to provide information about each drug that a pharmacist might give a patient at his discretion without securing the prior approval of the physician.

In theory, the idea is sound when one considers our prior discussion of a duty to warn, but in practice, it may represent the opening of Pandora's box.

CONCLUSION

There can be no doubt that a change has taken place in pharmacy and this will continue both in the practical and educational fields. With leadership coming from organised pharmacy and a group of educators who have found counterparts in medicine, dentistry and nursing, together they are charting health professions education through a transitional period which historically will be seen as a change from categorical to an integrated approach to the education process in the health field. No-one denies that this change will take many years to complete and it will be arduous and at times painful.

Medicine is the link connecting the associated professions of Medicine, Dentistry, Veterinary Science and Nursing to their natural ally – the Pharmacist. Years of training and experience have prepared today's pharmacist as a drug expert. This role can be seen as one of opportunity. If the pharmacist successfully communicates his knowledge to both patient and physician, he has met the highest ideals of his profession.

What is really at stake however, is better health care for all.

CLINICAL TEACHING OF STUDENTS IN UNIVERSITY AND PRACTICE

MICHAEL YOUNG*

In the United Kingdom one of the requirements during the education of a veterinary student is that he or she should receive extra mural tuition of at least 26 weeks during the vacation of the University course, a requirement that has been one of the tenets of our veterinary education from its outset. Thus has been combined the values of University and academic education with the practical and individual communication of acquired experience and expertise that our forefathers in all trades and professions gained as apprentices.

In recent years the standard of intelligence of the student intake has markedly increased and the Universities with a ratio of 5 : 1 applicants to places, have been able to choose the cream from amongst our young people for the 335 or so places available. At the same time, expansion of knowledge has made it impossible to cram into a syllabus of 14 to 18 terms, depending on the University, all that a veterinary student should be taught or learn. It is not surprising, therefore, that an imbalance has developed and most markedly between the time and effort allotted to the pre-clinical and paraclinical subjects on the one hand and clinical teaching on the other. This is especially so as true clinicians amongst the teachers are in a minority and are sometimes looked upon as not true academics.

The result has been a highly intelligent graduate well versed in the basic sciences of the pre-clinical and paraclinical subjects and yet often unskilled in what could be called the 'art' of veterinary medicine and surgery. He may be capable of intellectual feats of reasoning but incapable of performing the simpler tasks and procedures which still occupy a large part of the working day of a veterinary surgeon, and are often undertaken in the presence of the client. Such deficiency has not gone un-noted by those who take such graduates into employment and by the more discerning animal owners.

It should be said straight away that most, if not all, such graduates learn fast and with their high intelligence and good background training soon overcome these early deficiencies, becoming in a few months, highly competent. This is little consolation to those principals who have nursed them through their early days after qualification whilst paying them high salaries, no consolation to their aggrieved clients, and probably, although no poll of opinion can or has been taken, no consolation to their animal patients.

Not all new graduates can be so classified. Some students with real vocational urge to enter veterinary practice, of strong personality and determination to succeed have made sure that the deficiencies of their veterinary education at University were made up by practical experience during their extra mural tuition, a period commonly termed 'seeing practice'. Such a student would first set aside as much as possible of his vacation time for this purpose, probably far exceeding the 26 weeks required of him by University regulations. He would choose practices not for their convenience in situation but from information provided by members of the profession with whom he was already friendly, from fellow students and from reference to the 'black book' often unofficially kept by students at his Veterinary School, listing the merits and de-merits of individual practices and their principals. My spies have related to me the relevant entries under my own name. Such feed back is both interesting and salutory.

Once the principal of the practice has agreed to his presence, our determined student will make sure he is useful, not only to the principal but to the lay staff employed. Doors will be opened, professional equipment transported, correspondence posted, examination tables cleaned and obstreperous animals held. All these and other simple actions which make life easier for the Veterinary Surgeon will be undertaken unobtrusively but efficiently and soon the latter will come to accept the presence of the student, realise his competence and come naturally to discuss with him each case, and permit him to do more and yet more simple veterinary tasks under his supervision. Once this relationship and confidence is established it is but a small step to true apprentice training of which I will say more later.

Certainly, therefore, it may be said that the solution to the problem has lain in the student's own hands, but is this satisfactory? It is not only the task, but the duty of the Universities to ensure that every graduate is competent to practice, including those whose qualities do not extend to that determination and drive to which I have referred and who may, nevertheless, become good or even great veterinarians, not all of them in practice.

Such students may and probably will, form the majority for I would suggest that those who choose a career with animals are often those who do not relate easily to other human beings, – those who choose veterinary medicine are more than usually individualistic and those who are of the highest intellect often, though not always, find difficulty in communication with those of lesser intelligence.

In the United Kingdom a ground swell of opinion from practitioners, including new graduates who recognised the deficiencies in their training and had experienced the trauma of their early days in practice, resulted in consideration of the problem by the governing body of the profession, the Royal College of Veterinary Surgeons and was highlighted at a Conference on Veterinary Education organised by that body in 1969.

At first sight the answer was simple – post graduate preregistration training, as undertaken by our medical colleagues. However, a full debate on veterinary education, and this proposal in particular, showed that such a scheme was not then viable having regard to the problems of several species and different areas of veterinary activity into which a graduate could enter. The Universities at that stage proclaimed that there was insufficient time available during the course to teach more than then undertaken, especially as the type of tuition required could only be provided on an individual basis or to small groups.

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They also indicated in no uncertain terms that they had always considered that such tuition should be part of 'seeing practice.'

The Royal College accepted this proposition, partly because the practitioners present felt that perhaps, after all, *they were* best able to train students to become good practitioners, partly because this was an area in which there seemed to be room for improvement and not least because they could think of no other positive action. We thus set out to collate the various University regulations on extra mural tuition, to comment upon them and to offer advice on the ideal regulations at which to aim.

This took time. Our recommendations were debated and pronounced upon year by year and were commented on and endorsed, as then current, by a Government Committee of Inquiry into the Veterinary Profession, known as the Swann Committee after its Chairman, Sir Michael Swann.

These recommendations now read as follows: The extra-mural experience which a veterinary student should obtain may conveniently be described as follows

(a) farm experience;
(b) extra-mural tuition in clinical practice and other veterinary work, as an integral part of the veterinary degree course.

Farm Experience

This should be undertaken by the student prior to the clinical years of the veterinary degree course. The aim should be to ensure that the student acquires a varied experience of the handling and management of different species of animals and of animal production on a number of farms or in different livestock enterprises, such as a modern dairy herd, an intensive breeding and fattening pig unit, and a flock at lambing time. It is suggested that the minimum requirement for such experience should be a period of six weeks to be undertaken subject to the general guidance of the Veterinary School.

Extra-mural tuition in clincal practice and other veterinary work

The vacations during the later years of the veterinary degree course should be so utilised as to enable students to obtain extra-mural tuition in clinical practice and other veterinary work as an integral part of the course, which will complement the teaching given at the University. In view of the complementary nature of this extra-mural tuition it is considered essential that each Veterinary School should accept responsibility for its supervision. If this is not done, there can be no certainty that this period of extra-mural tuition will meet its objectives.

It is recommended that at least *26 weeks'* extra-mural tuition should be obtained by each veterinary student during the later years of the course. Special emphasis should be laid on the need for students to spend the major part of this period with veterinarians in general practice in order that they may obtain –

(a) additional clinical tuition complementary to the training they have been given in this field at the Veterinary Schools; and
(b) knowledge of practice organisation, practice and farming economics and relationships with clients.

The general aims and objectives should be to –

(i) see as wide a range as possible of disease conditions and their treatment in all species of domestic animals; and
(ii) gain practical experience in as many aspects of veterinary work as possible, including the handling of animals and proficiency in routine techniques.

It is accepted that a Veterinary School will normally expect most of the 26 weeks of extra-mural tuition to be utilised in clinical practice. The School may take the view that some part of this period should be devoted to complementing other aspects of the teaching on the course. The requirements will vary from School to School.

The degree of involvement of a Veterinary School in the organisation of extra-mural tuition for its students is a matter for decision by the University and the Veterinary School, but it is recommended that –

a) each Veterinary School should draw up and maintain a panel of extra-mural teachers;
b) regular contact should be maintained between a Veterinary School and its extra-mural teachers, for instance by encouraging the latter to visit the School and by inviting their comments upon students receiving extra-mural tuition from them;
c) information, guidance and advice should be offered to extra-mural teachers on the procedures which students might be expected to carry out and on general arrangements for extra-mural tuition, and extra-mural teachers should be provided with a copy of the instructions given by the School to the student.

It is recommended that extra-mural experience and tuition for veterinary students should be a specific and formal requirement within the curriculum, and that each University should (if this is not already the case) be asked to consider writing into the regulations for its veterinary degree course a provision for a minimum requirement of six weeks' farm experience to be undertaken prior to the clinical years of the course, and a provision for 26 weeks' extra-mural tuition in clinical practice and other veterinary work to be undertaken as an integral part of the course in the vacations of the later years.

Although these last recommendations have only just been agreed and issued it would be fair to say that, over the last six years, the Schools have already gradually commenced their implementation, albeit with some protest. There is, however, still one major weakness and that is the lack of any requirement to assess the experience gained by the student during extra-mural tuition and to ensure that the latter *does* complement the tuition provided by the School to fill in those gaps in knowledge and experience which are bound to have occurred. It is still quite possible for the student to comply with the regulations, undergo 26 weeks' of extra-mural training and never himself carry out a proper examination of an animal or lift a scalpel.

It is also possible that his practical experience at University will be little greater but I will come to this later.

One of our Veterinary Schools, Bristol, has during all its existence kept close control of the extra-mural tuition provided to its students and has also held regular week-end meetings, at the school, of practitioners, it, the University, has appointed as such tutors. Whilst such meetings enable the practitioners to see the School and its facilities and learn of the curriculum and from

the academic staff it is the "coffee time" discussions which prove the most salutory. One comes away on the Sunday evening realising how little one has accepted ones responsibilities to teach and train the next generation of the Profession and what a privilege it is to be allowed and expected to do so.

For myself there has been the added stimulus of personal involvement and the pleasure of guiding and training my own son during those periods in his vacation when he was not undergoing his statutory 26 weeks, all of which he served in practices other than my own. I also recently took a student who had twice failed his final examination in veterinary medicine and who I was prevailed upon by his University to take for six weeks training. Perhaps this was because I was one of his examiners and thus responsible for his period in limbo.

My approach to extra-mural tuition in my own practice is as follows – A student who has has no previous experience in a small practice and has yet to commence or has only just commenced his first clinical year, starts by being taught to do expertly those tasks commonly performed by lay staff. These will include dressings, bandaging, restraint, scrubbing up and laying out of instruments, simple boichemical tests, skin scrapings, – developing X-rays – the list is endless.

I firmly believe that a veterinarian should be able to perform with ease and expertise all these tasks he may require of his lay staff. The lay staff are encouraged to use and to train the student and particularly to pass on their own 'tricks of the trade.' Phone duty is also good training, especially at night, and the task of returning animals to their owners, not only enables the student to acquire the habit of easy and essential communication but also of collecting the fee.

The next stage is for the student to make a full examination daily of all in-patients and properly record conditions and symptoms observed. Together with this goes the administration of any medicaments prescribed and a developing proficiency in subcutaneous, intra muscular and intra venous injections. Unwanted animals left for destruction provide excellent material for the practice of intra venous injections, proficiency in which is essential before the student can be permitted to induce anaesthesia by this route. The knack of intra tracheal intubation can also be acquired at the same time.

The student must become obsolutely proficient in all these basic and routine tasks so that he can proceed with confidence to the next stage of his training.

Now comes the early ecquisition of surgical skills. The routine of a scrub up and preparation for sterile surgery will have been taught by the nursing staff but now our real task begins. The main experience the student needs in surgery is the handling of living tissue. Assisting during major operations is valuable, very valuable, but true understanding of what is involved only begins when the student has himself made an incision, found and clamped the bleeding vessels and sutured the tissues which have been traumatically or surgically parted. He cannot de Bride and repair too many wounds, he cannot excise too many small lesions. These are what an average practice can usually provide in abundance and what cannot be provided in sufficient quantity at the veterinary schools.

The value of such early experience in operative surgery can be enhanced by the student, himself, being required to discharge the patient and instruct the owner in post operative care. Later, the removal of the sutures in the presence of the owner provides the opportunity to listen to comment, adverse or complimentary, on the result. No one who has had to dig for sutures embedded in a complaining dog in the presence of an even more complaining owner will ever again tie them too tightly. The breakdown of the wound or presence of pus, and with the need for suitable explanation, is also a salutory lesson.

Once the student has learnt to handle tissue and instruments with confidence and care the stage is set for experience in the more precise operations and proceedures. It is not, or should not be, the task of the practitioner to teach orthopaedics, intra ocular surgery, or other advanced techniques, for these are likely to be better demonstrated and taught at University. It is the simple techniques, the spay, the castration, the removal of dew claws and the dental extraction which the student should be permitted and encouraged to perform, for although he will receive theoretical tuition and may gain some practical experience of such proceedures at his School, there will never be sufficient case material for him to gain proficiency. Furthermore, it is these proceedures which are likely to form the bulk of his surgical case load when first he goes into practice and on which he will base his confidence and establish his early reputation.

The student should also be allowed to examine animals and question their owners at first presentation, and himself make and, establish a diagnosis and suggest treatment. For him to do all this after the veterinarian has himself made his examination and pronounced thereon, does not begin to approach the value of allowing the student to start from scratch.

In these ways, and by discussion of cases, particularly over lunch consumed by the pint, will the experienced practitioner pass on his acquired knowledge and experience. The less experienced graduate employed in the practice will also have a part to play for he will point out the pit falls which he has too recently himself experienced and will better appreciate the problems facing the student about to embark on practical application after a surfait of theory.

Our Schools have also had some change of heart and emphasis. The pendulum has begun to swing. The Swann Committee in its report underlined the need for adequate case material and case experience during the clinical years and recommended that each School acquire a practice for teaching purposes, a recommendation that has already been taken up by two of the six schools, whilst a third has for a long time been in that happy position.

It seems now to be accepted that clinical tuition can only be satisfactory if adequate case material for teaching purposes is available. This can and is obtained in several ways.

1) A University farm practice.
2) A University out-patients clinic for small animals.
3) Staffing of a charity clinic for small animals.
4) Purchase of the veterinary work on certain specified farms.
5) Purchase of specific cases for demonstration.
6) Referral.

In assessing the case material requirement of a school for the teaching of students the need for the continued

education of clinical staff, for research and for the development of new techniques and advances in knowledge should not be forgotten. There must be a balance and it must be just right.

A well established University farm practice with clients used to, and accepting, the involvement of students is of particular value not only for the routine work generated but for the study of herd/flock treatment, disease prevention and both practice and farm economics. It is hopefully likely to provide adequate material for the teaching of pregnancy diagnosis. Perhaps most important it enables clinical staff to keep up to date with "on farm" veterinary problems and to embark on projects of trials and research.

One of the dangers inherent in the possession by the University of a farm practice is the division which may develop between the staff of the practice and their colleagues in the surgery and medicine departments. It is, in my view, essential that all the clinical teachers should be involved in the general work of the practice, even if this leads to some lack of continuity in the treatment of individual cases.

The purchase of the veterinary work on certain specified farms can only be considered second best to this.

The purchase of specific cases for demonstration can, of course, complement the benefits of a farm animal practice without truly offering an alternative.

A small animal clinic or charity clinic provides first opinion case material, together with its due proportion of routine and more specialised surgery: However, the surgical work is unlikely to provide anything like sufficient material for each student to gain a satisfactory degree of practical experience. It nevertheless does provide sufficient and varied work for the new graduate house surgeons to make good progress under the overall guidance of the senior surgeons on the staff and it is from this former group that the future consultants and professors of surgery are likely to emerge. The clinic also provides fair experience – client management and psychology so necessary for small animal practice. I say "fair" because the clinic will attract a type of client more amenable to student participation with the attendant risks to the pet than the average pet owner seen in practice.

It is right, and in my view, essential that the clinical departments of Veterinary Schools should also be centres of excellence to which veterinarians in practice refer cases for diagnosis, treatment and surgery. The clinical teachers should themselves be in the fore front of knowledge, should be acknowledged as consultants, and should be paid accordingly. Referred cases are an especial service of clinical material, of teaching value to the advanced undergraduate and the post graduate student. They are rarely of value for the teaching of basic principles or for the practice of surgical skills and, therefore, should be considered as additional and complementary to routine practice or clinic cases. There is the ever present danger that the clinical teacher regularly consulted by practitioner colleagues will cease to remember his first and foremost duty, to teach. It should be the task of his supervisors to provide a reminder when necessary.

Even with all these services, the flow of clinical material through the schools is unlikely to be sufficient in quantity and variety to provide satisfactory comprehensive clinical experience. Thus extra-mural tuition under proper overall direction is still necessary and perhaps essential. Each student and his or her needs should be considered separately.

Weakness with regard to species can be overcome by direction to a practice specialising in that animal. The farmers son may need further time in suburban practice, the town boy sent off to green fields and the problems of mastitis. Gaps in experience need to be identified and then, where possible, filled.

I have recently, as part of a small group, visited and inspected the clinical departments of all the veterinary schools in the United Kingdom, on behalf of my Royal College and during the tour certain facts came to our attention. Although both the final and penultimate years are reckoned to be the clinical years, effectively nearly all such teaching is confined to the last year before graduation. This is because para-clinical subjects intrude into the penultimate year together with examinations in these subjects. Human nature being what it is, students only concentrate on the hurdle immediately to be faced with other subjects coming a very poor second. Prior to this penultimate year the student, on average, will have received little or no clinical tuition, even in making and recording a proper examination of a patient. With notable exceptions the pre and para-clinical subjects are taught in isolation with little reference to the ultimate object of the exercise, a competent veterinary surgeon. (I, am, of course, fortunate that none of my colleagues who are teachers in these subjects are present for I am sure they would argue their case and cause with vehemence.)

In the final year, teaching is undertaken with enthusiasm and method but time is now very short and for the final term all eyes are set on the examinations to be passed rather than the knowledge to be gained. In most of our schools teaching is conducted in small groups, usually of six students and lectures are kept to a minimum. In some subjects specialists in their field teach their subject extremely well and most students are competent in radiology, opthalmology, anaesthesia and the theory of orthopaedic surgery, on graduation. Regretably the same cannot be said at present of the teaching of surgical skills, a subject which requires plenty of student time, staff time and case material, all of which is in short supply. Indeed we felt that time was the commodity in shortest supply and made recommendations hopefully to point the way to a solution to the problem. We felt that formel lectures had little place in the final year and that the essential lecture course should be completed before its commencement. We also felt that the current limitation of University teaching to the three normal University terms during final year should be superseded by a full year of clinical studies of say 48–50 weeks. During this time students individually or in groups could be seconded to specific practices or other Universities to complement their training. The University premises and its facilities, its clinic and veterinary practice and also the cases referred to its specialised clinical departments would then be utilised to the full and students would be involved for the whole year and, on rota, for 24 hours a day. Thus could commence their transition from undergraduate to practitioner.

I, personally, do not believe that even this change will swing the pendulum far enough for I am firmly of the opinion that the veterinary student should be in contact with clinical material from the outset of the course and that all the subjects taught, by whatever de-

partment of the University, should show relevance to the ultimate objective of a veterinary qualification and ability to practice veterinary surgery and medicine. This opinion is, I should add, mostly shared by the students but not by the University teachers. They have set their corporate face steadfastly against any such proposal. They are, however, beginning the accept the necessity for some basic clinical tuition before the student begins 'seeing practice' which means before the completion of the third year of the course. By then the student will in future, have been at least taught to make a proper clinical examination and to record his findings. From then onwards he will be encouraged and, indeed, required to keep records of his clinical experiences both within and without the University.

Consideration of the teaching of undergraduates and recommendations for improvement of the teaching must take into account the needs of the teachers. If, as I have suggested, the student is to receive more practical clinical tuition, much on an individual or small group basis, the staff student ratio must be reduced from the current level of 1 : 5 so that individual teachers have adequate opportunity and *time* for research, for meetings and congresses, for post graduate teaching and for gaining further clinical experience. It is, I believe, of paramount importance that University clinical staff should teach from their own practical up to date knowledge and experience and this is one of the main reasons why the veterinary school should receive a continual flow of the widest possible range of clinical material. It is also important that the clinical teachers have regular contact with veterinary practitioners and veterinary practices so that they remain aware of the changing problems their students will have on leaving University.

I have now put before you my thoughts and observations on clinical veterinary education, as I see it today, in my own limited experience, in my own country. I believe the position may be similar in other places. I believe most strongly that we all, both practitioners and teachers, have a duty to the next generations of veterinarians. With them lies our future and the future of our profession. We all have a part to play, a part which will be the most effective and beneficial and close ties are established and maintained between those who teach and those who practice.

BOOK REVIEW

BOEKRESENSIE

GUIDE TO HYGIENE AND SANITATION IN AVIATION

J. BAILEY, 2ND ED.

World Health Organisation, Geneva 1977
pp. 170, Figs. 37, Numerous Tables. Publ. Price Sw.fr.28

The present author has revised the 1st edition which appeard in 1960. The publication is intended for all those who are directly or indirectly concerned with ensuring the health of those who fly. With the steady increase in air traffic, this is a subject which will concern an increasing number of people as time goes on.

Detailed information pertaining to the health and hygiene aspects of aviation is presented under the following broad headings: food, water, liquid and toilet waste disposal, aircraft interior cleaning, cargo and vector control. A series of aanexes is included. These deal with such subjects as the characteristics of common food borne diseases, investigating food poisoning on board the aircraft, guide-lines for sanitary inspection of airports, drinking water quality and disinfection of water vehicle tanks, etc.

Although intended specifically for aviation circumstances, there is much in this soft cover booklet which is applicable to other situations in which food and water hygiene and vector control measures are applicable. Examples are the comprehensive tabulated summary of the important characteristics of food borne diseases (Annex 1) and Annex 5 which deals with the quality of drinking water.

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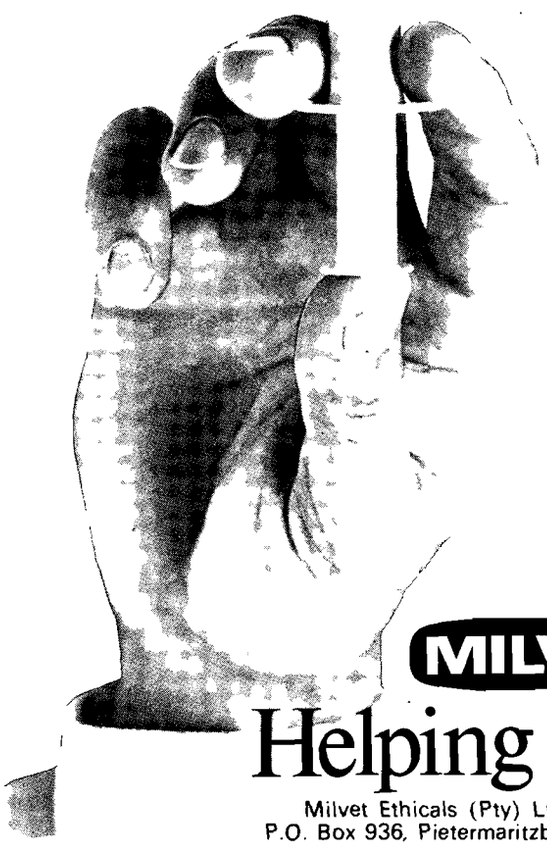

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BESMETLIKE OORSAKE VAN PERINATALE VREKTES BY HERKOUERS*

J.A.W. COETZER en A.P. SCHUTTE

ABSTRACT: Coetzer J.A.W.; Schutte A.P. **Infectious causes of perinatal mortalities in ruminants.** *Journal of the South African Veterinary Association.* (1978) **49** No. 2 89–98 (Afr. en.) Vet. Res. Inst. Onderstepoort Rep. South Africa.

The advantages and disadvantages of the different diagnostic techniques e.g. pathological and microbiological studies, immunoglobulin and specific antibody determinations and fluorescent antibody studies in relation to these mortalities are discussed. The most important pathological lesions in the placentas and foetuses are described.

INLEIDING

Die eise wat tans aan die veeboer en veral aan die suiwelprodusent gestel word, is enorm. Daar behoort geen twyfel te bestaan nie dat slegs diegene onder hulle wat daarin kan slaag om die voortplantingsdoeltreffendheid van die herkouer optimaal ingestel te kry, dreigende ekonomiese aanslae sal kan trotseer.

Optimale produksie is alleen moontlik indien die faktore wat onvrugbaarheid, perinatale vrektes en neonatale verliese in die hand werk, streng beheer word. Ongelukkig is daar sekere besmetlike oorsake van onvrugbaarheid wat wesentlik diagnostiese probleme inhou en waaroor ook nog heelwat verwarring bestaan. Dit is egter gebiedend noodsaaklik dat die etiologie van besmetlike onrugbaarheid met die minste versuim uitgewys moet word. Hiersonder is dit baie moeilik om beheermaatreëls te formuleer of om die eienaar van die korrekte advies te voorsien.

Dit word geredelik erken dat dit nie altyd moontlik is om 'n diagnose te maak nie – veral nie as die diagnose net op die afsondering van die veroorsakende organisme berus nie. Maar deur van meet af 'n meer omvattende ondersoek in te stel wat benewens 'n gedetailleerde post mortem-ondersoek van die fetus en vrugvliese en standaard mikrobiologiese prosedures ook histopatologie, kwantitatiewe immunoglobulien en spesifieke teenliggaampiebepalings insluit, kan veel meer vermag word. Enkele gedagtes in hierdie verband word weergegee.

Makroskopiese en histologiese ondersoeke[22 72]

Die gedetailleerde ondersoek van die fetus en vrugvliese kan dikwels inligting of leidrade verskaf wat die ondersoeker instaat sal stel om sekere mikrobiologiese prosedures te volg en sodoende die diagnose te vergemaklik.

Alhoewel dit soms moeilik kan wees om die weefselveranderinge soos met histopatologiese ondersoeke waargeneem, aan 'n spesifieke organisme te koppel, is daar tog dikwels letsels waar te neem wat as redelik spesifiek vir 'n besondere siektetoestand beskou kan word. Meer dikwels egter gebeur dit dat sekere van die makro- en mikroskopiese afwykings, alhoewel nie patognomonies vir 'n spesifieke organisme, wel as kenmerkend van 'n groep van organismes beskou kan word. Die histopatologiese veranderinge in verskillende organe beskou kan word. Die histopatologiese veranderinge in verskillende organe is gewoonlik niespesifiek en slegs 'n aanduiding of daar wel 'n besmetting plaasgevind het. Vir die betekenisvolle vergelyk moet word met die mikrobiologiese en serologiese resultate. Deur kennis hiervan te neem en spesifieke kleurmetodes en histochemiese tegnieke te gebruik, kan die veroorsakende organisme makliker geïdentifiseer word.

Dit is van die uiterste belang om ag te slaan op die tydsverloop tussen fetale afsterwing tot en met die ondersoek. Outoliese, veral gedurende die binnebaarmoederlike periode tree geweldig vinnig in en ook gouer in sekere organe as by ander. Dit gebeur dikwels dat outolitiese veranderings as letsels van besmetlike oorsprong gedokumenteer word. Hier word veral verwys na onderhuidse edeem, die aansameling van bloedkleurige vloeistowwe in liggaamsholtes, vervloeiing van die nierkorteks, die egalige rooibruin verkleuring van weefsels, versagting en gevlekte voorkoms van die lewer en die troebel inhoud van die abomasum. Dit is eweneens noodsaaklik om daarop te wys dat outolitiese veranderinge histologiese detail kan verbloem.

Die histopatologiese ondersoek moet nie net tot een of twee organe beperk word nie, maar 'n wye reeks van organe moet deurgaans ondersoek word. Dit dien hier ook vermeld te word dat indien die verwerping van die konseptus met die koorsreaksie in die moederdier saamval, die histologiese ondersoek dikwels negatief is.

Die normale makroskopiese voorkoms van sekere organe, die histologie asook die inflammatoriese reaksie van die fetus verskil tot 'n mate van die in die volwasse dier. Vir die sinvolle interpretering van die ondersoeker se bevindings is dit dus belangrik dat hy vertroud sal wees met die verskille.

Omrede die weefselveranderinge in die fetus dikwels niespesifieke reaksies is, is dit noodsaaklik dat vrugvliese saam met die fetus vir ondersoek aangebied moet word. Dit wil soms voorkom of die veearts nie altyd besef hoe belangrik vrugvliese vir die patologiese ondersoek en afsondering van patogene organismes is nie. Waar dit wel aangebied word is dit wenslik dat die plasenta deeglik ondersoek moet word omdat letsels nie altyd diffuus verspreid voorkom nie. Daar moet gelet word na die grootte en aantal kotiledons, die dikte van die interkotiledonweefsel, kleur en samestelling van die eksudaat, asook na die graad van outoliese wat teenwoordig mag wees.

Mikrobiologiese en serologiese ondersoeke

Die persoon wat ondersoeke na fetale verliese by die herkouer wil onderneem, moet goed onderlê wees in die standaard bakteriologiese, virologiese, protosoïese en mikologiese prosedures.

Finaliteit oor die etiologie van fetale afsterwing word slegs verkry na diepgaande ondersoeke en met die in-

*Referaat gelewer tydens die Wetenskaplike Kongres van die SAVV, Grahamstad, 1977

dentifikasie van die veroorsakende organisme. Ondervinding het geleer dat daar dikwels meer as een patogene organisme vanuit dieselfde fetus afgesonder kan word. Eweso dra dit veel meer gewig indien die mikrobiologiese resultate met die patologie, soos waargeneem, in verband gebring kan word. Daar moet ook rekening gehou word met die feit dat die geskiedenis en siektebeeld wat van 'n kudde ingewin word dikwels onvoldoende is en selfs misleidend mag wees. Daarom is die enigste uitweg 'n deeglike en wye mikrobiologiese ondersoek.

Dikwels gebeur dit dat die veroorsakende organisme nie meer teenwoordig is wanneer die ondersoeke uitgevoer word, of dat die organisme nie die baarmoederskans deurgedring het en nie die fetus bereik het nie. In sulke omstandighede is dit wenslik om bykomstige materiaal van ander diere uit dieselfde kudde te bekom om die betrokke patogene organisme af te sonder. Sekere prenatale virusbesmettings (bloutongvirus, Akabane virus, mukosasiekte virus, natuurlike en geattenueerde Wesselsbronvirus en geattenueerde Slenkdalkoorsvirus) in herkouers affekteer die normale ontwikkeling van die fetale brein. Die teratologiese afwykings van die brein is egter 'n letsel wat oor 'n geruime tyd ontwikkel en in meeste van die gevalle is dit nie meer moontlik om die virus van die brein of ander weefsels af te sonder nie wanneer die fetus geaborteer of dood of lewendig gebore word. Indien die fetus immuunkompetent was tydens die besmetting kan spesifieke teenliggaampiebepalings op die prekolostrum serum in die gevalle van groot waarde wees. Die magdom van serologiese toetse wat vir die identifikasie van teenliggame aangewend kan word en die siektetoestande waarvoor ondersoek kan word, is welbekend. Die waarde van sommige van die toetse is egter beperk.

Die ondersoek van enkel serummonsters van die moederdier het weinig diagnostiese waarde en kan tot verwarring of selfs foutiewe diagnoses aanleiding gee. Gepaarde sera (versamel kort na aborsie en weer 3–4 weke later) hou veel meer waarde is. 'n Styging van die teenliggaamtiter kan dikwels as diagnosties beskou word.

Immunofluoresensie (FA) as diagnostiese hulpmiddel by ondersoeke na perinatale verliese het gedurende die afgelope dekade van besondere waarde geblyk te wees. Die spesifisiteit van hierdie metode is vergelykbaar met die konvensionele serologiese metode terwyl die sensitiwiteit van hierdie metode gewoonlik baie beter is. Immunofluoresensie bied die geleentheid om ondersoeke gou en akkuraat af te handel. Ondersoeke na *Leptospira* en *Mycoplasma* is sprekende voorbeelde. Die afsondering en identifikasie van hierdie organismes kan enkele dae in beslag neem, terwyl met die FA metode die diagnose binne enkele minute afgehandel kan word.

Die kwantitatiewe bepaling van fetale immunoglobuliene [7]
31 32 57 59 61 62 63 70 80 83

Dit is maar eers redelik onlangs dat die waarde van hierdie analises vir die diagnose van besmetlike oorsake van perinatale vrektes besef is en toetse in hierdie verband beslag gekry het.

Data hieroor is nog beperk en die gedokumenteerde gegewens is ook dikwels teenstrydig – veral met verwysing na die stadium van fetale ontogenie wat aan immuunkompetensie gekoppel kan word. Om die immunologiese respons van die fetus te verstaan, is dit belangrik om 'n goeie begrip te hê van wanneer die fetus immuunkompetensie of immuunvolwassenheid bereik. Drie faktore is hier veral belangrik nl. die spesie betrokke, die lengte van dragtigheid en die tipe antigeen. In die herkouer is dit bekend dat die fetus instaat is om immunologies te reageer teen verskillende antigene op verskillende stadiums van dragtigheid.

By die bees en skaap is met die verloop van tyd aanvaar dat geen transplasentale uitruiling van maternale immunoglobuliene (Ig) plaasvind nie en dikwels is dit dan ook gekonstateer dat die kalf en die lam in 'n agammaglobulienemiese toestand gebore word. Uit resente werk blyk dit nie heeltemal korrek te wees nie, aangesien uitsypeling van Ig vanaf die moederdier na die fetus wel kan plaasvind indien daar patologiese veranderinge in die plasenta is. Maar van groter belang is die feit dat die fetus ook Ig kan vervaardig.

Dit skyn asof IgM en IgG die belangrikste indikators van infeksie in die beesfetus is. IgM is die eerste Ig wat deur die fetus vervaardig word en verskyn tussen 125 en 135 dae van dragtigheid in die serum van die beesfetus. Hierbenewens kan IgM bevattende selle reeds op 59 dae van dragtigheid en IgG bevattende selle teen 145 dae van dragtigheid in die milt en limfkliere van die beesfetus gedemonstreer word.

Geen fetus is agammaglobulienemies nie, maar het 'n lae vlak van Ig al was die fetus nog nie spesifiek antigenies gestimuleer nie. Indien die fetus egter antigenies gestimuleer word is daar 'n styging van die Ig. Die volwasse beesfetus se normale waardes vir IgG en IgM word as 0,16 mg/ml en 0,2 mg/ml respektiewelik aangegee. Waardes van meer as 0,38 mg/ml kan 'n indikasie wees dat daar wel *in utero* besmetting van die fetus plaasgevind het.

Gedurende die afgelope jaar is hierdie tipe analises op 160 geaboteerde fetusse gedoen. Van die gegewens verkry is dit duidelik dat 33% van die 51 fetusse waaraan geen besmetlike oorsaak gekoppel kon word nie wel 'n styging in Ig gehad het.

Hierdie ondersoeke blyk veral van waarde te wees in gevalle waar virusse verantwoordelik was vir die abnormale ontwikkeling van die fetale brein en waar die veroorsakende organisme nie meer afgesonder kan word nie. Hierbenewens kan die verhoogde Ig waardes in die prekolostrumserum of breinvloeistof van hidranenkefalie gevalle dikwels as 'n indikator van besmetting gebruik word en is 'n geruime tyd voordat spesifieke teenliggame geproduseer word teenwoordig.

ERKENDE BESMETLIKE OORSAKE VAN PERINATALE AFSTERWING BY HERKOUERS

1. Virusaandoenings

Die opsigtelike fetale abnormaliteite in die mens wat aan virusblootstelling toegeskrywe kan word is wel bekend. Hierteenoor is die uitwerking van virusse in beeste en skape en sogenaamde onopsigtelike abnormaliteite van die pasgebore kalf en lam eers onlangs met mekaar in verband gebring.

Die vermoë van die virus om die konseptus nadelig te beïnvloed word beheer deur die inherente weerstand of vatbaarheid van die moederdier, die virulensie van die virus en die ontwikkelingsfase van die embrio of fetus.

Die graad van beskadiging wat die virus kan veroorsaak hou verbamd met weefseltropisme en virulensie. Die virus kan fetale afsterwing met verwerping teweegbring of retensie met verstening as gevolg hê. Sekere virusse (bloutong-, Akabane-, Wesselsbron- en mukosasiektevirus) met 'n lae virulensie en spesifieke tropisme vir ontwikkelende breinweefsel het dikwels porenkefalie, hidranenkefalie of serebellare hipoplasie tot gevolg indien die skaap- of beesfetus gedurende die kritiese periode (100–130 dae in die bees en 45–55 dae in die skaap) van breinvorming besmet raak. Hierteenoor kan dit gebeur dat dieselfde virus, wat die dier gedurende 'n meer gevorderde stadium van fetale ontwikkeling binnedring, slegs 'n geringe selreaksie uitlok. Die virusse kan net kort na besmetting gedurende die sensitiewe periode uit breinweefsel afgesonder word. Hierna is pogings tot virusafsondering meesal onsuksesvol.

Hierdie aspekte moet deeglik in oorweging geneem word indien virusblootstelling met perinatale verliese gekoppel word. Omrede virusafsondering nie altyd moontlik sal wees nie kan indirekte bewyse soos die teenwoordigheid van virusteenliggaampies in fetale of prekolostrumserum dalk van nut wees om 'n diagnose te maak.

Veertien verskillende virusse is al uit fetale weefsel en plasentas afgesonder. Virusse wat tot die adenogroep (bees adenovirus), die picornagroep (bees enterovirus), die paramyxogroep (parafinfluenza-3), die orbivirusgroep (bloutongvirus) en die togavirusgroep (Slenkdalkoorsvirus) tuishoort kan met vrugresorpsie, fetale verwerping of neonatale vrektes in verband gebring word. Die patogenese van hierdie virusaandoenings is, met die uitsondering van enkele, onvoldoende beskrywe. Die meer belangrike van hierdie kan as volg saamgevat word.

(a) Slenkdalkoorsvirus (SDK) [13 14 15 16 20 21 64 68 69 73 74 79 84]

Hierdie virus gee aanleiding tot 'n siektetoestand by veral herkouers en is al vir ruim 25 jaar in die Republiek bekend. Gedurende die periode 1974–1976, na buitengewone goeie reëns en gunstige weersomstandighede vir die uitbreiding van die muskietpopulasie, het die virus weer wydverspreid voorgekom en is ernstige verliese hieraan toegeskryf.

Groot verliese het veral by skape voorgekom waar soms soveel as 90% mortaliteit in pasgebore lammers gedokumenteer is. Hierbenewens het tot 40% van dragtige ooie geaborteer en het sommige ooie ook as gevolg van post partum-steurnisse en ander komplikasies gevrek.

Ooie aborteer gedurende of kort na die koorsfase van die siekte. Waar die aborsies eers na die koorsfase voorkom, kan 'n diagnose dikwels op grond van die tipiese weefselveranderinge en meer spesifiek met histopatologiese veranderinge in die fetale lewer gemaak word. Die pannekrose met die kenmerkende verspreide foki van koagulatiewe nekrose wat ooreenstem met die in die pasgebore lam kan soms ook in gevalle waar gevorderde outoliese reeds ingetree het waargeneem word. Waar die aborsies met die koorsfase saamval is dit nie altyd moontlik om die virus uit die fetus af te sonder nie en is die kenmerkende lewerletsels soos beskryf meesal afwesig. Die karakteristieke lewerveranderinge het nooit gepaard gegaan met enige inflammatoriese reaksie in die brein nie.

Slenkdalkoors kan eweneens as 'n siektetoestand by beeste beskou word, maar hier word baie selde hoë mortaliteite of kliniese waarneembare simptome aan gekoppel. Enkele aborsiestorms is al met die siekte geassosieer. Volgens Swanepoel was daar gedurende 1969 in Rhodesië buitengewone hoë mortaliteite en aborsies by beeste as gevolg van SDK. Volgens hom is daar 'n goeie korrelasie tussen die histopatologiese diagnose van SDK en serologiese toetse in beesfetusse.

Gedurende die afgelope 3 jaar is die muisbrein geattenueerde SDK entstof wydverspreid gebruik. Nieteenstaande herhaalde waarskuwings het boere nogtans die entstof in dragtige ooie gebruik in 'n laaste poging om vrektes te beperk. 'n Variërende persentasie aborsies het onder sulke ooie voorgekom. Die fetusse het nie die kenmerkende lewerletsels getoon nie, maar van die ooie wat gedurende die laaste week van dragtigheid geënt was het lammers gehad met 'n uitgesproke limfositêre meningo-enkelfalitis. Veral van belang was die *hydrops amnii,* artrogripose en die hidranenkefalie wat by fetusse voorgekom het. Hierdie afwykings is onlangs eksperimenteel bevestig.

(b) Wesselsbronvirus (WBS) [16 17 74 85]

Weiss en medewerkers[85] het in 1956 hierdie virus vir die eerste keer uit skape in die Wesselsbrondistrik afgesonder. Tydens hierdie uitbreek het baie van die swaardragtige ooie geaborteer. Hierdie werkers het ook daarin geslaag om aborsies met die virus in skape te verwek. Enkele volwasse doodgebore lammers is ook aangeteken.

Uit resente werk blyk dit dat die stadium van dragtigheid by die ooi belangrik is in soverre dit die persentasie van aborsies en die voorkoms van breinletsels betref. Slegs enkele van die ooie wat op 42–84 dae dragtigheid met die virus besmet is, het geaborteer of fetale resorpsie ondergaan. Van die ooie het die kenmerkende *hydrops amnii* ontwikkel en by die fetusse van hierdie ooie is hidranenkefalie en artrogripose waargeneem.

Beeste is ook vatbaar vir WBS virus, maar dit is nog nie voorheen gerapporteer tot watter mate die virus aan aborsies gekoppel kan word nie. Alhoewel WBS virus wydverspreid voorkom in Rhodesië was die siekte nog nie geassosieer met aborsies in beeste nie. Die bevindings is onlangs eksperimenteel bevestig deur beeste tussen 101 en 147 dae van dragtigheid te besmet. Uit die resultate blyk dit dat WBS nie 'n belangrike oorsaak van aborsie in beeste is indien besmetting tydens díe stadium plaasvind nie maar dat die virus wel teratologiese afwykings in die brein van die fetus kan veroorsaak.

(c) Bloutong (BT) [2 18 26 49 50 55 56 58 60 86]

Dit is reeds vir 'n geruime tyd bekend dat díe virus porenkefalie en hidranenkefalie in die fetale brein kan veroorsaak indien ooie op 'n sekere stadium van dragtigheid met die natuurlike of gemodifiseerde entstofvirus besmet sou raak. Bewyse is ook reeds gelewer dat die natuurlike virus in die beesfetus tot hidranenkefalie aanleiding kan gee indien die koei gedurende die kritiese periode van 100–130 dae dragtigheid besmet raak.

Hidranenkefalie en porenkefalie word slegs by uitsondering in pasgebore lammers hier ter plaatse aange-

teken en dit nieteenstaande die feit dat bloutongentstof dikwels in dragtige ooie gebruik word. Die rede hiervoor mag wees dat meeste ooie reeds op 'n jong ouderdom aan die virus blootgestel word hetsy deur enting of natuurlike besmetting en gevolglik 'n basiese immuniteit besit.

(d) Akabane virus[5 33 48 52]

Akabane virus kan ook met artrogripose en hidranenkefalie by pasgebore kalwers in verband gebring word. Selfs aborsies en doodgebore kalwers is al aan die virus toegeskrywe.

Die virus is al geassosieer met hierdie sindroom in Japan, Australië en Israel en sedertdien is daar ook serologiese aanduidings dat die virus wydverspreid in Suid-Afrika voorkom veral in kuddes waar hierdie sindroom waargeneem is.

Dieselfde abnormaliteite is ook reeds eksperimenteel verwek in skaapfetusse.

(e) BVD/Mukosasiektevirus (BVD/MS)[7 9 10 11 24 25 35 36 37 38 39 67]

Volgens serologiese ondersoeke kom hierdie virus ook wydverspreid in die skaap- sowel as die beespopulasie in Suid-Afrika voor. Die virus is reeds met perinatale verliese by beeste maar nog nie by skape in verband gebring nie. Alhoewel nie 'n belangrike oorsaak van perinatale verliese nie, is BVD/MS virus wel al geassosieer met sporadiese aborsies in beeste.

Behalwe vir fetale verwerping kan die virus fetale verstening veroorsaak of aanleiding gee tot doodgebore kalwers. Die vatbaarheid van die fetus vir die virus verminder namate fetale ontwikkeling vorder. Hipoplasie van die serebellum is die mees kenmerkende letsel wat deur die virus veroorsaak word. Retinale degenerasie, katarakte, hipoplasie en neuritis van die oogsenuwee gaan dikwels hiermee gepaard. Omdat dit 'n virus is wat ook slymvliese aantas word bloedings en ulserasies 1–3 mm in deursnee dikwels in die mondholte, esofagus, larinks, tragea, abomasum en konjunktiva gevind.

(f) Aansteeklike bees rinotrageïtis (IBR/IPV)[12 24 35 40 41 42 46 51]

Die invloed van hierdie virus op die asemhaling- en geslagstelsel van die bees is welbekend. Die virus kan behalwe vir fetale verwerping ook aanleiding gee tot fetale verstening en enkefalitis in kalwers.

Besmetting kom die kudde heelwaarskynlik deur draergevalle binne. Verspreiding geskied deur kontak, drinkbakke en voer. Die bul kan die virus vir lang periodes dra en afwisselend in die semen uitskei.

Aborsies kan gedurende enige tyd van dragtigheid voorkom, maar vind hoofsaaklik tussen 6–8 maande dragtigheid plaas. Selfs die geboorte van swakontwikkelde kalwers, wat binne enkele ure na geboorte vrek, kan voorkom. Sulke afwykings word dikwels met *Chlamydia*-besmettings gekoppel en kan verwarrend wees.

Die fetus word in 'n gevorderde stadium van vervloeiing verwerp. Die kenmerkendste makroskopiese letsel is die erge bloederige edeem om die niere wat soms teenwoordig is. Fokale nekrose in die lewer en limfkliere word dikwels waargeneem, maar kan ook in die nier, milt, adrenaalkorteks, brein, vel en pankreas voorkom. Histopatologies word die letsel in die nier gekenmerk as 'n erge hemoragiese nekrose van die nierkorteks.

Hoewel intranukluêre inklusies (Cowdry tipe A) nie konstant voorkom nie, kan dit wel in die adrenaal en hepatosiete voorkom.

Die letsels in die vrugvliese is minimaal en die degenerasie volg sekondêr op die afsterwing van die fetus. Daar is 'n hoë konsentrasie van virus in die kotiledons en van hier versprei die virus hematogeen of via die amniotiese vloeistof na die fetus. Die kotiledons is dus 'n goeie weefsel vir virusisolasie omdat virus soms nie van die fetale organe afgesonder kan word nie. Die diagnose kan ook gebaseer word op die aanwysing van 'n styging in neutraliserende teenliggaampies in gepaarde serummonsters.

(g) Bees Parainfluenza-3 Virus[32 75 76]

Alhoewel die virus uit die vrugvliese en baarmoederafskeidings van koeie wat geaborteer het afgesonder kan word, is die rol van die virus nog nie baie duidelik omlyn nie. Slegs enkele van die dragtige diere in 'n kudde aborteer en geen siektesimptome word waargeneem nie. Die geboorte van klein, swakontwikkelde, maar lewendige kalwers is ook 'n moontlikheid.

Die metode van oordraging en verspreiding is ook nog nie duidelik nie. Aborsies het egter voorgekom in kuddes waar dragtige koeie kontak gehad het met kalwers wat asemhalinginfeksies getoon het.

Daar is gewoonlik geen makroskopiese letsels in die fetus nie, maar mikroskopies is daar 'n nekrotiese brongiolitis in die akute stadium wat later ontwikkel in 'n fokale interstisiële pneumonie met peribrongiolêre rondesel infiltrasie.

Die diagnose berus op virusafsondering uit fetale weefsel en die demonstrasie van 'n stygende teenliggaamkonsentrasie. Enkel serumondersoeke het geen waarde nie. 'n Hoë persentasie van die beesbevolking het teenliggame teen die virus.

2. Bakteriese aandoenings

Net soos in die geval van virusaandoenings is daar 'n hele aantal bakteriese siektes wat direk of indirek by die voortplantingsdoeltreffendheid van herkouers betrokke is. Enkele voorbeelde word aangehaal.

(a) Chlamydiose[43 53 65]

Aborsies is slegs een manifestasie van 'n meer komplekse siektebeeld wat deur *Chlamydia psittaci* organismes veroorsaak word. Afhangende van klimaatsomstandighede, boerderymetodes, natuurlike weerstand en ouderdom van die dier kan die siektebeeld verskillende vorms aanneem. Benewens erkende aandoenings soos ensoötiese aborsie van skape en episoötiese aborsie van beeste blyk dit dat hierdie organismes ook verantwoordelik gehou kan word vir uitbreke van konjunktivitis, longontsteking en dermsteurnisse by lammers en kalwers.

Chlamydia organismes kan slegs intrasellulêr vermenigvuldig. Die besmetlike vorms, nl. die elementêre liggaampies is rond tot ovaalvormig en wissel in grootte van 200–300 nm. Die nie-besmetlike vorms, nl. die retikulêre liggaampies is pleomorfies en wissel in grootte van 500–1000 nm. *Chlamydia* organismes is besonder gevoelig vir sonlig, hitte en uitdroging. Antibiotika

beïnvloed slegs die elementêre liggaampies en het geen uitwerking op die retikulêre liggame nie.

Weinig is bekend oor hoe die organisme 'n vatbare populasie binnedring, dissemineer en verskillende siektetoestande verwek. Uit eksperimentele waarnemings blyk dit egter dat oordraging waarskynlik *per os* geskied en dat vrugvliese, baarmoederafskeidings en dooie kalwers die bron van besmetting is. Dit is ook bekend dat die organisme uit die dermkanaal en faeces van gesonde skape, bokke en beeste afgesonder kan word en dat hierdie draerdiere moontlik die siekte in vatbare kuddes kan laat posvat.

Alhoewel dieselfde organisme vir aborsies by beeste sowel as by skape verantwoordelik gehou word, blyk dit tog dat kontak tussen beeste en skape nie noodsaaklik vir die verspreiding en vestiging van die organisme is nie.

Die aantal koeie wat aborteer wissel na gelang van die immuunstatus van die kudde. In 'n vatbare populasie sal 30–40% van die dragtige diere aborteer. In kuddes waar die besmetting reeds gevestig is aborteer slegs enkele diere. Dit gebeur ook selde dat koeie 'n tweede keer aborteer.

Aborsies kom hoofsaaklik voor in koeie wat 7–8 maande dragtig is. Geen klinies waarneembare simptome van dreigende aborsie is in die koeie te bespeur nie. Heel dikwels word swak ontwikkelde maar lewendige kalwers gebore wat binne enkele ure vrek.

Beesfetusse wat voor sewe maande ouderdom verwerp word, wys weinig kenmerkende letsels behalwe bloederige onderhuidse edeem en vogaansameling in die bors- en buikholte. Die organe is gewoonlik in 'n gevorderde stadium van vervloeing en die vrugvliese is ooglopend edemateus en verdik.

Voltydse doodgebore kalwers, asook die wat lewendig aankom maar wat binne enkele ure vrek, wys speldpunt bloedings op die slymvliese van die tragea, tong, bekholte, abomasum en die konjunktiva. 'n Vergrote, geswolle lewer met onreëlmatige oppervlakte word soms aangetref. Die kenmerkendste letsel is egter die duidelike waarneembare limfadenopatie. Veral die mesenteriese limfknope is vergroot en edemateus. Ander makroskopiese letsels van belang is septale longedeem, askites, serofibrineuse pleuritis en peritonitis.

Histopatologies bestaan die lewerletsel uit 'n matige portale reaksie, Kupfferselproliferasie en enkele verspreide nekrotiese foki. Fokale nekrose in die nier met infiltrasie van mononukluêre selle asook nekrose in die milt tesame met proliferasie van die retikulo-endoteelsisteem word soms gesien. Die brongopneumonie soos vir *Brucella* gevalle beskrywe kom selde voor. Hierteenoor is pneumonitis en perivaskulêre "cuffing" van die kapillêre in die brein en meninges baie meer algemeen.

Chlamydia psittaci veroorsaak 'n nekrotiese plasentitis. 'n Fibrinopurulente eksudaat infiltreer die plasenta en 'n geelbruin eksudaat word gewoonlik aangetref tussen die uterus en chorion in die interplasentale en periplasentale areas. Die allantochorioniese membrane is edemateus met foki van nekrose, leukosiet infiltrasie en 'n nekrotiese vaskulitis. Verder is die plasentome nie erg aangetas nie en die geelgrys marokko-leeragtigheid van die interkotiledon area wat 'n kenmerk is in brucellose, word nie in chlamydiose waargeneem nie. In albei die siektes kan groot hoeveelhede eksudaat die vrugvliese bedek. Organismes is meestal tot die interkotiledon area beperk.

Die persentasie ooie wat aborteer wissel na gelang van die immuunstatus van die kudde. In ten volle vatbare kuddes mag 60–70% van die ooie aborteer. In kuddes waar die besmetting meer kronies is, kan van 5–10% aborsies per jaar plaasvind.

Die ooi toon geen tekens van sistemiese siekte of naderende aborsie nie. Die eerste teken van vrugresorpsie is 'n swak lampersentasie. Die boer verkeer onder die indruk dat die ooie oorgeslaan het.

Vroeg in die uitbreek sal die oplettende boer aborsies onder ooie wat 2–3 maande dragtig is, opmerk. Dikwels is bloedvlekke aan die hakskene en op die melkspieël die enigste sigbare teken. Die nageboorte, wat dikwels agterbly, mag ook sigbaar wees. Hierdie lammers word selde gevind. Tekens van verstening of mummifisering word algemeen gesien. Oppervlakkig beskou, mag dit verkeerdelik as misvormings gediagnoseer word.

Namate die verwagte lamtyd naderkom, word groter lammers wat kort na afsterwing verwerp word en wat heeltemal vars mag wees, opgemerk. Gedurende hierdie tydperk begin ook die eerste vroeggebore, klein, maar lewende lammertjies aankom. Hulle mag, hoewel swak, heeltemal lewensvatbaar wees en onder gunstige toestande normaal ontwikkel.

In die geval van aborsies en vroeggebore lammers bly die nageboorte gewoonlik vassit, hoewel die lam ook net so in sy vrugvliese afgegooi mag word. As komplikasie van die agtergeblewe nageboorte kan baarmoederontsteking ontwikkel. In die Republiek word sulke gevalle egter selde teëgekom.

(b) Vibriose[24 35 65]

Vibriose is 'n toestand wat in beeste aanleiding gee tot vroeë embrionale afsterwing, onreëlmatige estrussiklusse, verlaagde vrugbaarheid en met uitsondering enkele aborsies. Dit moet as een van die belangrikste oorsake van onvrugbaarheid in die Republiek beskou word. Slegs enkele van die *Vibrio* spesies of *Campylobacter* soos dit vandag bekend staan is patogeen. Wat die bees betref is slegs *C. fetus* subsp. *foetus* en *C. foetus* subsp. *intestinalis* van belang. Eersgenoemde kan net in die geslagskanaal van die bees vermenigvuldig nie.

Die steriliteitsverwekkende *C. foetus* subsp. *foetus* word by uitstek deur middel van dekking versprei. Onder spesiale omstandighede is dit egter wel moontlik dat hierdie organismes van bul tot bul oorgedra kan word – bv deur hantering gedurende versameling van saad en deur besmette beddegoed.

Die vroulike dier is deurgaans vatbaar vir die siekte. Die organisme gee aanleiding tot 'n anterior vaginitis en fokale endometritis. Meeste van die vroulike diere ontwikkel 'n weerstand na blootstelling. Enkeles bly egter draers en dien as bron van besmetting vir die volgende teelseisoen. Bulle kan na blootstelling tydelik of permanent besmet raak. Die jonger bulle verloor weer die infeksie maar bly 'n gevaar gedurende die teelseisoen, want hulle tree op as meganiese draers. Bulle ouer dan 3 jaar bly gewoonlik permanent besmet.

C. foetus subsp. *intestinalis,* in teenstelling, is van meer belang sover dit aborsies betref. Hoe die toestand in die bees versprei is onbekend. Dit is egter duidelik dat hierdie stam 'n besondere tropisme vir die dragtige baarmoeder vertoon maar die organisme kan ook uit die faeces van normale beeste en skape afgesonder word.

Indien aborsies voorkom is dit gewoonlik beperk tot enkele gevalle in koeie wat tussen 4–7 maande dragtig is. Slegs met uitsondering word groter fetusse verwerp. Die verse of koeie wys geen siektesimptome nie en geen spesifieke letsels in in die fetus waarneembaar nie. Die fetus word kort na afsterwe in 'n vars toestand verwerp.

Die vrugvliese is edemateus en bloedryk, maar is egter nie so erg aangetas as wat in *Brucella*-besmette gevalle gevind word nie. Die kotiledons is bloedryk, bros en nekrose kan in enkeles waargeneem word.

Vibriose by skape is 'n siektetoestand wat gekenmerk word deur laat aborsies, die geboorte van volwasse dooie lammers of swak lewendige lammers wat binne die eerste paar dae na geboorte vrek. Die veroorsakende organisme is *C. foetus* subsp. *intestinalis* en besmetting geskied deur die inname van besmette kos of drinkwater.

Die besmetting is eers gedurende die laaste paar jaar in skape in Suid-Afrika vasgestel en dit is hoofsaaklik onder ingevoerde diere waar die probleem ernstige afmetings aangeneem het.

Die belangrikste teken van die teenwoordigheid van vibriose in skape is die voorkoms van aborsies 2–4 weke voor die lamseisoen aanbreek. In werklikheid is daar enkele vroeë aborsies wat gewoonlik ongemerk verbygaan. Dit is juis hierdie aborsies wat die weiding en waterbronne besmet. Ooie 4 maande dragtig is besonder gevoelig vir die infeksie en 'n aborsiestorm word ontketen. Tussen 20–60% van vatbare ooie mag aborteer 10–18 dae na infeksie. In kuddes waar daar alreeds 'n weerstand bestaan is die aborsies selde meer as 50%. Hierdie enkele aborsies kan ongesiens verbygaan of soms as normaal aanvaar word. Daarom is dit hoofsaaklik net met nuwe uitbreke waar die diagnose gemaak word.

Campylobacter foetus subsp. *intestinalis* (Berg serotipe C) wat aborsies by skape kan veroorsaak gee aanleiding tot 'n kenmerkende letsel in die lewer van die fetus. Die letsel kom in ongeveer 10% van lammers voor en bestaan uit 'n aantal ligbruin omskrewe opgehewe nekrotiese areas van 1–2 mm tot self 10–20 mm in deursneee. Hierdie lewerletsels moet egter onderskei word van putrifaksie en nekrobassilose wat met naelstringinfeksies gepaard gaan.

(c) Leptospirose[24 35 71 78]

Leptospirose is 'n siektetoestand wat 'n wye gasheerspektrum openbaar en net met uitsondering as oorsaak van fetale verwerping by beeste in Suid-Afrika in verband gebring kan word.

Die indringsvermoë van *Leptospira* is afhanklik van die virulensie van die organisme en die weerstand wat die gasheer openbaar. Indringing vind plaas deur die slymvliese, velwonde en deur *per os* inname van besmette water. Die organismes lokaliseer in die urinogenitale stelsel en word deur die uriene uitgeskei. Die introduksie van besmette beeste, varke of knaagdiere word deurgaans verantwoordelik gehou vir die verspreiding onder 'n vatbare populasie.

Kliniese simptome van besmetting kan aborsies voorafgaan, maar dit kan ook dikwels gebeur dat geen tekens dat daar 'n afname in melkproduksie is, dat die melk rooi gekleur is en dat die koei vir 'n dag of twee kroes is. Onder sekere omstandighede kan mortaliteit onder kalwers en enkele volwasse beeste voorkom. In hierdie gevalle is die geswolle geel lewer, algemene geelsug en bloedings in die niere kenmerkend.

Alle besmette koeie sal nie noodwendig aborteer nie. Die koei aborteer gewoonlik 2–3 weke na die akute fase verby is. Die fetus word 24–48 uur na afsterwe verwerp en dus is daar weinig kenmerkende letsels te bespeur.

Omdat die fetus eers 24–48 uur na afsterwe verwerp word is dit uiters moeilik om die organisme uit fetale weefsel af te sonder. Uriene en vrugvliese van verdagte gevalle kan vir immunofluoresensieondersoeke aangebied word. Die organismes kan ook met spesiale kleuring (Levaditi en Warthin-Starry) in die histologiese snitte van niere en vrugvliese opgewys word. Immunofluoresensie verskaf beter resultate in geoutoliseerde fetusse, maar in vars materiaal is histopatologie gekombineer met kweking van die bakterium meer doeltreffend as immunofluoresensie.

Besmette gevalle kan ook geredelik serologies geïdentifiseer word. Hiervoor word serummonsters benodig wat gedurende die akute fase en weer 3 weke later geneem is. 'n Styging in die agglutinasie-titer kan as betekenisvol aanvaar word.

(d) Listeriose[8 23 24 35 47]

Listeriose kom wêreldwyd voor, het 'n wye gasheerreeks en word gekenmerk deur septisemie, enkefalitis of sporadiese aborsies. Dit gebeur selde dat meer as een van die drie sindrome tesame voorkom. Behalwe vir die onlangse uitbreek in bokke in die westelike Kaapprovinsie, is die siekte nog nie vantevore by herkouers in die Republiek vasgestel nie.

Die organisme dring die liggaam binne deur slymvliese van die asemhaling- en spysverteringstelsels. Die lae indringingsvermoë verklaar waarom slegs enkele diere klinies-waarneembare simptome van besmetting openbaar. Latente draers kom dikwels voor. Behalwe dat *L. monocytogenes* wydverspreid in die natuur voorkom en dat besoedelde hooi, kuilvoer en water heelwaarskynlik die bron van besmetting is, wil dit voorkom asof bosluise (*Dermacentor* en *Ixodes* spesies) ook 'n rol in die verspreiding van die siekte kan speel. Die organisme kan uit semen van besmette bulle afgesonder word, maar nog geen bewys van veneriese oordraging is gedokumenteer nie.

So ver dit aborsies betref, is dit hoofsaaklik koeie wat tussen 7–8 maande dragtig is wat fetale verwerping ondergaan. Die dier wys geen siektesimptome nie en ook geen tekens van 'n dreigende aborsie nie. As die besmetting voor die 7de maand van dragtigheid posvat kan dit gebeur dat die konseptus eers 4–5 dae na afsterwe verwerp word.

As gevolg van die geoutoliseerde toestand van die fetus is die histopatologiese ondetsoek van die fetus onbevredigend. Makroskopiese letsels word in ongeveer 13% van lammers en fetusse gesien en bestaan uit fokale nekrose van die lewer, long, milt en adrenaalkorteks. Mikro-absessies kan mikroskopies in bogenoemde organe voorkom in die afwesigheid van makroskopies waarneembare letsels. Koagulatiewe nekrose in die lewer kan dikwels in meer as 70% van gevalle gesien word en die bakterieë kan dan in die foki opgewys word.

Fokale nekrose in die kotiledons kan in party gevalle erg wees sodat wit kolletjies met die blote oog sigbaar is.

Die diagnose berus op die afsondering en identifikasie van die organisme. Deur die orgaanmateriaal vir 'n tydperk (1–6 maande) by 4°C te stoor word die kweking van die bakterium vergemaklik. Verwarring met *Streptococcus* en *Haemophilus* organismes in smere van aangetaste orgaanmateriaal is moontlik. Hierdie probleem kan oorbrug word indien van immunofluoresensie-tegnieke gebruik gemaak . word.

(e) Salmonellose[24 28 29 30 35]

Salmonellose is 'n siektetoestand van huisdiere wat 'n wesenlike gevaar vir die mens inhou. Aborsies is slegs 'n enkele sindroom van *Salmonella*-besmettings en indien aborsies in die besmette kudde voorkom is dit dan ook net in enkele gevalle en selde meer as 5–10% van die besmette diere.

Die organisme het 'n wêreldwye verspreiding en word net by uitsondering as die oorsaak van aborsies by beeste in die Republiek gevind. *Salmonella dublin* is by uitstek die serotipe wat uit fetale materiaal afgesonder kan word. Hierdie organismes is moeilik van ander dermbakterieë te onderskei en derhalwe moet uitgebreide serologiese en biochemiese ondersoekmetodes gebruik word. Die organisme vestig gemaklik in die dermkanaal en 'n groot aantal dermdraers kan geïdentifiseer word. Enkele van die serotipes kan vir etlike maande in grond lewe.

Daar bestaan die moontlikheid dat geslagtelike oordraging in skape kan plaasvind, maar geen bewys is egter gevind dat dit in beeste ook kan gebeur nie. Besmetting kom gewoonlik die kudde binne deur die aankoop van draerdiere, maar 'n verdere moontlikheid is dat knaagdiere voedselbronne soos karkas- en vismeel kan besmet.

Geen siektetekens word in die gevalle wat aborteer gesien nie. Aborsies vind gewoonlik plaas in koeie wat tussen 6–8 maande dragtig is en fetale afsterwe volg op 'n *in utero* septesemie. Die konseptus word in 'n gevorderde toestand van ontbinding verwerp, maar klein afgebakende areas van lewernekrose kan soms nog waargeneem word.

Die diagnose word gebaseer op die isolasie van *Salmonella* organismes uit die konseptus, vrugvliese en baarmoederafskeidings. Versigtigheid moet aan die dag gelê word wanneer fetale materiaal, wat met faeces besoedel is, ondersoek word. 'n Foutiewe gevolgtrekking van *Salmonella* betrokkenheid is 'n rieële moontlikheid.

(f) Brucellose[15 24 35 65]

Die aborsie-verwekkende eienskap van *Brucella* is welbekend en bly een van die belangrikste probleme waarmee ons te kampe het. By beeste is *B. abortus* die belangrikste oorsaak van aborsies. Alhoewel die patogeniteit van stamme wissel blyk dit tog dat *B. abortus* in die breë gekenmerk word deur 'n lae besmetlikheid en geringe weefselvernietiging.

Die organismes lokaliseer in die baarmoeder en uierlimfkliere en besmette koeie skei periodiek organismes in die melk uit. Slegs 10% van besmette koeie sal 'n tweede keer aborteer.

By beeste geskied aborsie gewoonlik tussen 6–8 maande van dragtigheid en waar die infeksie 'n vatbare populasie binnedring kan tot en met 40% van die diere aborteer. In kuddes waar brucellose reeds die kroniese vorm aangeneem het, word dikwels lewendige, swak ontwikkelde en besmette kalwers gebore wat binne enkele dae na geboorte vrek.

Brucella veroorsaak 'n nekrotiese plasentitis. Alhoewel die letsel in die fetale plasenta redelik kenmerkend is kan dit nie as patognomonies vir die siektetoestand beskou word nie. Soortgelyke letsels maar in 'n ligter graad kan deur *C. foetus* subsp. *intestinalis* organismes veroorsaak word. 'n Meer uitgesproke plasentitis kan met sekere swambesmettings waargeneem word.

Dikwels kan 'n aansameling van 'n reuklose, vuilgeel en effens slymerige eksudaat, wat enkele geelgrys flokkules bevat, in die interkotiledon spesies tussen die endometrium en die chorion waargeneem word. Die fetale membrane is deurgaans verdik en edemateus terwyl net enkele van die kotiledons nekroties is. Die interkotiledonareas direk rondom die kotiledons is gewoonlik verdik en het 'n leeragtige tekstuur.

Histologies word die letsel gekenmerk deur uitgesproke nekrose van die epiteelselle van die chorion villi. Baie van hierdie afskilferende en nekrotiese epiteelselle is gepak met kolonies *Brucella* organismes. Vaskulitis, trombose, edeem en infiltrasie van inflammatoriese selle en veral neutrofiele word dikwels in die aangetaste gedeeltes opgemerk.

Makroskopies kan in die fetus van 8–9 maande ouderdom wat gewoonlik kort na afsterwing verwerp word, strooikleurige vloeistof aansameling in die liggaamsholtes gesien word. Fibrienstolsels en fibrienstinge kan ook dikwels in die buik en borsholte waargeneem word. Die fokale gedissemineerde brongopneumonie of klein gryswit foki van nekrose, 1–2 mm in deursnee in die long, tesame met die vergrootte lewer en limfkliere is redelik kenmerkend. Die abomasum is dikwels gevul met 'n taai, troebel, geelkleurige vloeistof.

Histopatologies bestaan die letsel in die long uit 'n brongopneumonie, vaskulitis, trombose in enkele vate en verspreide foki van nekrose. Wat die lewer betref word dikwels mikrogranulome, proliferasie van die Kupfferselle en 'n proliferatiewe portale reaksie waargeneem.

Die konseptus wat voor 7 maande ouderdom verwerp word is deurgaans in 'n gevorderde stadium van vervloeiing en daar kan prakties geen weefselveranderinge waargeneem word nie. In teenstelling hiermee kan 'n diagnose van Brucella-besmetting in 85% van gevalle op grond van die histopatologiese veranderinge in ouer fetusse en vrugvliese gemaak word.

3. Protosooïese siektes en Rickettsias

(a) Toxoplasmose[3 4 24 34 35 54 81 82]

Alhoewel die organisme reeds in die mens, die hond en kat geïdentifiseer is, is nog net enkele gevalle by skape in die Republiek aangetref. Nog geen gevalle is by beeste aangeteken nie.

Waar dit wel voorkom word prakties geen siektesimptome in die vroulike dier (behalwe vir die verwerping van besmette fetale materiaal) waargeneem nie. As die besmetting gedurende die vroeë fases van dragtigheid plaasvind kan vrugresorpsie plaasvind en die skaapboer onder die indruk laat verkeer dat sy ooie oorgeslaan het. Aborsie geskied gewoonlik laat in dragtigheid en tot 15% van die ooie kan aborteer.

Die afsondering van die organisme is besonder moei-

lik en derhalwe berus die diagnose op serologiese ondersoeke en histopatologiese identifikasie.

Die fetusse toon meestal geen makroskopiese letsels nie en slegs in sommige gevalle kan organismes en histopatologiese letsels in die brein, hartspier, lewer en long aangetref word. Die letsels in die brein blyk meer konstant voor te kom en bestaan uit 'n inflammatoriese reaksie wat soms gepaard gaan met sentrale nekrose. Siste word nie algemeen gesien en kom gewoonlik voor waar geen nekrose teenwoordig is nie.

Die letsels in die vrugvliese is besonder karakteristiek en is veral duidelik waarneembaar as die vrugvliese in fisiologiese soutoplossing gespoel word sodat die chorioniese villi los in die vloeistof dryf. Waar die kotiledon normaalweg rooi-pers tot rooibruin gekleurd is, kom die aangetaste kotiledons as helder- tot donkerrooi voor. Wit vlekkies (klein wit sagte nodules van 1–3 mm in deursnee) kom verspreid voor in die chorioniese villi van die kotiledons. Die organismes kan redelik maklik in die areas met 'n vroeë stadium van nekrose opgemerk word. In erg besmette gevalle kan areas van mineralisasie ook in die kotiledons aangetref word.

(b) Trichomoniase[24 35 65]

Trichomoniase is 'n streng veneriese siekte van beeste wat deur *Trichomonas foetus* veroorsaak word en baie ooreenstem met die siektebeeld van vibriose.

Hierdie organisme het 'n tipiese piriforme morfologie, is ongeveer 5–15 μm lank en 7 μm in deursnee. Die organisme is vry beweeglik en word gekenmerk deur 3 anterior flagella en 'n golfmembraan. Die *Trichomonas foetus* organismes van beeste in die Republiek is almal serologies identies.

Hierdie geslagsiekte kan net deur koitus versprei. Na blootstelling ontwikkel meeste vroulike diere 'n weerstand wat egter maar van korte duur is. Net soos in die geval van vibriose is daar egter enkele gevalle waarin die organisme gevestig raak en die diere dan as draers optree.

Klinies kan hierdie aandoening nie van *C. foetus* subsp. *foetus* infeksies onderskei word nie. Die nadelige effek op die voortplantingsdoeltreffendheid van die bees is beperk tot die embrionale periode. Abnormale estrussiklusse en vroeë vrugafsterwing met swak bevrugtingssyfers is die kenmerkende eienskappe. Enkele gevalle van piometra kan in *T. foetus*-besmette kuddes gevind word.

(c) Babesiose en anaplasmose[6]

Dragtige koeie wat rooiwater of anaplasmose opdoen aborteer dikwels gedurende die koorsreaksie van die siekte. Transplasentale oordraging van die babesia's kan plaasvind en dan kan die organismes in die rooibloedselle in brein- en bloedsmere gedemonstreer word.

Indien besmetting van die fetus plaasvind, is die fetus gewoonlik anemies, mag 'n ikterus toon en die lewer en milt is meestal vergroot. Die lewer het ook 'n meer geel-oranje voorkoms as normaal a.g.v. die degeneratiewe veranderinge en aansameling van galpigmente.

(d) Q-Koors (Coxiella burnetti besmetting)[44 66 74 77]

Alhoewel hierdie siekte reeds 20 jaar gelede in beeste in die Republiek aangeteken was is dit maar slegs onlangs dat hierdie organisme met perinatale verliese by skape en beeste in verband gebring is.

Redelik min is bekend oor die simptomatologie van Q-Koors as natuurlike besmetting in herkouers. Die organisme kan soms as 'n suiwer kultuur van plasentas en uit baarmoedervog afgesonder word sonder dat daar enige siektetekens te bespeur was. Dit is gewoonlik net enkele van die vroulike diere wat na natuurlike blootstelling aborteer. In die kuddes waar grootskaalse aborsies voorkom en waar *Coxiella* organismes afgesonder word moet ook ondersoek ingestel word na die teenwoordigheid van ander patogene organismes. Dikwels is daar 'n gemengde besmetting met byvoorbeeld *Chlamydia, Toxoplasma* of *Vibrio.*

Die belangrikheid van hierdie organisme lê nie soseer opgesluit in die aborsies wat veroorsaak word nie, maar wel in die wesenlike gevaar wat dit vir die mens inhou. Die organisme kan weke lank buite die liggaam lewe en derhalwe is dit noodsaaklik dat die besmette foki geïdentifiseer en vernietig word.

Die letsels in die plasenta stem baie ooreen met die wat in *Brucella*-besmettings aangetref word, maar is gewoonlik ligter van graad. Morfologies kan *C. burnetii* met moeite van *Brucella* onderskei word.

4. Swambesmettings[1 19 27 45]

Dit is slegs met uitsondering dat aborsies as gevolg van swambesmetting voorkom. Selfs met eksperimentele blootstelling kom fetale verwerping net in enkele gevalle voor.

Bykans 80% van alle aborsies wat aan swambesmettings toegeskrywe kan word, word deur *Aspergillus fumigatus* veroorsaak. *Mucor, Absidia* en *Rhizopus* spesies word slegs sporadies afgesonder.

Hierdie swamme kom wydverspreid voor in die natuur en alhoewel stamme heel dikwels uit voer en hooi afgesonder word kan hierdie besmettingsbron tot dusver nog nie aan sporadiese aborsies gekoppel word nie.

Die rede waarom slegs enkele gevalle (selfs na daaglikse blootstelling) aborteer bly onbekend. Klaarblyklik moet daar, soos in die geval van *B. abortus* en *C. foetus* subsp. *intestinalis* besmetting, 'n primêre letsel in die liggaam wees. In die geval van swamme is die primêre letsels in die longe, lewer of dermkanaal, waarvandaan die swam die baarmoeder binnedring.

Fetusse van tussen 6–8 maande oud word gewoonlik verwerp, terwyl die vrugvliese eers enkele dae later vrykom. Die meeste fetusse kom normaal voor, terwyl enkeles kenmerkende velletsels, wat soos omlope lyk, toon. Hierdie velletsels kom meer dikwels om die oë, die kop en op die rug voor. 'n Brongopneumonie gaan soms hiermee gepaard. Die nageboorte het 'n kenmerkende leeragtige tekstuur en die kotiledons is merkbaar vergroot en nekroties, grysgeel areas 5–10 mm in deursnee strek soms in die plasentome in.

Die swamme kan mikroskopies met behulp van spesiale kleuring in en om plasentale bloedvate in die kotiledons en soms ook in die interkotiledon areas waargeneem word.

Aspergillus en *Mucor* mag kontaminante wees in die veld of in die laboratorium en dus moet die isolasie van een of ander van die swamme met die histopatologie van die plasenta gekorreleer word alvorens 'n besliste diagnose daarvan gemaak kan word. Aan die anderkant kan 'n definitiewe diagnose slegs op grond van die

histopatologie gemaak word alhoewel die volledige identifikasie van die swam soms bemoeilik word sonder isolasiewerk.

Swamme kan veral van 3 lokaliteite in die fetus afgesonder word nl. van die maaginhoud, velletsels en soms ook van die longe. Vir isolasie van die swam moet die vrugvliese en maaginhoud ook altyd ingestuur word. Volgens Miller en Quinn[45] is daar dikwels 'n erge follikulitis in die ooglede van swambesmette fetusse en swamdrade kan in die gekeratiniseerde epidermis gedemonstreer word. Dit is dus raadsaam om van die ooglede monsters vir isolasie en histopatologie te neem indien 'n swambesmetting vermoed word.

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THE EFFECT OF SOME ORGANOPHOSPHORUS ACARICIDES AND THE TIME OF APPLICATION ON LARVAE OF COMMON TICKS IN THE EASTERN CAPE OF SOUTH AFRICA

Y. RECHAV, G.B. WHITEHEAD and S.B. TERRY*

ABSTRACT: Rechav Y.; Whitehead G.B.; Terry S.B. **The effect of some organophosphorus acaricides and the time of application on larvae of common ticks in the eastern Cape of South Africa.** *Journal of the South African Veterinary Association* (1978) **49** No. 2, 99–101 (En) Tick Research Unit, Rhodes University, Grahamstown, 6140, Rep. of South Africa.

Mortality curves of larvae of five common tick species dipped in dioxathion, chlorphenvinphos and oxionthiophos are presented. The mortality of three species of ticks were examined at various times of the day. The results showed that at 14h00 the larvae were more sensitive to acaricide treatments than at other times of the day.

INTRODUCTION

The ticks *Amblyomma hebraeum* (Koch), *Boophilus decoloratus* (Koch), *Hyalomma marginatum rufipes* (Koch), *Rhipicephalus appendiculatus* Neumann and *R. evertsi evertsi* Neumann are known as vectors of *Theileria parva, T. lawrencei, T. mutans, T. annulata, Babesia* spp., *Anaplasma* spp. and *Cowdria ruminanium,* and because of this are considered as serious threats to successful farming of stock in many areas of South Africa. (For details see appendix.)

The common method of tick control in this country is by immersing domestic animals in tanks containing acaricides. However, the tick species mentioned above have developed resistance to chlorinated hydrocarbon acaricides,[2 3 13 14 15 16] this has stimulated further investigations into the use of new acaricides such as the organophosphorus compounds. Unfortunately, strains of ticks resistant to organophosphorus compounds have also developed[4 5].

It is known that some arthropods show a diel periodicity in their sensitivity to insecticides[6 7 9 10 11 12]. This phenomenon has been found in the housefly[7 10 11 12], beetles[9] and mites[8]. This fact might be used for improving control.

To our knowledge, no published information is available on the susceptibility of ticks at various times of the day to acaricides. Such information is essential for succeeding in our efforts to improve tick control.

This paper gives the mortality curves of the offspring of ticks, which had been collected at various farms in the Eastern Cape Province of South Africa, and that had been exposed to dioxathion*, chlorphenvinphos**, and oxionthiophos***. The ticks used have been retained in our laboratory and will serve as a standard reference to detect resistant strains of ticks which may in the future be collected from various farms. Preliminary experiments showed that diel periodicity in sensitivity of tick larvae to acaricides occurred.

METHODS

Batches of larvae bred from field collections were dipped in various acaricide preparations using Whitehead's method[14]. About 100 three week old larvae were immersed in 10 ml of the acaricide. The larvae were agitated in the suspension of the tested acaricide for 1 min, then separated from the acaricide preparation by pouring the mixture (suspension and larvae) through gauze, over an Erlenmeyer funnel which had previously been connected to a vacuum pump. The larvae were rinsed twice with distilled water and then transferred on the gauze to filter paper envelopes. The envelopes were maintained for 24 h at 26°C and 75% RH before recording mortality. Distilled water controls were run concurrently with each test and corrections for control mortality were made by using Abbot's formula.

Each test was repeated 12 times with larvae hatched from batches of 25 females and which had been kept under light:dark regimes of 12:12.

The temperature of the various acaricide preparations at the time of application was kept constant (22 + 1°C) i.e. the possibility of variable results due to temperature fluctuations was eliminated.

RESULTS

The mortality curves, based on five concentrations, of each acaricide are presented in Fig. 1.

The data in Fig. 1 show that a slight increase in the concentrations of the test acaricide greatly increased the mortality of *A. hebraeum* larvae. In *B. decoloratus* the result of increasing the concentrations of the acaricides was only a slight increase in mortality.

The LC50 values (percentage concentration required to produce 50% mortality) of the various acaricides, expressed as a percentage of active ingredient (a.i.) are presented in Table 1. Although data showing both LC50 and LC99 would be of considerable value, we

Table 1: MEAN PERCENTAGE CONCENTRATION (A.I.) OF THREE ACARICIDES REQUIRED TO PRODUCED 50% MORTALITY IN FIVE SPECIES OF TICK LARVAE

	% Concentration of active ingredient of:		
Species	Dioxathion (Delnav)	Chlorphen-vinphos (Supona)	Oxion-thiophos (Bacdip)
Amblyomma hebraeum	0,000039	0,00086	0,000056
Boophilus decoloratus	0,00020	0,0012	0,00030
Hyalomma m. rufipes	0,000042	0,0041	—
Rhipicephalus evertsi	0,00022	0,00031	0,000061
R. appendiculatus	0,00028	—	0,00010

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* Delnav: Coopers S.A.

** Supona: Shell Chemicals

***Bacdip: Bayer

present only LC50 values and statistical calculations showing the slope of the mortality lines (Fig. 1) to avoid extrapolations.

The mortality of three species of ticks treated at various times of the day were examined. The results are presented in Table 2. The data of these preliminary experiments show that, at 14h00, the larvae were more sensitive to acaricide treatment than at other times.

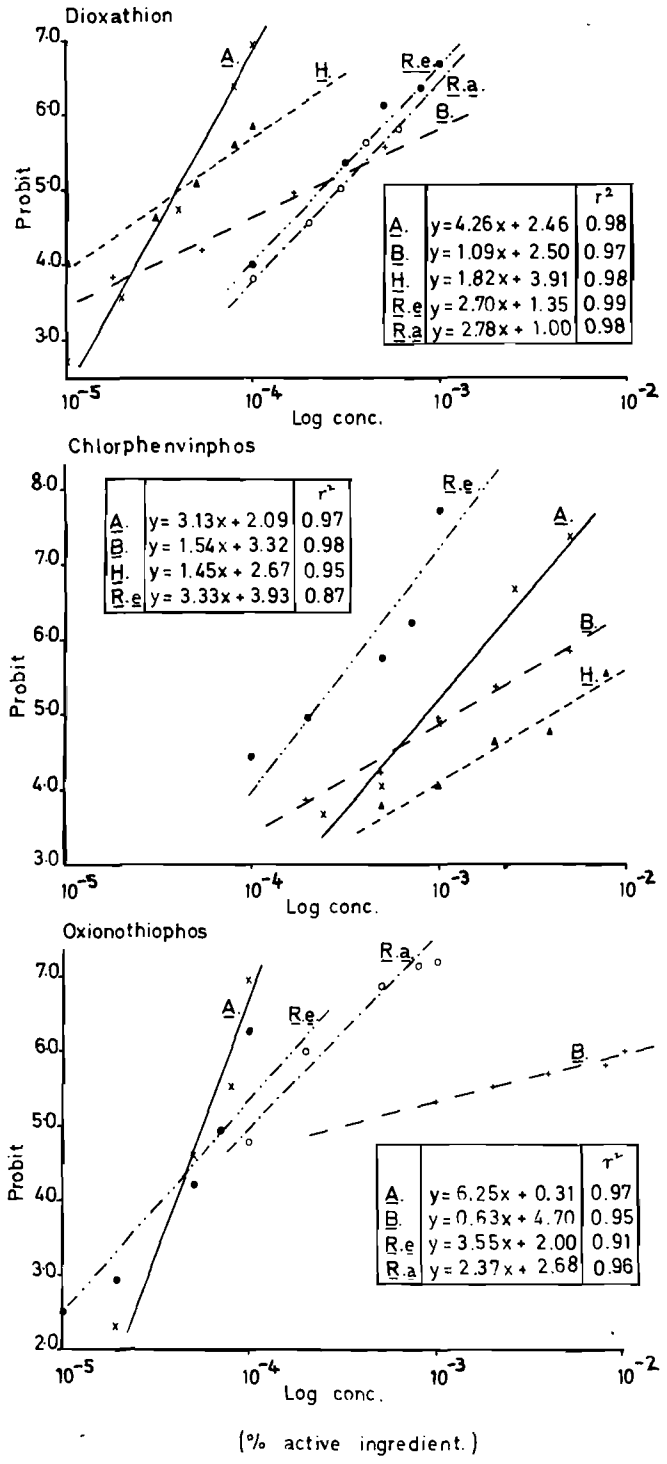

Fig. 1 The effect of dioxathion, chlorphenvinphos and oxionthiophos on larvae of: *A. hebraeum* (X–X), *Boophilus decoloratus* (X–X), *Hyalomma martinatum rufipes* (A–A), *Rhipice phalus appendiculatus* (O-.-.-O) and *Rhipicephalus evertsi evertsi* (O-..-..-O). The slope intercepts and the correlation coefficients %- ... - ... -% are given for each curve.

Table 2: MEAN LC50 VALUES (% A.I.) AS A FUNCTION OF TIME OF APPLICATION. THE LARVAE WERE KEPT IN LIGHT:DARK 12:12H FOR THREE WEEKS BEFORE THE APPLICATION

	06h:00	10h:00	14h:00	18h:00
Dioxathion (Delnav)				
B. decoloratus	0,00023	0,00033	0,00011	0,00018
A. hebraeum	0,000037	0,000066	0,000034	0,000035
H. m. rufipes	0,00081	0,00068	0,00029	0,00027
Chlorphenvinphos (Supona)				
B. decoloratus	0,0016	0,0013	0,0009	0,0013
A. hebraeum	0,00084	0,00066	0,00010	0,00010
H. m. rufipes	0,0093	0,0093	0,0020	0,0027
Oxionthiophos (Bacdip)				
B. decoloratus	0,00046	0,00037	0,00025	0,00052

DISCUSSION

Mortality curves of tick larvae which had been treated with three different acaricides are presented. No known susceptible reference strain was used, so the mortality curves cannot be compared, but, from Fig. 1, there is some indication that *B. decoloratus* and *H. m. rufipes* might have developed resistance to the acaricides examined*. This indication comes from the fact that the distribution of sensitivity to acaricides in tick populations behaves according to Gauss distribution, but when resistance has developed, the distribution of sensitivity becomes asymetric, and as a result, the slope of the mortality regression line becomes shallower.

The results obtained indicate that the larvae of ticks, exhibit diel periodicity in sensitivity to acaricides. It was found that the greatest sensitivity of tick larvae, irrespective of species, to various acaricides occurred at 14h00. The fact that all species used were susceptible to acaricides in a natural regime of light:dark, indicates that this is probably an endogenic character of the ticks. The LC50 values at 06h00 are higher than the values obtained at 14h00. The differences in concentration required to produce a 50% mortality at the various times of the day is small. Although these differences are small, (proof of this in the field, or a stall test, might be impossible to establish in view of the minute nature of the variations shown) it looks as if dipping cattle at 14h00 might give better results.

Appendix: RELATIONS BETWEEN THE PATHOGENS AND THEIR VECTORS

Tick vector	Pathogen
Boophilus decoloratus	*Babesia* sp?, *Anaplasma* sp?
Rhipicephalus appendiculatus	*Theileria parva* *T. lawrencei* *T. mutans*
R. e. evertsi	*T. parva*, Tick Toxicosis T. mutans
Hyalomma rufipes	*T. annulata*
Amblyomma hebraeum	*Cowdria ruminantium*

*It has been observed that *H.m. rufipes* does not respond to chemicals as do other ticks (Baker, 1977, Personal Communication).

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MUSCLE IRRITATION CAUSED BY DIFFERENT PRODUCTS CONTAINING OXYTETRACYCLINE

A. IMMELMAN,* W.S. BOTHA** and DRICKY GRÏB*

ABSTRACT: Immelman A; Botha W.S.; Grïb, Dricky. **Muscle irritation caused by different products containing oxytetracycline.** *Journal of the South African Veterinary Association* **(1978) 49** No. 2, 103–105 (En) Dept. Phys., Pharm. and Tox. Fac. Vet. Science, Univ. Pretoria, Box 12580, Onderstepoort 0110. Rep. of South Africa.

Muscle irritation studies were done in rabbits using 9 different commercial products. Eight products had propylene glycol and 1 had polyvinylpyrrolidone as a vehicle. Macroscopical examination 3,6 and 10 days after intramuscular injection indicated that the irritation caused by the 8 products with propylene glycol as a vehicle were very similar. The product with polyvinylpyrrolidone caused much less damage and healing took place more quickly. Saline used as a control caused no tissue damage.

INTRODUCTION

"All tetracycline antibiotics, when administered intramuscularly may cause severe tissue damage, characterized by necrosis and polymorphonuclear infiltration. Oxytetracycline administered in a propylene glycol-water solvent produced the least tissue damage"[2].

Oxytetracycline as the injectable antibiotic is used by veterinarians and farmers in this country. The clinical signs of pain and irritation after an intramuscular injection are well known to them and they often claim that one commercial product causes less pain and irritation than another.

Lesions may remain in the muscle tissue for a considerable period and it is therefore preferable that injected animals should not be slaughtered within 30 days for human consumption. In an attempt to minimize this undesirable side effect, oxytetracycline was formulated by some manufacturers which uncorporated polyvinylpyrrolidone as a vehicle. The irritant affects of this preparation were compared to those caused by products containing propylene glycol as a vehicle. The results of this comparison form the basis of this report.

MATERIALS AND METHODS

Young male Californian rabbits of approximate 2,5 kg body mass were used. They were housed in individual pens which were maintained at a room temperature of 23°C. Commercial rabbit pellets and fresh water was available *ad libitum.*

The animals were divided randomly into 6 groups, each consisting of 6 animals. The skin over both Mm. longissimus dorsi was shaved of hair 24 hours before the onset of the trial.

Using a 0,5 × 6 mm needle the particular oxytetracycline preparation was injected at a dose of 10 mg/kg body mass into one M. longissimus dorsi. The muscle was penetrated at an angle of 45° in an anterior direction and the drug was deposited 15 mm deep. The volume of the injections varied according to the concentration of the preparations used. Normal saline was used for the control and the volume was calculated as the average of all the other volumes injected.

The oxytetracycline injectable solutions used are commercial products available in South Africa and are listed in Table 1. The exact formulations of these products are not known to us but 8 products have propylene glycol as a vehicle and 1 product has polyvinylpyrrolidone as a vehicle. All were within the expiry date given on the label. The concentrations of oxytetracycline stated on the label were not verified.

Table 1.

Trade name	Manufacturer	Concentration mg/ml	Formulation
Hi-Tet.	Milborrow	120	P.G.
Dabicycline	Chemveld	150	P.G.
Datomycin	Datons	125	P.G.
Beekabycin	Meds Laboratories	100	P.G.
Curramycin	Agricura Laboratories	123	P.G.
Oxycline	Panvet Laboratories	125	P.G.
Oxyvet	Bayer Laboratories	125	P.G.
Liquamycin	Pfizer Laboratories	100	P.G.
Tarramycin 100	Pfizer Laboratories	100	P.V.P.

P.G. – Propylene Glycol.
P.V.P. – Polyvinylpyrrolidone

Each product was injected into 6 rabbits and different products were injected into the left and right longissimus dorsi muscles. On d 3, 6 and 10 two animals per group were killed by injecting pentabarbitone sodium intravenously. The injection sites in the muscles were examined for macroscopical lesions and specimens were taken in 10% buffered formalin for histological evaluation. After fixation for 48 hours a block of muscle tissue incorporating the lesions was cut in a longitudinal section. These blocks were processed and sections 4 μm thick were cut and stained with haematoxylin and eosin (HE).

RESULTS

On macroscopical examination the lesions caused by those products containing propylene glycol as a vehicle were very similar. Fig. 1 represents a typical lesion seen 3 days after the injection. At the site of deposition of the injected material there was a well demarcated area of necrosis. The necrotic tissue varied in colour from light to dark yellow; the colour change being considered to be caused principally by the yellow pigmented oxytetracycline although the necrotic process also contributed to it. In a few instances small haemorrhages were present. In the majority of cases there was no visible haemorrhage. No signs of inflammation were present. On the 6th day the lesions were very similar to those seen after 3 days. Even after 10 days the lesions were very similar to those described above (Fig. 2).

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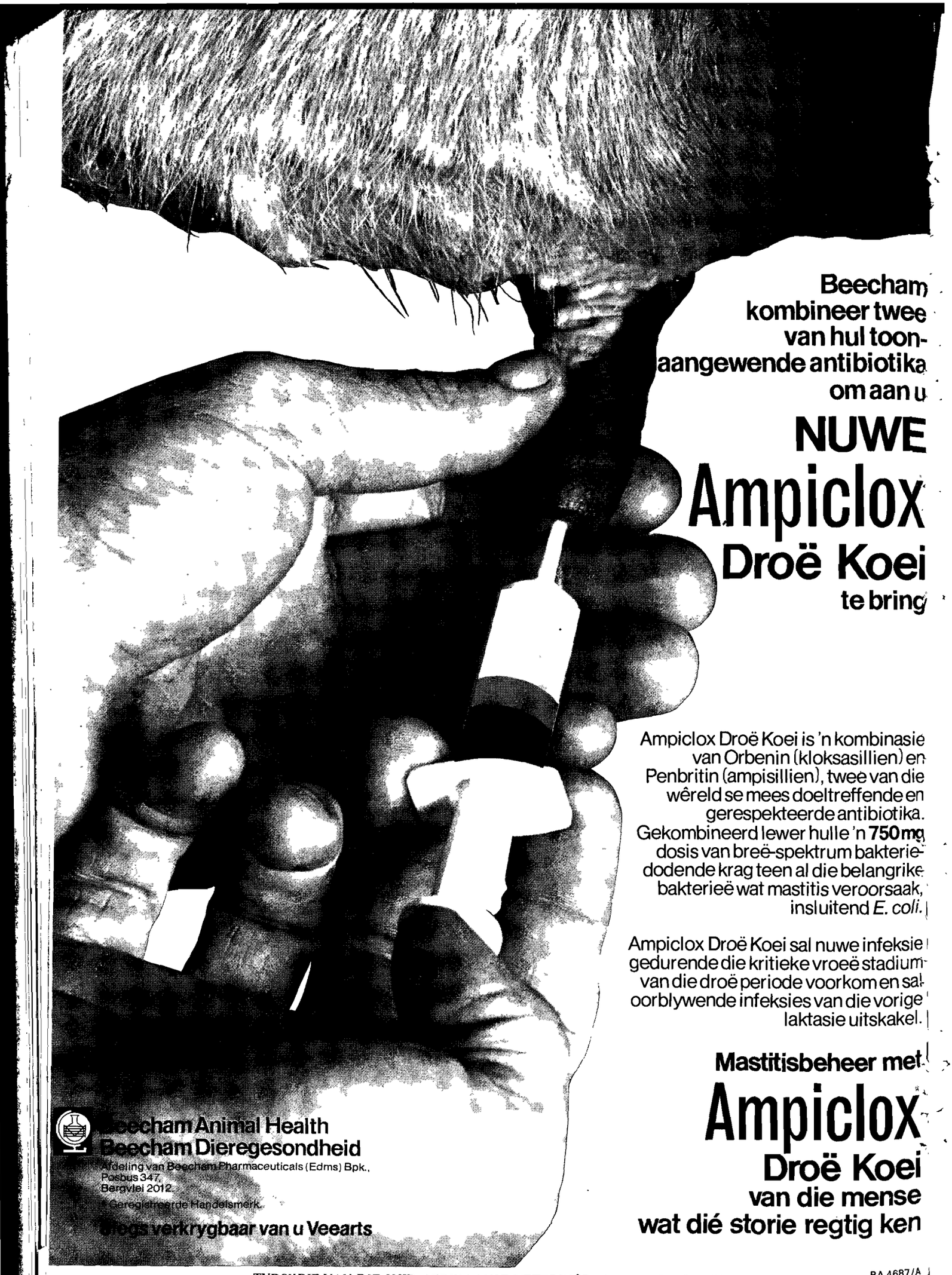

Beecham kombineer twee van hul toon-aangewende antibiotika om aan u

NUWE Ampiclox Droë Koei

te bring

Ampiclox Droë Koei is 'n kombinasie van Orbenin (kloksasillien) en Penbritin (ampisillien), twee van die wêreld se mees doeltreffende en gerespekteerde antibiotika. Gekombineerd lewer hulle 'n **750 mg** dosis van breë-spektrum bakterie-dodende krag teen al die belangrike bakterieë wat mastitis veroorsaak, insluitend *E. coli*.

Ampiclox Droë Koei sal nuwe infeksie gedurende die kritieke vroeë stadium van die droë periode voorkom en sal oorblywende infeksies van die vorige laktasie uitskakel.

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AN INTRODUCTION TO DISEASES OF PIGEONS IN SOUTH AFRICA

F.W. HUCHZERMEYER*

ABSTRACT: Huchzermeyer, F.W. **An introduction to diseases of pigeons in South Africa.** *Journal South African Veterinary Association* (1978) **49** No. 2, 107–108, (En) Department of Infectious Diseases, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

The paper mainly deals with racing pigeons. Beginning with organization, importance and cost of the pigeon sport it then details investigational procedures and techniques for drug administration before discussing some of the infectious diseases common in South Africa as well as the problem of poor racing performance.

In this country pigeon keeping has developed mostly without the help of the veterinary profession. It is only with the introduction of restrictions in the sale of antibiotics and antimicrobials that the pigeon owner has become aware of services which the profession is capable of rendering, and that members of our profession are required to look at disease problems in pigeon.

Farming with pigeons for meat production, using heavy breeds like the "King", which is capable of attaining more than 1 kg live mass, is still very much in its infancy. Therefore pigeon keeping in South Africa is predominantly a hobby with two main purposes, showing and racing. As the latter places extremely high demands on the bird, this paper will deal mainly with racing pigeons. Approximately 2,5 million racing pigeons are owned by 20 000 registered club members in South Africa. Each of the 35 unions organises about 20 races per season, with ± 50 000 pigeons racing every weekend of the season in this country. Races are flown over distances varying between 200 km and 1200 km with prizes to be won on a race of up to R2 000. In addition top prizes of pools are in the region of R300. The cost of racing to the average fancier and to South Africa's pigeon racing community as a whole is detailed in Table 1.

Table 1: COST OF PIGEON RACING

	to the fancier	total in S.A.
Capital outlay:		
loft	R1 500	R30 million
stock	R1 000	R20 million
clock	R250	R5 million
Total	R2 750	R55 million
Annual recurring expenditure:		
feed	R250	R5 million
race money	R250	R5 million
incidental expenses	R100	R2 million
Total	R600	R12 million

When confronted with a disease problem, the first step to take is the clinical examination of one or several birds. For this purpose the bird is held in the upturned hand, its sternum resting in the palm, head towards the holder, both legs gripped between index and middle finger and these two fingers and the thumb closing dorsally over wing tips and tail. The age of the bird is determined by reading the year of birth, which is recorded on the ring. The various parts of the body are now visually examined and palpated.

An oesophageal swab for the examination for *Trichomonas gallinae* is taken in the following way: The bird's neck is stretched by lifting the head while the beak is held open between two fingers. A swab soaked in saline is inserted through the beak and gently pushed into the oesophagus and with a rotating movement right down into the crop. After a few up and down movements it is again withdrawn. A drop of saline is squeezed out of the swab onto a slide, covered with a cover slip and examined microscopically at magnifications from 100 × to 400 ×. Instead of a swab a wire loop can be used and the mucus collected by stirring the loop into a drop of saline on a slide.

In cases of conjunctivitis it may be useful to take corneal impression smear by pressing a cleaned slide against the opened eye. It is then stained and examined microscopically for bacteria and *Chlamydia.*

A drop of blood for a bloodsmear is obtained by puncturing with a fine needle the brachial vein, where it crosses the medial aspect of the elbow joint. As the nuclei of avian erythrocytes tend to obscure the view, the bloodsmear should be very thin.

A fresh faecal sample is examined for worm eggs and coccidia oocysts either directly by mixing into a drop of water on a slide and covering with a cover slip or after flotation. Whenever possible a post mortem examination should be carried out on one or two freshly dead or euthanized birds following the same technique as used for poultry.

No investigation is complete without a visit to the loft. The procedure for the investigation at the loft is set out in Table 2.

Table 2: INVESTIGATION AT THE LOFT

1. The loft
   - 1.1. size, design, materials
   - 1.2. state of maintenance, cleanliness
   - 1.3. inside temperature; insulation
   - 1.4. ventilation, draughts
   - 1.5. implements
     - feeders
     - waterers
     - nests
   - 1.6. feed store, quality of feed
2. The stock
   - 2.1. number of birds, stocking density
   - 2.2. clinical examination of some birds
3. Records
   - 3.1. stock records
     - breeding
     - introductions of new stock
   - 3.2. racing records

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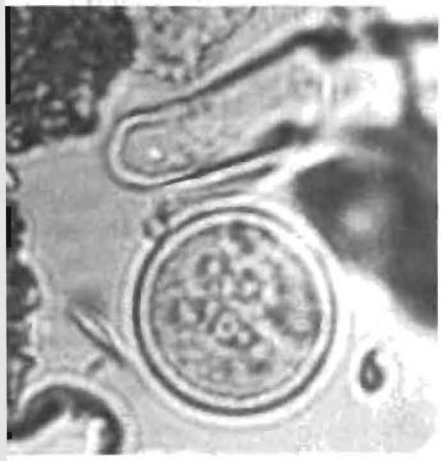

Fig. 1 Cyst of *Entamoeba coli* from baboon (approx. × 1000).

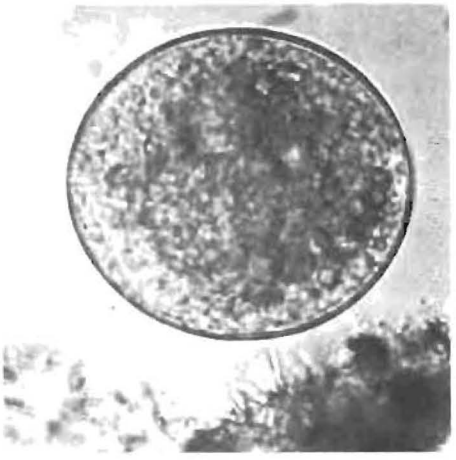

Fig. 2 Cyst of *Balantidium coli* from baboon (approx. × 1000).

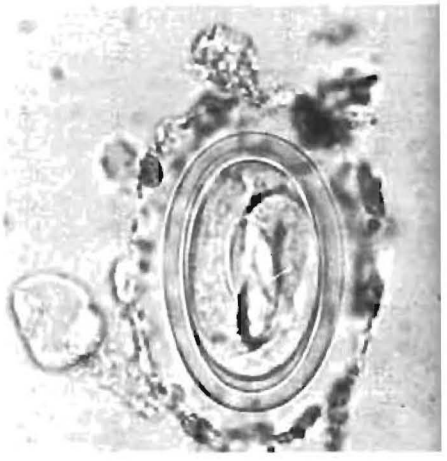

Fig. 3 Egg of *Abbreviata caucasica* from babo (approx. × 500).

Fig. 4 Egg of *Trichuris? trichiura* from baboon (approx. × 500).

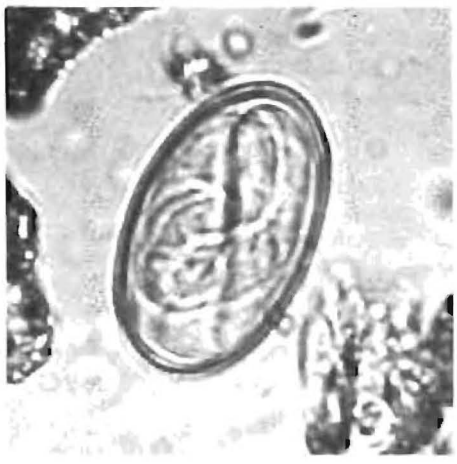

Fig. 5 Egg of *Streptophargus* sp. from baboon (approx. × 800)

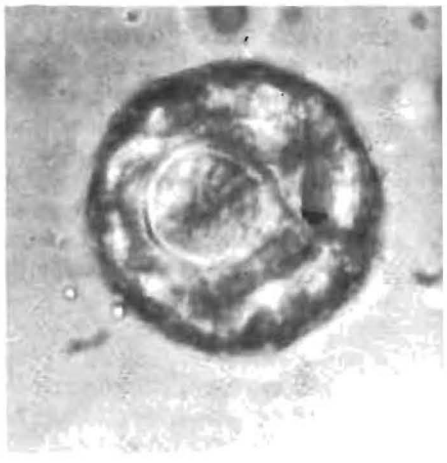

Fig. 6 Eggs of *Bertiella studeri* from baboon (approx. × 800).

Fig. 7 Egg of Oesophagostomum sp. from baboon (approx. × 500).

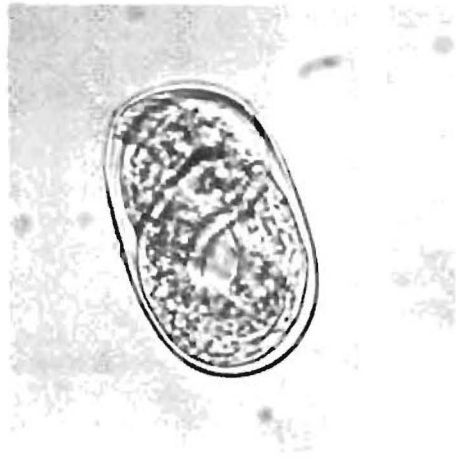

Fig. 8 Egg of *Strongyloides fulleborni* from baboon (approx. × 500).

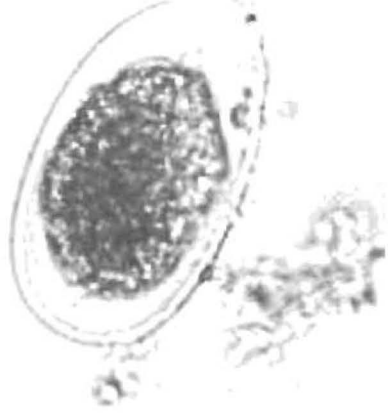

Fig. 9 Egg of *Trichostrongylus* sp. from baboo (approx. × 500).

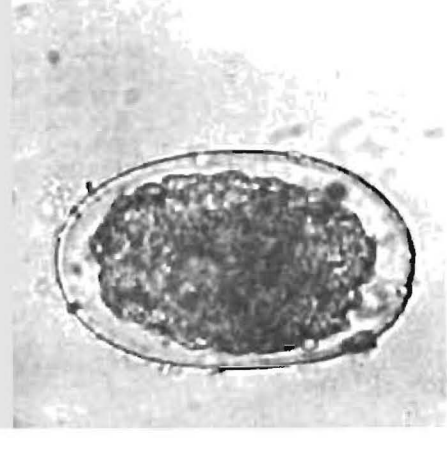

Fig. 10 Egg of *Ternidens deminutus* from baboon (approx. × 500).

Helminths

Eggs of *Abbreviata* (= Physaloptera) caucasica (Fig. 3) with a mean size of 57 μm × 25,6 μm; *Trichuris* sp *(?trichiura)* (Fig. 4) with a mean size of 62,5 μm × 3 μm; and *Streptophargus* sp. (Fig. 5), with a mean size c 42,5 μm × 25,6 μm were recovered from one anima each and one animal was found to be passing proglot tids and eggs of *Bertiella studeri* (Fig. 6). These latte eggs had a mean diameter of 46,6 μm and the embryo phores had a mean diameter of 19.1 μ.

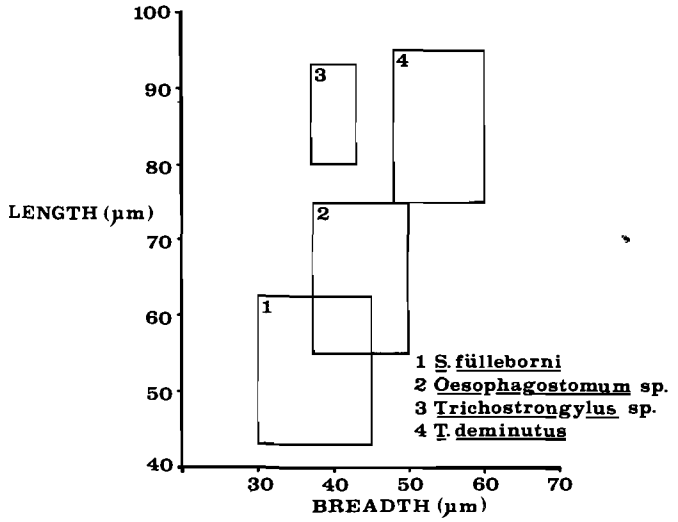

Fig. 11 "Block graph" to compare the measurements of the eggs of *S. fülleborni, Oesophagostomum* sp., *Trichostrongylus* sp. and *T. deminutus* from baboons.

Of the strongyle-like nematodes, 15 animals (62,5%) were found to be passing eggs of *Oesophagostomum* spp. (mean size 63,45 µm × 40,5 µm) (Fig. 7) and 9 animals 37,5%) were found to be passing small, fully-embryonated eggs of *Strongyloides fülleborni* (mean size 55,9 µm × 36,3 µm) (Fig. 8). From one animal, freeliving females of *Strongyloides* sp. were reared which proved to be identical to those of *Strongyloides stercoralis* of human origin in that they lacked the characteristic "waist" seen in *S. fülleborni*[9 16]. S. stercoralis has previously been recorded from baboons[14].

Eggs of *Trichostrongylus* sp. (Fig. 9) were recovered from two animals and one animal was found to be infected with *Ternidens deminutus* (Fig. 10).

The eggs of these strongyle nematodes can be identified on the basis of size and/or shape as can be seen from Figs. 7–10 and size as seen from Fig. 11.

Thus eggs of *S. fülleborni* are very small and are fully developed when passed. Eggs of all the other strongyle-like species are passed in the 8-cell to multi-cell stage of development and are nearly always larger than those of *S. fülleborni.* As well as differences in size between eggs of *Trichostrongylus* sp. and those of *T. deminutus* and *Oesophagostomum* sp. the eggs of the former species are more pointed at one or both ends, while those of the latter two species have blunt poles in most cases.

Fig. 11 has been constructed on the basis of measurements collected in the present series and on measurement given by Goldsmid[5 8]; Jessee *et al*[12]; Pampiglione and Ricciardi[16]. This graph is useful as, in addition to giving the range of egg sizes of the species concerned and their degree of overlap, it can be used directly to read off the species from the length and breadth of strongyle-like eggs found in baboon faeces in Southern Africa. Where size overlaps occur, shape and stage of development can help in the species-identification as explained previously.

In an autopsy on the animal infected with *T. deminutus,* worms were found in both the small and large intestine, a finding in agreement with that of McConnell *et al*[13] but at variance with most studies where the worms have usually been found in the large intestine only[7 15].

Our experience[10] has shown that mebendazole (Vermox) is a very effective broad-spectrum anthelmintic for intestinal nematodes, the pills being given embedded in bananas and at a dosage regimen of 100 mg b.d. for three days.

In general, the parasitological findings in the present series were similar to those of McConnell et al[13] from baboons from the same geographical area. Thus in both studies, infection with *Oesophagostomum* (82% and 62,5%) and *Balantidium* (52% and 54,2%) was common and infection with *T. deminutus* (3% and 4,2%) was uncommon. This is of interest when compared to the results of Goldsmid[9] on baboons from Rhodesia where infection with both *Oesophagostomum* and *T. deminutus* was extremely common.

These reports of the presence of *T. deminutus* in baboons in the Northern Transvaal are of considerable interest as this species can infect man and in the light of the frequency of human infection with *T. deminutus* in neighbouring Rhodesia[6 7]. In fact, in view of the possible zoonotic implications of infections with *A. caucasica, T. deminutus, Oesophagostomum* sp. and *S. fülleborni*[9], not to mention species such as *B. coli, T. trichiura* and *Bertiella studeri,* further parasitological investigations of the local African populations in this area of the Transvaal would be of great value.

The present survey is also of importance to veterinarians as a guide to the species of intestinal parasites and commensals commonly found in baboons in Southern Africa, especially in view of the increasing use of these animals in medical and veterinary schools and research laboratories.

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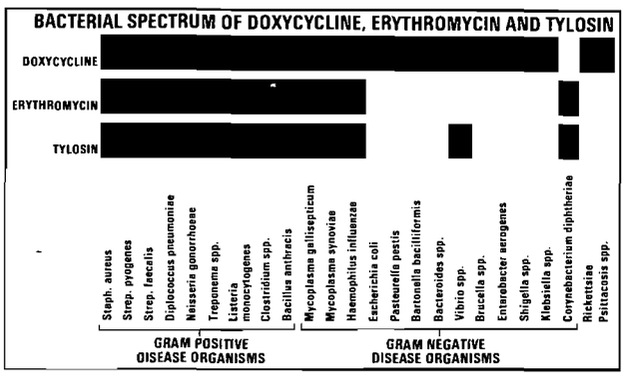

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THE MICROBIOLOGY OF POLONY

B.A. PRIOR and C. CASALEGGIO

ABSTRACT. Prior, B.A.,; Casaleggio, C. **The microbiology of polony,** *Journal South African Veterinary Association* (1978), **49** No. 2, 115–119 (En), Dept. of Microbiology, Univ. Orange Free State, Bloemfontein 9300, Rep. of South Africa.

A survey of 25 polony samples of different brands available to the consumer in Bloemfontein showed that significant numbers of various micro-organisms were present. Aerobic micro-organisms, psychrophiles, micrococci, pseudomonads, *Microbacterium thermosphactum,* lactobacilli, yeasts and moulds were enumerated by the plate count technique. The mean aerobic plate count was 15 850/g and counts ranged between less than 100/g and greater than 1 million/g. Selective plate counts indicated that psychrophiles, pseudomonads and lactobacilli were the main groups present. During storage at 5°C for 12 d, the mean pH of polony declined from 6,32 to 6,20 while the aerobic count increased 13 fold. Counts of all groups increased during storage at 5°C especially *M. thermosphactum,* yeasts and moulds. No salmonellae were found and counts of *Escherichia coli* or *Staphylococcus aureus* were less than 3/g.

Storage of 5 polony samples at 1,1° ± 1°C until they spoilt after 8 wks resulted in the aerobic count increasing to greater than 10^8/g. Lactobacilli and psychrophiles increased most rapidly while pH decreased from 6,20 to 5,57. Organisms isolated most frequently from spoilt polony were yeasts, micrococci, lactic acid bacteria and corynebacteria. It is concluded that standards for microbiological quality of polony are unnecessary.

INTRODUCTION

Polony consists of comminuted meat (protein and fat), prepared emulsions, cereals, flavouring substances and other additives. Polony is generally prepared by mixing with ice and emulsifying the ingredients at temperatures varying from −2°C to 8°C. Cooking time varies between 30 min (125 g pack) to 5 h (for bulk sizes) followed by pre-cooling at temperatures varying from 6 to 10°C and storage at temperatures from 3°C upwards before distribution[6]. The relevant regulations require a total meat content of not less than 75%[1].

No reports on the microbiology of polony in South Africa are known to exist. Studies on vacuum-packaged bologna however, a processed meat similar to polony, showed that lactic acid bacteria were the principle group of micro-organisms that multiplied during refrigerated storage. The pH dropped to less than pH 5 within 13 weeks and the amount of lactic acid increased to between 0,6 and 0,8%[14].

The type of spoilage occurring in processed meat is dependent on the physical and chemical conditions which prevail in the product. A slimy or sour type of spoilage results from the presence of lactic acid bacteria such as *Microbacterium thermosphactum* and yeasts in meats containing fermentable carbohydrate and of relatively low oxygen concentration[12].

This paper reports on the numbers and types of microorganisms present in polony and their growth during refrigerated storage.

MATERIALS AND METHODS

Sampling and shelf life tests

From May to November 1976, five samples each of five brands (total of 25 samples) of French polony were bought from different supermarkets in Bloemfontein and brought to the laboratory within 2 h. Each sample consisted of a single 500 g pack or two packs of 250 g. Half of the sample (250 g) was immediately examined and the remainder 12 d later after storage at 5°C.

In November, 1976 five 1-kg samples were bought for long term shelf-life trials, sealed in polythene plastic bags and stored at 1,1°C (standard deviation of 1,0°C; maximum temperature reached was 3,2°C). Periodically an aliquot of one hundred g of each sample was examined. At the time of purchase, all samples were judged to be in good condition.

Sample preparation

Samples of either 100 g- or 250 g-size were prepared for plate counting by the International Standards Organization (ISO) method[3]. The sample was twice minced through a sterile mincer. Ten g of the mince was weighed into a sterile blender (M.S.E. Atomix) and 90 ml of NaCl-peptone water (0,85% NaCl; 0,1% peptone) was added. The sample was homogenized at 6 000 rpm for 30 s and 12 000 rpm for 60 s. Decimal dilutions were made by transferring 10 ml to 90 ml of the NaCl-peptone water. The remainder of the minced sample was tested for the presence of salmonellae.

Isolation and counting of micro organisms

Plate counts were carried out by spreading 0,1 ml of the decimal dilution over the surface of the agar by means of a sterile glass rod. The aerobic count of micro organisms was determined on ISO Agar[3] after incubation at 30°C for 3 d. The number of psychrophilic micro-organisms was determined on ISO Agar after incubation at 5°C for 10 d. Pseudomonads were counted on the selective agar of Solberg *el al.*[20]. after incubation at 25°C for 3 d. Mannitol Salt Agar (Oxoid) was used to enumerate micrococci after incubation at 37°C for 3 d. Yeasts and moulds were enumerated on Potato Dextrose Agar (Merck) after incubation at 25°C for 5 d. Lactobacilli were enumerated on the selective medium of Rogosa *et al.*[17] after incubation at 25°C for 5 d. All inoculated plates were covered with a thin layer of the selective medium to create micro-aerophilic conditions for the growth of lactic acid bacteria.

The presence of salmonellae was determined by the method of Georgala and Boothroyd[9]. *S. aureus* were enumerated by the 3-tube Most Probable Number (MPN) technique of Giolitti and Cantoni[10]. *E. coli* were also enumerated by a MPN-technique. Nine millilitres of MacConkey Broth (Merck) was inoculated in triplicate with one ml of a specific dilution and incubated at 37°C for 48 h. Tubes positive for gas and acid were presumed to contain coliforms. One millilitre from each positive tube was added to 9 ml fresh MacConkey broth and 9 ml Tryptone water (Difco) and these tubes were incubated at 44°C for 48 h in a water bath. If acid, gas and indole were produced, the presence of *E. coli* was considered confirmed and the MPN/g of polony was calculated.

Physical tests

The pH of the polony was determined by placing the electrode of a Metrohm E 196 S pH meter into minced polony and measuring the pH. The hydration of the polony was determined by the extractrelease volume (ERV) method[11].

Identification procedures

Micro-organisms were isolated from the polony at the start and end of the storage trial. The isolates were repeatedly streaked out to ensure culture purity and held on slants of Stock Culture Agar (Difco).

The isolates were divided into groups based on their

Table 1: pH AND ERV[1] OF POLONY INITIALLY AND AFTER STORAGE AT 5°C FOR 12 D

		Brand					All
		1[3]	2	3	4	5	samples[4]
pH (Day 0)	Mean	6,44	6,44	6,14	6,34	6,22	6,32
	S.D.[2]	0,09	0,10	0,15	0,64	0,16	0,31
pH (Day 12)	Mean	6,03	6,37	6,11	6,31	6,16	6,20
	S.D.	0,41	0,11	0,21	0,68	0,09	0,37
ERV (Day 0)	Mean	56 ml	66 ml	47 ml	75 ml	54 ml	60 ml
	S.D.	16 ml	16 ml	21 ml	4 ml	20 ml	19 ml
ERV (Day 12)	Mean	56 ml	65 ml	46 ml	72 ml	53 ml	59 ml
	S.D.	26 ml	11 ml	22 ml	6 ml	22 ml	19 ml

1 Extract-release volume
2 Standard deviation
3 Mean of 5 samples
4 Mean of 25 samples

Table 2: LOG_{19} COUNTS/g OF MICRO-ORGANISMS IN POLONY ON DAY OF PURCHASE (D0) AND AFTER STORAGE AT 5°C FOR 12 D (D12)

		Brand					All
		1[2]	2	3	4	5	samples[3]
Aerobatic count	Mean	5,1	4,8	4,0	3,5	3,6	4,2
(Day 0)	S.D.[1]	1,9	1,9	1,7	1,1	1,3	1,6
Aerobic count	Mean	6,6	5,8	5,4	3,4	5,0	5,3
(Day 12)	S.D.	2,0	1,5	2,1	0,9	1,4	1,9
Psychrophiles	Mean	3,8	3,2	4,3	2,5	2,8	3,3
(Day 0)	S.D.	1,3	2,5	1,5	0,9	1,2	1,6
Psychrophiles	Mean	6,1	4,7	6,0	3,2	4,5	4,8
(Day 12)	S.D.	2,5	2,3	2,3	1,4	1,6	2,2
Pseudonomads	Mean	3,8	4,0	3,0	2,7	3,1	3,3
(Day 0)	S.D.	1,7	2,0	1,2	1,0	0,8	1,4
Pseudomonads	Mean	6,3	4,9	5,1	3,0	3,3	4,4
(Day 12)	S.D.	1,2	1,9	2,0	0,9	0,8	1,8
M. thermosphactum	Mean	2,5	3,4	2,6	2,5	2,0	2,6
(Day 0)	S.D.	1,1	2,8	1,4	1,1	0	1,4
M. thermosphactum	Mean	5,0	4,3	3,9	3,0	2,8	4,1
(Day 12)	S.D.	2,3	2,1	2,0	0,9	1,1	2,0
Micrococci	Mean	3,5	3,2	3,0	2,3	2,9	3,0
(Day 0)	S.D.	1,3	0,7	0,8	0,6	1,4	0,8
Micrococci	Mean	5,4	4,3	4,3	2,7	3,7	4,1
(Day 12)	S.D.	2,1	1,6	1,5	0,6	1,3	1,6
Yeaste and Moulds	Mean	2,9	3,0	2,7	2,1	2,6	2,6
(Day 0)	S.D.	1,4	0,7	1,6	0,2	1,0	1,1
Yeasts and Moulds	Mean	4,3	3,7	5,5	3,2	2,7	4,0
(Day 12)	S.D.	1,3	1,6	2,1	0,8	1,2	1,7
Lactobacilli	Mean	3,7	2,8	2,9	4,7	2,1	3,3
(Day 0)	S.D.	2,0	1,1	1,9	1,7	0,2	1,7
Lactobacilli	Mean	5,8	4,5	4,7	3,8	2,6	4,4
(Day 12)	S.D.	1,6	2,1	1,9	1,5	1,6	1,8

1 Standard deviation
2 Mean of 5 samples
3 Mean of 25 samples

Table 3: DISTRIBUTION OF COUNTS/g OF MICRO-ORGANISMS IN POLONY ON PURCHASE (D0) AND AFTER STORAGE AT 5°C FOR 12 D (D12)

Recovered on media selective for	No. of samples with microbial counts of							
	$<10^2$	10^2 to 10^3	$>10^3$ to 10^4	$>10^4$ to 10^5	$>10^5$ to 10^6	$>10^6$ to 10^7	$>10^7$ to 10^8	$>10^8$
Day 0								
Aerobic count	2	3	9	4	3	2	2	0
Psychrophiles	11	5	2	4	2	0	1	0
Pseudomonads	7	5	5	4	1	1	1	0
Microbacterium thermosphactum	22	0	0	1	1	0	1	0
Micrococci	5	9	8	3	0	0	0	0
Yeasta and moulds	13	6	3	1	2	0	0	0
Lactobacilli	15	2	1	3	3	1	0	0
Day 12								
Aerobic count	2	0	6	3	5	4	3	2
Psychrophiles	6	1	4	1	3	4	6	0
Pseudomonads	4	0	7	4	5	2	3	0
Microbacterium thermosphactum	7	2	6	2	2	3	3	0
Micrococci	2	6	7	2	5	2	1	0
Yeasts and moulds	6	4	5	4	6	0	0	0
Lactobacilli	5	2	5	1	6	4	2	0

Gram reaction, morphology and ability to produce catalase. Gram negative, catalase positive rods were divided into fermentative and non-fermentative groups and further classified into genera[5]. Published methods were used to classify lactic acid bacteria[18 21], *Bacillus* spp.[5], micrococci[2 4] and corynebacteria[4 5 13].

RESULTS

Survey of polony

No statistically significant variation in the pH between the various brands was found on Day 0 + 12 (Table 1). The mean pH of all samples dropped from 6,32 to 6,20 and while the mean pH of each brand decreased, this was not statistically significant ($P \leq 0,05$). The difference in ERV between brands was statistically significant ($P \leq 0,05$) but the ERV of each brand and the mean of all brands remained constant during storage (Table 1).

Table 2 shows the mean $\log_{10}$ counts of the five different brands of polony (n = 5) stored for 0 and 12 d at 5°C and the mean $\log_{10}$ count of all samples of polony (n = 25). The mean aerobic count of all samples increased 12 fold from $\log_{10}$ 4,2 to $\log_{10}$ 5,3 during storage. The psychrophilic count (Table 2) showed a 32 fold increase during storage.

The distribution of counts at 0 and 12 d for total aerobic count and selective plate counts are shown in Table 3. At time of purchase, 72% of the samples (n = 25) had a count of less than 100 000/g while none of the samples had a count greater than 100 million/g. After storage at 5°C for 12 d, the percentage of samples having counts of less than 100 000/g was reduced to 44 while 8% of the samples had a count greater than 100 million/g. These latter samples were judged to be watery but no off-odours were noticed. During storage, the selective plate counts of the other groups of micro-organisms increased (Table 3). This was especially noticeable in the distribution of counts for *M. thermosphactum* and psychrophiles.

The pH-values of the samples decreased during storage (Table 4). Whereas only 4% of the samples had a pH less than 6 at time of purchase, these had increased to 16% during storage. The decrease in pH possibly resulted from acid production by microbial contaminants.

Table 4: DISTRIBUTION OF pH-VALUES OF POLONY ON PURCHASE (D0) AND AFTER STORAGE AT 5°C FOR 12 D (D12)

	No. of samples with pH values of						
	<5,6	5,6 to 5,8	>5,8 to 6,0	>6,0 to 6,2	>6,2 to 6,4	>6,4 to 6,6	>6,6
D0	1	0	0	7	6	8	3
D12	1	2	1	9	6	4	2

Salmonella spp. were absent in all samples. Counts of *S. aureus* and *E. coli* were less than 3/g in all samples.

Extended shelf-life studies

During storage at 1,1° ± 1°C for 8 weeks, there was a 300 fold increase in the total aerobic count from $\log_{10}$ 5,75 (S.D. = 1,4; n = 5) to $\log_{10}$ 8,24 (S.D. = 0,19; n = 5) (Fig. 1). At 8 weeks all the samples were judged to be of watery consistency and have sour off-odours. During storage, the pH declined steadily from pH 6,20 to 5,57.

All counts of micro organisms on selective media, except those of micrococci, rose during storage (Fig. 2). Counts of psychrophiles and lactobacilli increased most rapidly (> 10 000 fold), pseudomonads rose a 100 fold, yeast and moulds, 500 fold and *M. thermosphactum*, 170 fold. This order of groups growing most rapidly varied from that of the shorter 12 d storage trial at 5°C. However, Fig. 2 shows that lactobacilli and psychrophiles increased most rapidly between 2 and 6 weeks. The groups of organisms isolated from the aerobic count plates are shown in Fig. 3. During storage, the proportions of Gram negative rods and *Bacillus* sp. decreased while the proportion of yeasts and moulds increased.

Table 5: FREQUENCY AND IDENTITY OF MICROBIAL FLORA ISOLATED FROM POLONY

Organism	No. of Isolates
Gram positive bacteria:	
Bacillus polymyxa	1
Bacillus cereus	7
Bacillus megaterium	5
Baciolus coagulans	1
Micrococcus luteus	11
Staphylococcus epidermidis	2
Staphylococcus sp.	1
Streptococcus faecium	8
Streptococcus faecalis	2
Streptococcus durans	4
Aerococcus viridans	8
Microbacterium thermosphactum	7
Unidentified catalase positive, non-sporeforming rods	16
Gram negative bacteria:	
Acinetobacter calcoaceticus	7
Pseudomonas sp.	1
Yeasts	14
Dead or unidentified	5
Total	100

Table 5 identifies the organisms isolated from the aerobic count plates. Most frequently isolated genera were *Bacillus, Streptococcus* and *Micrococcus* and unidentified Gram positive rods Samples of slime taken from spoilt polony after storage yielded *Bacillus cereus, Micrococcus luteus,* a yeast and two isolates of *M. thermosphactum.*

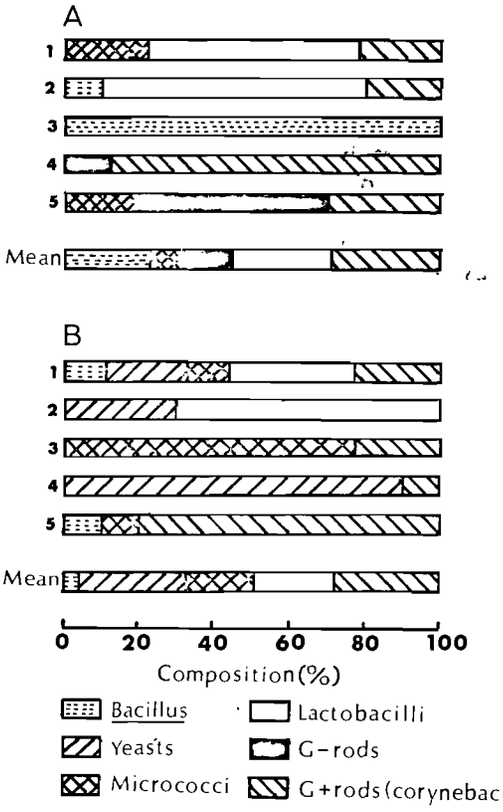

Fig. 3 The dominant contaminants of polony. Each numbered bar represents the results from one sample of one brand of polony. The A group of bars are the results from initial isolations on purchase. The B group of bars are the results from isolations after storage at 1,1°C for 8 weeks. Mean bars give average results for the 5 polony brands before and after storage.

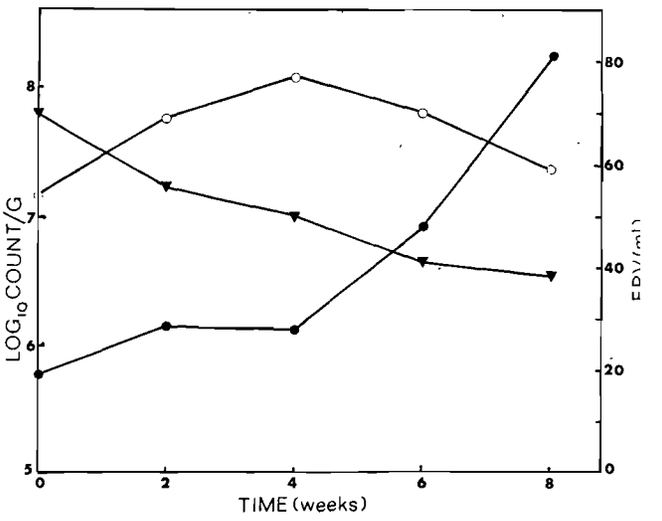

Fig. 1 Changes in total aerobe count, extract release volume (ERV) and pH during storage of polony at 1,1°C.
●, $\log_{10}$ count/g; ○, ERV; ▽, pH.

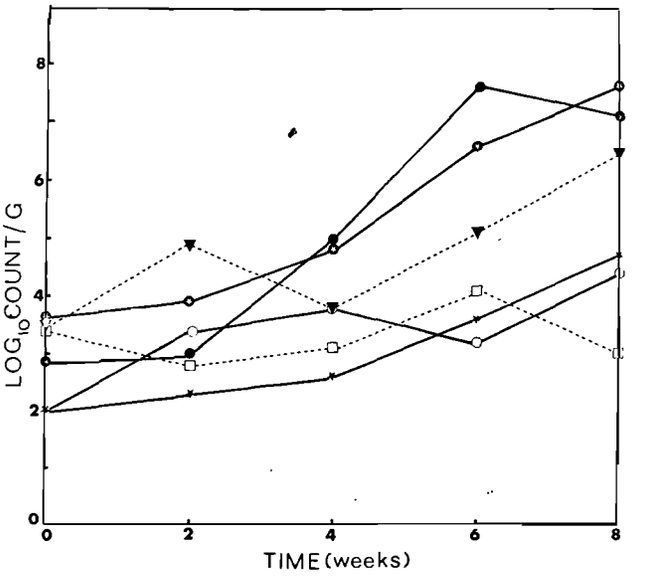

Fig. 2 Changes in counts of micro-organisms in polony during storage at 1,1°C.
●, psychrophiles; ●, lactobacilli; ▽, pseudomonads; ★, yeasts and moulds; ○, *M. thermosphactum;* ☆, micrococci.

DISCUSSION

This survey shows that polony available to the consumer contains significant numbers of various micro-organisms and these organisms find polony to be a suitable environment for growth. A comparison of the aerobic and psychrophilic count (Table 2) suggest that a significant proportion of the flora is psychrophilic and this proportion increased during refrigerated storage. Increases in the mean counts of all samples of pseudomonads, *M. thermosphactum, micrococci, yeasts and moulds and lactobacilli varied between 12 and 32 fold during storage while the increase in the count of M. thermosphactum* ($P \leq 0,05$) and yeasts and moulds ($P \leq 0,05$) was most significant. Selective plate counts indicate that pseudomonads and lactobacilli are the largest groups of micro-organisms to be found in polony.

Differences in the aerobic counts (Table 2) between the five brands were statistically significant ($P \leq 0,05$)

with Brand 1 showing the highest count and Brand 4, the lowest. Signifi cant differences ($P \leq 0,05$) between brands in the counts of pseudomonads and micrococci were also found.

Spoilage of polony during refrigeration was characterized by a sour odour, watery consistency, reduction of pH and aerobic counts of greater than 100 million/g (Fig. 1). The reduction of the pH suggests that the spoilage organisms are able to metabolize the carbohydrates to acid. The ERV showed no definite trend. Some workers[12] [19] have successfully used this test as a means of judging progress of spoilage in meats but, in this instance, it appeared to be unreliable.

The wide variation in the number and types of micro-organisms present in polonies bought at supermarkets (Table 3) suggest that:

(a) the heat treatment varies between different batches and brands of polony and/or
(b) the original microbial quality of the meat and other ingredients used for polony manufacture differs and/or
(c) the time between manufacture and purchase by the consumer varies widely and/or
(d) the temperature during storage, distribution and retailing could have been considerably higher than optimal.

These factors require further investigation. Heating during the preparation of polony would be selective for the survival of endospore-forming bacilli, however these studies show that these organisms are not the dominant micro-organisms present at the time of purchase by the consumer. As contamination of polony after heating is probably minimal, the principle groups of organisms must have survived the heating process.

The absence of *Salmonella* spp. and counts of *E. coli* and *S. aureus* less than 3/g from the meat suggest that these organisms were destroyed during heat processing or were not initially present in the meat. The second possibility is unlikely as previous studies on raw meats show *E. coli* and *Salmonella* to be frequently present[15] [16]. *E. coli* is often used as an indicator of the hygienic quality of foods. However, the absence of *E. coli* in polony while other organisms of possible faecal origin such as streptococci (Table 5) are present, suggest that *E. coli* may be unsuitable as an indicator of faecal contamination.

In recent years, the use of standards to control microbial contamination in meats have been enacted in various parts of the United States[7]. In South Africa, such standards have been introduced for marine foods[1]. Partly cooked marine food such as various shellfish and fish are required to have less than one million organisms/g. Application of such a standard to polony after 12 d storage would result in 16% failing to comply. The relatively long period of time during which polony may be stored under refrigeration before becoming spoilt, the absence of Salmonellae, small number of *E. coli* and *S. aureus* encountered in this survey and the lack of any reported cases of foodborne sickness after consumption of polony in South Africa suggest that standards for polony are unnecessary.

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ULTRASTRUCTURE OF LUTEOLYSIS INDUCTION BY PROSTAGLANDIN F_2-ALPHA IN THE NON-PREGNANT EWE

L.A. VAN DER WALT

ABSTRACT: Van der Walt L.A. **Ultrastructure of luteolysis induction by prostaglandin F_2-alpha in the non-pregnant ewe.** *Journal of the South African Veterinary Association.* (1978) **49** No. 2, 121–123 (En) Dept. Chem. Path., SAIMR and Univ. Witwatersrand, Box 1038, Johannesburg 2000, Rep. of South Africa.

Ultrastructural morphology was monitored at intervals of time in corpora lutea of superovulated ewes following treatment with Prostaglandin F_2-alpha. Lutein cells displayed an initial increased metabolic rate as marked by lipid accumulation, spherical mitochondria and a high respiratory rate. Three hours following injection, complete disorganisation of the cell was apparent; mitochondria are ruptured and a clear state of autolysis exists. The possible mode of luteolytic action of Prostaglandin F_2alpha in the ewe is discussed.

INTRODUCTION

Long an enigma to the chemist, prostaglandins have more recently found utility to the community of physiologists, gynaecologists, immunologists and biochemists. Interest in the role of these unique lipids in female reproductive physiology flared anew upon the discovery of their pregnancy-terminating effect. Prostaglandin F_2-alpha (PGF) has been shown to have luteolytic activity in the human[3] and in several animal species, including the rat[7], hamster[2], rabbit[1], monkey[4] and sheep[5]. Studies pertaining to the luteolytic effect of PGF have concentrated mainly on biological techniques rather than on detailed morphological studies. Luteal disorganization following PGF injection in the pregnant hamster has been reported in a microscopic study[2]. Subsequently, an ultrastructural study was conducted on the effect of PGF on the corpus luteum of the pregnant rat[6]. These authors demonstrated clear luteolysis and were responsible for raising some interesting points on the nature of PGF-induced luteolysis. Our observations have led us to investigate the induction of luteolysis in the ewe employing electron microscopy but concentrating upon the primary ultrastructural effects of PGF on the non-pregnant corpus luteum.

MATERIALS AND METHODS

Mature, cycling ewes were employed as experimental animals. They were maintained on an *ad libitum* feeding regime in outdoor pens. Prior to surgery, individuals were isolated in metabolism cages for 24 h and deprived of food and water during this time.

Oestrous synchronization and superovulation was accomplished using progesterone-impregnated Silastic® implants; these were inserted and left *in situ* for 14 d. On removal, a single dose of 5 000 IU pregnant mare serum was administered subcutaneously. Twenty-four h later, 20 μ of oestradiol-17β was injected intramuscularly. Progesterone assays confirmed the presence of viable corpora lutea, indicative of satisfactory ovulation.

The first day of oestrous was designated as day 0. On day 10, the animals were anaesthetized with sodium pentothal and maintained on fluothane and bitrous oxide. Only those animals having a minimum of four corpora lutea on either or both ovaries were utilized for experimentation.

One corpus luteum was initially removed, quartered and immediately weighed on an analytical balance. One quarter of each corpus luteum was dissected into squares of ca. 1 mm and fixed in 4% gluteraldehyde for electron microscopic evaluation. All other tissue was plunged into liquid nitrogen and stored until further processing. The dorsal aorta was then occluded caudal to the ovarian blood supply and 300 μ of PGF, dissolved in 1 ml of sterile physiological saline carrier, was injected into the dorsal aorta over a period of 20 s. The occluding clamp on the dorsal aorta was retained in place for 1 m following injection. The initial corpus luteum was designated as the control sample (C), while those corpora lutea removed after injection represented 1, 2 and 3 following PGF administration.

Control animals received a single injection of physiological sterile saline, the mode of administration and collection of corpora lutea being identical to the experimental animals. Corpora lutea from these animals served as controls to examine the influence of long-term anaesthesia and surgical procedures. All animals were ovariectomized following procurement of luteal tissue.

Tissue for electron microscopic evaluation was fixed in 4% gluteraldehyde in 0,075 M Millonig-phosphate buffer (pH7,2) at 4°C for 36 g. Post-fixation was carried out using 1% osmium tetroxide in the above buffer at room temperature for 2 h. Luteal tissue was then dehydrated in a series of graded alcohols. Propylene oxide was used as the transition solvent and sections were allowed to stand for 1 h in an epoxy resin: propylene oxide (1:1) mixture, then in a more concentrated resin. Capsulated sections were polymerized in an oven at 50°C for 48 h. Sections were cut at 500A and mounted on 200-mesh grids with parlodiumcarbon as the supporting film. Staining was accomplished using 2% uranyl acetate followed by immersion in lead citrate for 8 m. Samples were examined using an RCA EMU-3G electron microscope at 50 KV.

Biochemical analyses were carried out on the remainder of the luteal tissue. These results will be dealt with in a subsequent publication. In all, six animals were used in each of the experimental and the control groups.

RESULTS AND DISCUSSION

Figure 1 illustrates an electron micrograph of a control corpus luteum prior to PGF treatment. In this tissue, a minimal amount of lipid material is present while distinct microvillous protrusions of the membrane into the perivascular area may be observed. A few small vesicles

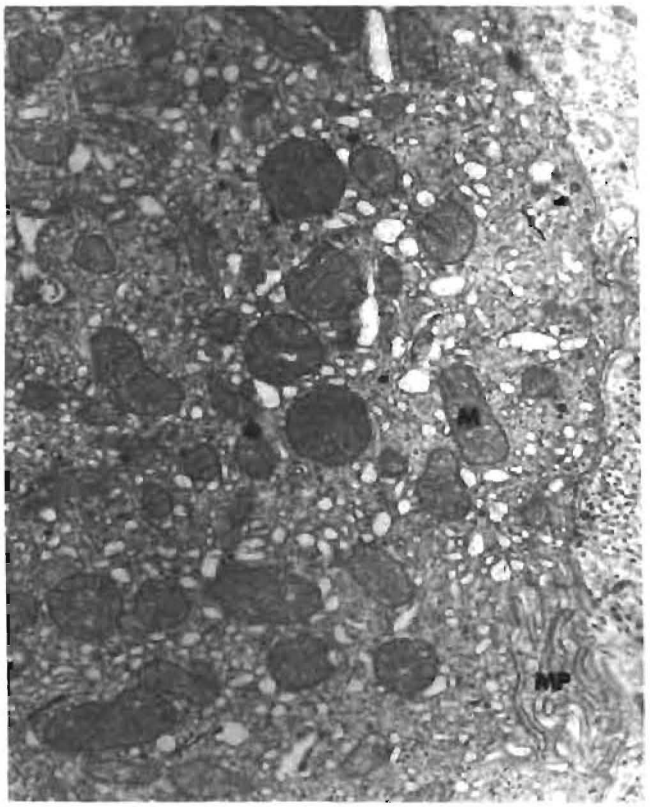

Figure 1. Electron micrograph of a control corpus luteum prior to PGF treatment. Mitochondria (M) display tubular cristae and elongated structure. Minimal lipid material (L) is present while microvillous protrusions (MP) are evident.
(x 13500)

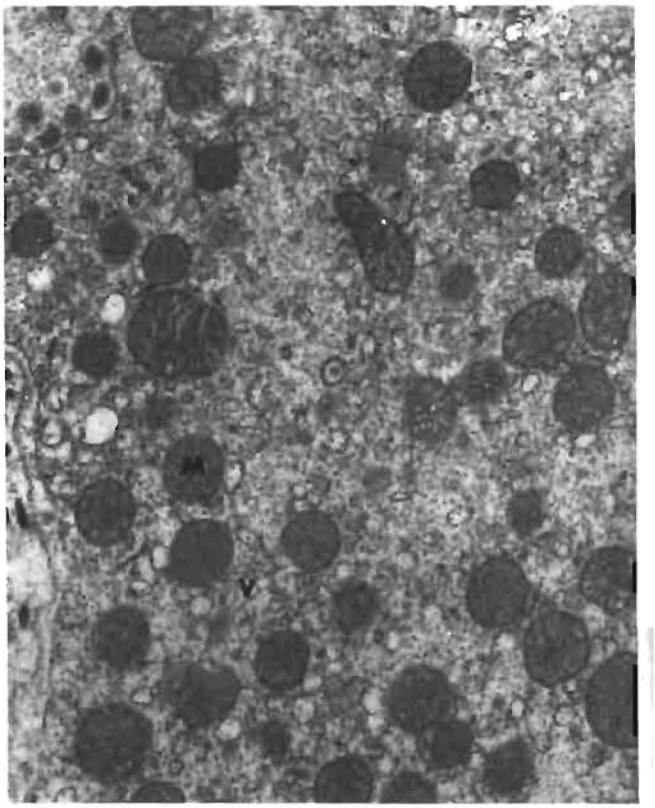

Figure 2. Electron micrograph of luteal tissue one h following PGF administration. Mitochondria (M) are more spherical in character while the field contains a large number of small vesicles (V).
(x 13500)

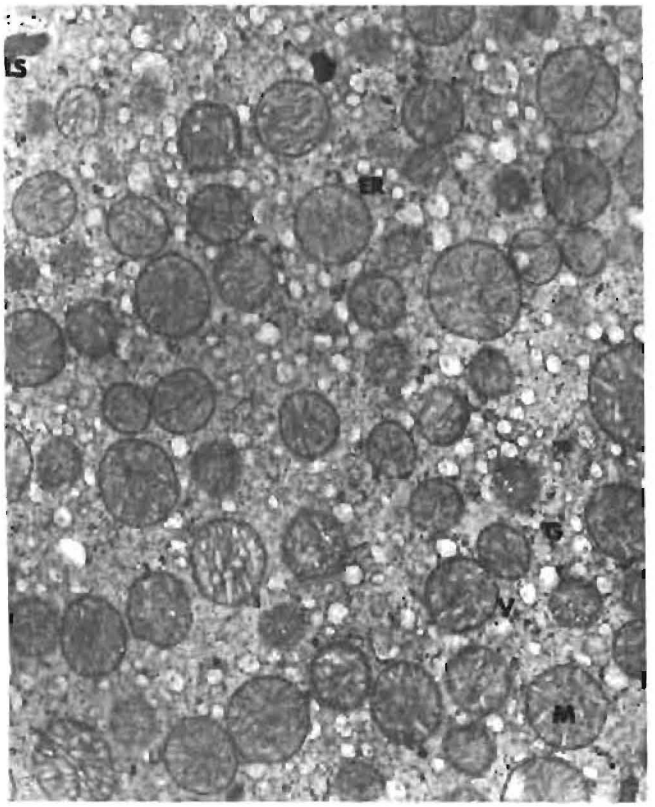

Figure 3. Electron micrograph of luteal tissue 2 h following PGF treatment. Mitochondria (M) are mostly spherical in shape with dense cristae matrix and numerous glycogen granules (G) are evident. Vesicles (V) are swollen and endoplasmic reticulum (ER) is smooth in character. The initiation of a lysosome (LS) may be noticed. This tissue is highly metabolic.
(x 13500)

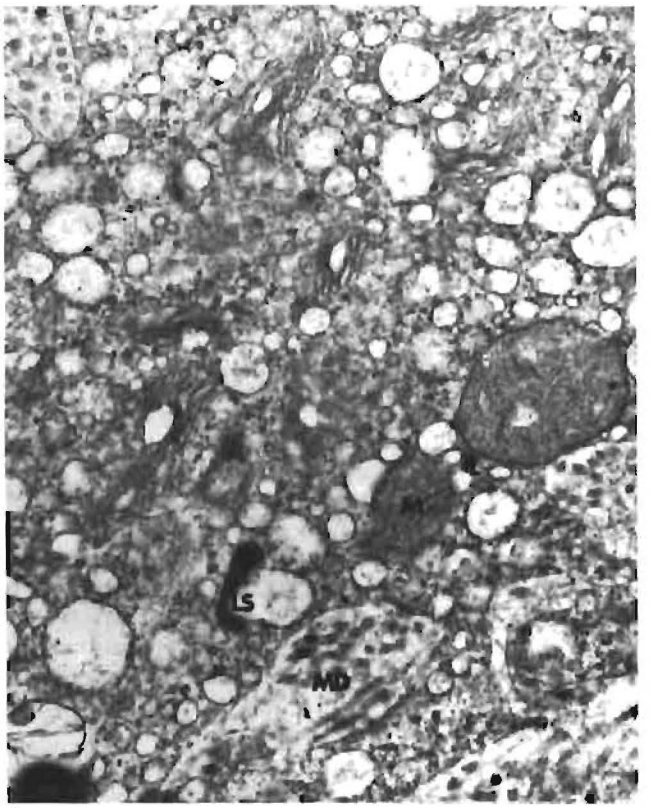

Figure 4. Electron micrograph of luteal tissue 3 h following PGF treatment. Mitochondria (M) are ruptured and attenuated. An increase in lipid droplets (L) and lysosomal bodies (LS) indicate an advanced state of autolysis. Membraneous debri (MD) is present. The tissue appears generally unhealthy and degenerative.
(x 13500)

are present, while the mitochondria appear healthy and display elongated structure. Few golgi apparatus are observed but those present appear well developed. The pronounced vesiculated appearance of the cytoplasm may be the result of dilation and distension of the tubular, agranular endoplasmic reticulum. The cristae of the mitochondria appear tubular, indicative of an active metabolism.

Figure 2 is representative of tissue collected 1 h post-PGF administration. At first scrutiny, this tissue appears to be normal, healthy and highly active. The mitochondria are, however, of more spherical character, while this stage also appears to contain a larger amount of smaller vesicles than evident in the control tissue. Tissue obtained 2 h post-injection is characteristic of an extremely elevated metabolism (Figure 3). This stage has a high respiratory rate, appears to contain a high ADP and substrate content and shows a marked increase in numbers of glycogen granules. The mitochondria are now mostly spherical in character and possess dense cristae matrices. Vesicles are more numerous and appear swollen, indicating greater vesicular volume.

More lipid droplets make their appearance while few golgi apparatus are apparent at this stage. No microvillous protrusions could be observed. The endoplasmic reticulum is smooth in character and the appearance of free ribosomes can be demonstrated. The initiation of a lysosome is evident in this figure.

Figure 4 shows the state of the corpus luteum 3 h following PGF administration. There appears to be a reduction in the cytoplasmic volume and disorganization of the agranular endoplasmic reticulum. Membraneous debris is present in the extracellular space. The character of the cells is not that of healthy, active metobolic tissue, but represents a state of utter chaos. Mitochondria, when not ruptured, are frequently attenuated and often enveloped translucent areas. There is an increase in lipid droplets and the appearance of lysosomal bodies indicate an advanced state of autolysis. Pycnotic nuclei and distorted cristae both indicate an advanced stage of degeneration.

Control animals showed no ultrastructural changes throughout treatment. All corpora lutea examined at all intervals displayed no degenerative change and one could safely contend that the trauma of anaesthesia and surgery was not responsible for corpus luteum regression. All experimental animals regressed as hallmarker by the ultrastructural data as described above. From these experiments it would seem that PGF does indeed cause luteolysis when administered at the site of action and at the dosage employed. It would appear that the effect of PGF may be more rapid in action than was previously assumed. A single dose at site initiates luteolysis that may be ultrastructurally apparent at 2 to 3 h following administration.

Regression of the corpus luteum in natural circumstances is typified by a decrease in progesterone secretion. This decrease has been well documented. A sharp, rather dramatic progesterone peak following PGF treatment has been reported[2] but no satisfactory explanation thereto offered. This surge has been rather vaguely attributed to a systemic effect of PGF on the pituitary, but has most often been speculatively glossed over as artefactual. This study substantiates these data and it may be assumed that the lutein cells of the ewe, upon PGF exposure, may initiate a "recovery". Similar suggestions have been made by several authors on other species[2 6]. The mechanism by which PGF actually induces luteolysis is unclear. Since the initial effect of PGF upon regression may be reversed by the concurrent administration of progesterone, it seems as if an inherent local recovery mechanism may accomplish a similar effect. This protective phenomenon may then buffer small changes in local PGF release but is limited to small PGF changes. Once this buffer effect has been overcome, regression is rapid and irreversible. We are confident that the corpus luteum does indeed attempt a recovery as evidenced by the increased metabolic rate following PGF treatment and prior to gross degenerative changes.

Preliminary biochemical data substantiates this hypothesis. A distinct increase in progesterone secretion does occur prior to luteal demise. Chemically then, as well as ultrastructurally, the corpus luteum of the ewe literally falls apart 3 h following PGF administration. One is hesitant, however, to endeavour to correlate this chain of events to that of natural luteolysis employing this experimental design.

ACKNOWLEDGEMENTS

The author wishes to extend appreciation to Upjohn Company for supplying the PGF utilized in this study.

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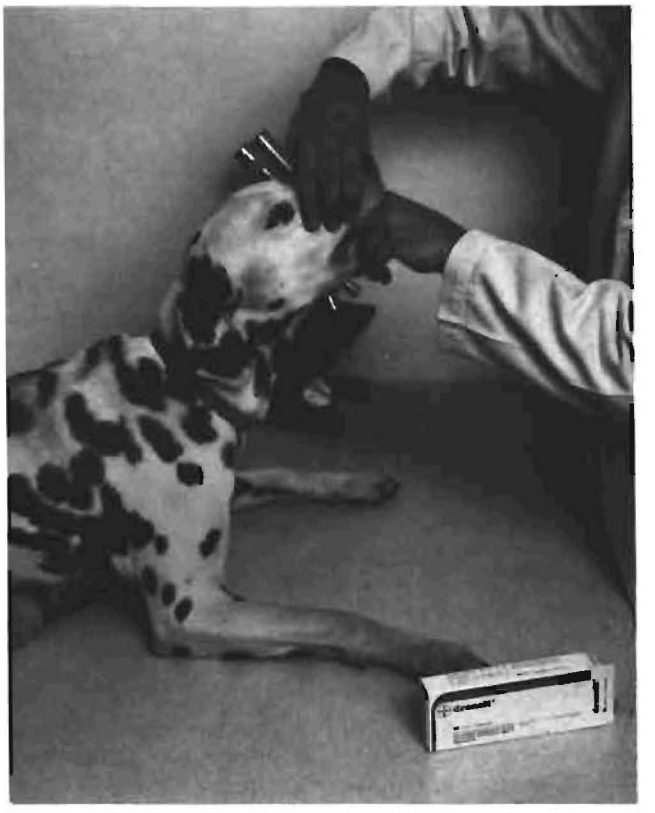

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NITROXYNIL*
ANTHELMINTIC ACTIVITY IN CATTLE FOLLOWING SUBCUTANEOUS INJECTION

A.C. WELLINGTON

ABSTRACT: Wellington A.C. **Nitroxynil: Anthelmintic activity in cattle following subcutaneous injection.** *Journal of the South African Veterinary Association* (1978) **49** No. 2, 125–126, (En), Health Research Station, P.O. Box 1130, 6000 Port Elizabeth, Rep. of South Africa.

Nitroxynil injected subcutaneously at 10 mg/kg live mass achieved Class A efficacy when evaluated by the non parametric method against adult *Fasciola gigantica, Haemonchus placei, Bonustomum phlebotomum* and *Oesophagostomum radiatum* in cattle. The compound was not effective against adult *Cooperia spp.* at the same dosage.

INTRODUCTION

Nitroxynil (4-cyano-2-iodo-6-nitrophenol) has been used in the treatment of fascioliasis in cattle and sheep since 1966.

Davis, Lucas, Rosenbaum and Wright showed nitroxynil to be active against *Fasciola hepatica* in sheep and calves at levels of 5–20 mg/kg live mass and Roy and Reddy[9] showed it to be effective against *Fasciola gigantica* in cattle, buffaloes and sheep at 10 mg/kg. Guilhon and his co-workers[3] showed it to be active against *Haemonchus contortus, Bunostomum phlebotomum* and *Oesophagostomum radiatum* at levels of 10–20 mg/kg, and Lucas[5] found nitroxynil at 10 mg/kg to be effective against all stages of *H. contortus* in sheep and cattle.

The present paper presents the results of a trial using the non-parametric method (NPM)[2] to evaluate the efficacy of nitroxynil against the adult stages of *F. gigantica, Haemonchus placei, B. phlebotomum, Cooperia spp.* and *O. radiatum* in artificially infested cattle.

MATERIALS AND METHODS

Twenty-two, three-month-old Afrikaner calves were acquired and brought on to the Terenure Research Station, where they were treated with levamisole at 10 mg/kg live mass *per os,* and five days later with parbendazole at 60 mg/kg live mass *per os.* The calves were housed on expanded metal grids before and during the trial and received a ration of hammer-milled lucerne.

The viability of the metacercariae of *F. gigantica* was evaluated microscopically – by movement of the caeca under pressure[4]. Each of the calves was dosed *per os* with 300 metacercariae on day −98. The calves were infected per-cutaneously with infective larvae of *B. phlebotomum* and then orally with infective larvae of *H. placei, O radiatum* and *Cooperia spp.* in the numbers and at the times given in the experimental design summarized in Table 1.

On day zero, nitroxynil, as the commercially available 34% m/v solution, was injected subcutaneously into each of 12 calves at a dose rate of 10 mg/kg live mass. Twenty-two days after treatment slaughter of these animals commenced at the rate of three animals per day.

The abomasal ingesta were heated to 60°C, formalnised and sieved through a sieve, with 150μm apartures the rasidue being collected and bottled for later examination. Worms were recovered from the intestinal ingesta by the methods described by Reinecke[6 7 8]. In all cases 28 gauge bolting cloth (apertures of 750 μm) was used rather than the fine mesh as advocated by Reinecke[6 7 8].

Small bowel scissors were used to open the bile ducts and the liver flukes were washed into a tray. The liver was sliced transversely into 5 mm thick slices and the flukes were expressed from the cut surface by pressure. The slices were then placed on expanded metal mesh in trays containing normal saline in a waterbath at 40°C for three hours. Flukes were again expressed and the liver was then minced, bottled, formalinised and subsequently examined for any remaining flukes.

One-tenth aliquots of the ingesta collected were examined microscopically to ascertain whether any larval stages were present. This having proved negative, total macroscopic counts were carried out. The digested abomasal and intestinal walls were examined microscopically with the aid of a stereoscopic-microscope and total worm counts were carried out.

Counts of *O. radiatum* below the reduced median in the treated animals and those on either side of the median in the control animals were recounted to check the validity of the original counts.

Table 1: EXPERIMENTAL DESIGN

Day	No. of metacercariae or infective larvae administered to each animal and other actions
−98	300 metacercariae of *F. gigantica*
−47	3 216 larvae of *B. phlebotomum* per cutaneously
−40 to −21	A total of 2 630 larvae of *O. radiatum* was divided and dosed daily *per os*
−30 to −15	A total of 5 027 larvae of *H. placei* was divided and dosed dialy *per os*
−20 to −9	A total of 6 052 larvae of *Cooperia* spp. was divided and dosed daily *per os*
0	12 calves were treated with nitroxynil administered subcutaneously at 10 mg/kg live mass
+22 to +31	The 22 calves in the trail were slaughtered and processed for helminth recovery.

RESULTS

The ranked worm burdens of the treated and control calves, the mean percentage reduction of worms in the treated calves and the efficacy rating according to the non-parametric method of evaluation are presented in Table 2.

*'TRODAX' 34% m/v Maybaker (S.A.) (Pty) Ltd., Port Elizabeth.

Table 2: THE EFFICACY OF INJECTABLE NITROXYNIL AGAINST VARIOUS HELMINTHS IN CATTLE

	Numbers of worms recovered				
Group	*F. gigantica*	*H. placei*	*Cooperia* spp.	*B. phlebotomum*	*O. radiatum*
Control	73	97	15	29	100
	76	347	71	42	147
	90	462	81	46	185
	117	717	856	49	240
	118	965	1 075	53	244*
	124	1 226	1 185	75	308*
	147	1 250	1 527	172	318
	153	1 642	1 570	205	462
	165	1 729	2 121	209	488
	173	1 965	2 283	242	560
Group mean	123,6	1 040	1 078,4	112,2	305,2
Treated	0	0	0	0	0
	0	0	8	0	10
	1	0	851	0	11
	1	0	878	0	14
	2	0	1 005	0	22
	3	0	1 168	0	28
	4	0	1 488	0	30
	5	1	1 548	0	38
	5	1	1 699	0	45*
	6	2	1 837	0	53*
	9	6	1 901	0	65*
	12	12	2 109	1	107
Group mean	4	1,8	1 207,7	0,08	35,3
Group mean reduction	96,8%	99,8%	−12%	99,9%	88,4%
Control median	121	1 095,5	1 140	64	276
NPM Efficacy rating	A	A	X	A	A

*Recounted.

CONCLUSION

Using the non-parametric method of evaluation, nitroxynil injected subcutaneously at a dose rate of 10 mg/kg live mass achieved Class A efficacy against adult *F. gigantica, H. placei, B. phlebotomum* and *O. radiatum*. The compound was not effective against adult *Cooperia* spp. at the same dosage.

ACKNOWLEDGEMENTS

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HEAT STRESS MORTALITY IN NICARBAZINE FED CHICKENS

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ABSTRACT: Buys S.B.; Rasmussen R.W. **Heat stress mortality in nicarbazine fed chickens.** *Journal of the South African Veterinary Association* (1978) **49** No. 2, 127–128 (En). Veterinary Research Institute, 0110 Onderstepoort Rep. South Africa.

Significantly higher mortalities were found in nicarbazine fed chickens than in amprolium fed chickens when exposed to the same heat stress conditions.

INTRODUCTION

In order to prevent the development of drug resistance of Coccidia to coccidiostats, various drugs may be used in rotation during the growth cycle of broilers. One such drug is nicarbazine* which is used in chickens from day-old to five weeks of age. However, noteworthy side-effects were reported by Sammelwitz[3] [4] who during studies on the effect of heat stress on broilers, observed increased mortality in chickens fed normal levels of nicarbazine when they were exposed to higher, temperatures.

Circumstantial evidence that this phenomenon is a problem in South Africa was recently obtained from a farm in the Western Province where high mortality was observed in certain broiler houses during a heat wave. An investigation revealed that the temperature inside the houses rose to a maximum of 40°C thereby creating conditions where deaths from heat stress could be expected. The mortality rate, however, was higher among chickens fed a ration supplemented with nicarbazine (125 ppm) than among those which received other coccidiostats. It was decided therefore to determine whether the phenomenon of increased susceptibility to heat stress in broilers fed nicarbazine could be repeated experimentally.

MATERIALS AND METHODS

Unsexed CG X WR chickens, raised on a commercial starter ration (22% protein), were used in all these trials. The food of half the birds was supplemented with nicarbazine at 125 ppm, and that of the other half with amprolium plus ethopabate** at 125 ppm. These chickens were housed on wood shavings in pens allowing 0,055 m² per chicken. Food was available *ad libitum* and lights were left on for 24 h a day.

For the purpose of the experiment the chickens fed on the 2 different rations were divided at random and by age into groups of 20. Age groups of 1, 2, 3, 4 and 5 weeks were represented, giving a total number of 10 groups, half of which received nicarbazine and the other half amprolium plus ethopabate in their feed.

Ten pens with a surface area of 1 m² each were constructed with wire mesh floors. They were stacked in one separate room and each pen was equipped with a maximum-minimum thermometer. The temperature in this room was regulated by a blower heater capable of minimizing variation to ± 0,5°C. The 10 groups of chickens were randomly distributed into these pens.

The experiment lasted 6 hours. In Trials 1 and 2 the chickens were kept for 6 hours at a temparature of 40° C, and in Trial 3, for 6 hours at 36°C. In Trial 1 a water-trough was installed in each pen; no water-troughs were supplied in Trials 2 and 3. Furthermore in Trial 1 the wire mesh floor was covered with brown paper to stop droppings from one pen contaminating to one beneath. This procedure was abondoned because the paper became wet and the birds layed down on the wet paper to cool themselves. Consequently in Trial 2 and 3 the brown paper cover was omitted.

*Nicrazin – MSD (Pty) Ltd., P/Bag X3, Halfway House 1685.
**Amprol plus – MSD (Pty) Ltd., P/Bag X3, Halfway House 1685.

RESULTS

The results are presented in Table 1.

Mortalities were found only in Trial 2 in which the highest mortalities occurred in the nicarbazine fed groups.

At the 95% confidence level, the higher mortality in the nicarbazine fed groups compared with that in the amprolium group is highly significant in the 5-, 4-, 3- and 1-week groups, and significant in the 2-week group.

The birds in all the trials suffered from increased respiration and a watery excretion.

DISCUSSION

Birds can regulate their temperature by means of convention, radiation and evaporation. It was found that at an air temperature of 35°C evaporative heat loss may account for almost all of the heat loss[1].

It would thus appear that the birds cooled themselves with the water in the troughs in Trial 1, and the same effect was also achieved when the brown paper became wet with the watery excretion and thus no mortality was found in Trial 1.

In birds kept at a room temperature of 20°C the water intake was about 1,75 times the food consumption, whereas at 34°C there was a 4-fold increase[2]. Although these workers did not mention this development, one would expect a higher water excretion with such a high intake of water. In our experiments a higher water excretion was found in all three trials. Unless birds were dehydrating themselves, it is hard to explain why the birds should have a watery excretion right to the end of the 6-hour period during which no water was available to them (see Trials 2 and 3).

SUMMARY

When birds 1 to 5 weeks of age were fed on nicarbazine or amprolium as a coccidiostat and then exposed to an ambient temperature of 40°C significantly higher mor-

Table 1: MORTALITY IN CHICKENS FED COCCIDIOSTATS AND EXPOSED FOR 6 HOURS TO VARIOUS TEMPERATURES

Trial	Age (weeks)	Flooring	Exposure temperature	No. of birds in each group	Mortality Amprolium fed	Mortality Nicarbazine fed
1	5	Wire mesh covered with brown paper	40°C	20	0	0
Water	4	Wire mesh covered with brown paper	40°C	20	0	0
troughs	3	Wire mesh covered with brown paper	40°C	20	0	0
supplied	2	Wire mesh covered with brown paper	40°C	20	0	0
	1	Wire mesh covered with brown paper	40°C	20	0	0
2	5	Wire mesh only	40°C	20	5	15
Water	4	Wire mesh only	40°C	20	3	12
troughs	3	Wire mesh only	40°C	20	0	12
not	2	Wire mesh only	40°C	20	4	5
supplied	1	Wire mesh only	40°C	20	1	5
3	5	Wire mesh only	36°C	20	0	0
troughs	4	Wire mesh only	36°C	20	0	0
not	3	Wire mesh only	36°C	20	0	0
supplied	2	Wire mesh only	36°C	20	0	0
	1	Wire mesh only	36°C	20	0	0

talities were found in the nicarbazine fed chickens. At an ambient temperature of 36°C no mortalities occurred in either of the groups.

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BELANGRIKE GEBRUIKKODE VIR SUIWELBOERE

Die Buro vir Standaarde het pas 'n omvattende gebruikkode gepubliseer wat aanbevelings vir die skoonmaak en ontsmetting van suiweltoerusting dek. Dit sluit alle toerusting in wat by die produksie van melk en die verwerking en vervaardiging van melkprodukte gebruik word.

In die verskillende afdelings van die kode word die skoonmaak en ontsmettingsprosesse op suiwelplase en melkerye gedek asook van toerusting wat by die vervaardiging en verpakking van suiwelprodukte gebruik word. Die klem val hoofsaaklik op higiëne aangesien melk 'n baie goeie medium is waarin mikroörganismes kan groei. Gevolglik kan die belangrikheid van skoonmaak en ontsmetting van toerusting nie oorbeklemtoon word nie.

Om 'n bevredigende standaard te verkry, moet toerusting doeltreffend skoongemaak word sodat oppervlaktes wat met melk in aanraking kom, vry is van melkoorblyfsels en van bakterieë wat die openbare gesondheid in gevaar kan stel of die produkte kan laat bederf.

Daar moet egter op gelet word dat ondanks die aanbevelings in die kode ondernemings wat met die hantering van melkprodukte gemoeid is, steeds aan al die vereistes van toepaslike wette, veordenings en regulasies moet voldoen.

Landbounuus, 5 Mei 1978

THE USE OF CATTLE TO PROTECT SHEEP FROM BLUETONGUE INFECTION

E.M. NEVILL

ABSTRACT: Nevill E.M. **The use of cattle to protect sheep from bluetongue infection.** *Journal of the South African Veterinary Association* (1978) **49** No.2, 129–130 (En) Vet. Res. Institute, 0110 Onderstepoort, Rep. of South Africa.

Studies on the host preferences of *Culicoides imicola,* the vector of bluetongue virus in South Africa, are reviewed. There is agreement that this species prefers to feed on cattle but will also feed on other bovidae and sheep. Over a seven year period cattle kept near sheep on a Natal farm appear to have appreciably reduced the incidence of bluetongue in the sheep. In addition to immunization this "decoy" approach is therefore recommended to assist in the protection of stock from insect borne diseases such as bluetongue and possibly African horsesickness and Rift Valley fever.

INTRODUCTION

Between January and May 1960, du Toit[3] kept five bluetongue-susceptible sheep within 50 m of eight cattle in an open camp at Onderstepoort. By March all the cattle had contracted bluetongue (BT) but the sheep remained fully susceptible until the end of the test period. Du Toit concluded that "... sheep are not very attractive to *Culicoides* for the purpose of blood feeding and that the insects appear to feed much more readily on cattle which, when run in close proximity to sheep, may serve to protect such sheep from natural infection by drawing the insects to themselves".

The *Culicoides* spp. to which du Toit referred in this 1962 paper would have been predominantly *C. imicola (=C. pallidipennis)* as this species constituted more than 94% of the *Culicoides* spp. collected in light traps placed near cattle, horses and sheep at Onderstepoort[6] and du Toit had in 1944 already proved it to be the vector of BT in work done at Onderstepoort[2].

Nearly 10 years elapsed before more detailed studies were published on the host preferences of *C. imicola* in the form of blood meal indentifications using the precipitin test and by making use of forage ratios. The forage ratio technique compares the percent of engorged insects which have fed upon a given vertebrate host with the percentage that host comprises of the total population of hosts available in the insect's habitat. A forage ratio of 1 indicates neither preference nor avoidance of the indicated host animal; greater than 1 indicates selective preference; and values less than 1, avoidance in favour of other hosts[5]

In Kenya, Walker & Davies[8] used precipitin tests which showed that *C. imicola* fed on cattle and sheep. At Onderstepoort Nevill & Anderson[6], using precipitin tests, showed *C. imicola* to feed only on mammalian blood, predominantly cattle and horses and at least in one case on sheep. In Israel, Braverman *et al.*[1] used light traps in sheep pens to collect engorged *Culicoides* for precipitin tests. Despite the proximity of the sheep to the trap only 48 *C. imicola* contained sheep blood compared with 62 which had cattle blood. They also found that the monthly average forage ratio for *C. imicola* was 1,3 for cattle and only 0,2 for sheep indicating a very marked preference for cattle and thus confirming du Toit's observation[3].

In more recent work in Kenya, Walker & Boreham[7] using precipitin tests showed again that *C. imicola* blood meals were predominantly from cattle and wild Bovidae and to a much lesser extent from sheep or goats. The forage ratio also showed a preference for cattle and an analysis of light trap catches showed that more engorged *C. imicola* were collected near cattle than near sheep. Despite the above findings they concluded that high population densities of *C. imicola* do not seem to be necessary for feeding on sheep to occur and that cattle will be unlikely to provide much protection for sheep against the bites of *C. imicola* when together with sheep.

This conclusion, however, assumes that little or no immunity to bluetongue exists in a flock and that infected *Culicoides* do in fact get the opportunity of feeding on susceptible sheep. Although Foster *et al.*[4] in the U.S.A. have shown that the bite of a single infected *Culicoides* midge is sufficient to infect a sheep with BT, the chance of this happening will, however, depend on the incidence of infected individuals in the local *Culicoides* population and the number of *Culicoides* which get the opportunity to feed on sheep. If this number can be reduced so also will the BT challenge.

The following field experience demonstrates that a combined approach to a BT problem using immunization and cattle as bait hosts can be used successfully where immunization alone is inadequate.

CASE HISTORY

Mr. and Mrs. Neil MacGillivray, of Karkloof near Howick in Natal, started a stud Suffolk flock in 1965 as a secondary enterprise to their Friesland dairy herd. Because of abundant water the animals are run on irrigated pastures. The annual rainfall ranges from 760 to 1 150 mm while in 1975/76 1 400 mm was recorded. During the first few years the area experienced drought and BT was not a problem. However, in the summers of 1968/69 and 1969/70 BT was severe and at least six of 50 lambs of six months and older died while those which recovered did poorly and were useless for stud purposes. In addition the cost of treatment to prevent death from secondary infections was extremely high.

Prior to 1970 all sheep were immunized annually in December when the lambs were six months old. However, at this age lambs lose their colostral immunity at a time when the prevalence of BT is increasing. Because of the severity of their problem and the uniqueness of their situation the owners thereafter immunized lambs at two to 2½ months old and again four to six weeks later. Adult sheep were immunized annually in September. Theoretically this programme is inadvisable since inoculation of lambs at such an early age should result in existing colostral immunity interfering with the development of active immunity induced by the vaccine. However, colostral immunity is limited to the types of BT virus against which the ewe has antibodies so if these are absent or limited then early immunization of lambs can initially be warranted.

On the MacGillivray's farm *Culicoides zuluensis* and *C. gulbenkiani* are more numerous than *C. imicola* in spring, but in summer *C. imicola* is the dominant species (32-65%) with *C. zuluensis* responsible for between 22% and 30% of the light trap catches. The recognized vector of BT is thus abundant on this farm in summer and so presents a similar situation to that at Onderstepoort.

In December 1970 the writer visited their farm and recommended that they test du Toit's[3] suggestion of keeping cattle near the sheep in the hope that the *Culicoides* vectors would prefer to feed on the cattle. Except for the 1975/76 season when floods interfered with farming practices, they have continued to keep between six and 15 dry cows or pregnant heifers near 15 to 160 sheep of all ages. The sheep paddocks are small ranging from 0,1 to 0,2 hectare and if possible the cattle are kept in adjacent paddocks.

Thus in 1970 a new prophylactic programme involving immunization of two to 2½ month-old lambs repeated four to six weeks later combined with the presence of cattle near the sheep, was introduced. Since then no serious cases of BT in adult sheep have been seen although some very young late lambs born in October/November have died. The latter cases suggest that it is only necessary to immunise early to prevent BT. However, in the summer of 1975/76 when cattle could not be kept near the sheep, all the older rams contracted BT and the breeding programme was disrupted.

Seven years of experience of keeping cattle near sheep have convinced these farmers of the value of cattle in protecting sheep from serious BT infections and they refuse to do without the cattle.

DISCUSSION

It is unlikely that the immunization programme is solely responsible for the protection obtained since, as mentioned before, the early immunization practised could in fact be interfering with colostral immunity if the ewes have some degree of immunity. The almost complete freedom from serious BT since cattle were placed near the sheep seven years ago, and the relapse in 1975/76 when cattle were absent suggests therefore that cattle are playing an important rôle in protecting sheep from BT on this farm and substantiate du Toit's[3] findings.

The premise that preferred hosts may be used to protect man or stock from insect-borne diseases is not new and had also been suggested by Hess and co-workers in 1968[5]. In blood meal and forage ratio studies on *Culex quinquefasciatus* at a site in Hawaii, they noted marked differences in preferences between host species within the same class, such as chickens (forage ratio 3,5) and ducks (forage ratio 0,1). For mammalian blood meals this forage ratio was 3,7 for dogs as opposed to 0,3 for cows. They suggested that "... such data would be most useful for determining the relative values of different domestic animals in providing zooprophylaxis against mosquito-borne diseases (deviation of blood-sucking mosquitoes from man to other hosts)". At this site they felt "... that chickens and dogs would be the most effective species for zooprophylaxis against *C. quinquefasciatus*".

It is possible that this approach could be widely used in South Africa if the host preferences of the vectors of diseases such as Rift Valley fever (RVF), African horsesickness (AHS) and Wesselsbron virus disease were known.

For example, equines are unaffected by RVF but cattle, sheep and man are suceptible. Horses or donkeys when run with or kept near cattle and sheep may reduce the incidence of RVF in these animals. Because of the multiplicity of AHS virus strains it is difficult to fully immunize horses and at present owners are advised to stable the horses to reduce the chance of their being bitten by *Culicoides* vectors. This is of limited value since *Culicoides* do enter stables. Protection could probably be considerably improved if less valuable horses or cattle were kept in the vicinity of the stables to provide the midges with readily accessible and acceptable alternate hosts.

It is therefore suggested that in addition to the continuous development and improvement of vaccines, simultaneous research into the use of zooprophylaxis to protect stock from insect-borne diseases, he encouraged and that this approach be tested in the field whenever the opportunity arises.

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THE EFFECT OF NITROXYNIL* ON *PARAFILARIA BOVICOLA* INFESTATIONS IN CATTLE

A.C. WELLINGTON

ABSTRACT: A.C. Wellington. **The effect of nitroxynil on** *Parafilaria bovicola* **in cattle.** *Journal of the South African Veterinary Association* (1978) **49** No. 2, 131–132 (En) Box 1130, 6000 Port Elizabeth, Rep. S. Africa.

The Efficacy of nitroxynil administered at two dosage levels was evaluated against natural infestations of *Parafilaria bovicola* in cattle.

Lesion area were reduced by 55% and visible carcass lesions by 29% when nitroxynil was used at 10 mg/kg live mass and repeated after 72 hours, and by 95% and 90% respectively when nitroxynil was used at 20 mg/kg live mass and repeated after 72 hours.

INTRODUCTION

Viljoen and Boomker[2] treated cattle infested with *Parafilaria bovicola* with nitroxynil and demonstrated a 93% decrease in the mean affected lesion areas and a 90% decrease in the mean number of carcass lesions when compared with those in untreated control animals.

The present trial was carried out in order to examine the effect of nitroxynil at two dosage regimes on *P. bovicola* in older animals than used by Viljoen and Boomker[2].

MATERIALS AND METHODS

(i) Selection Experimental Animals and Treatment

During October, 1977, 35 cattle (cows, oxen and bulls) on a farm in the Thabazimbi district (North-western Transvaal) were selected from a herd of animals exhibiting lesions of *P. bovicola* and were randomly divided into three groups:

1. One group of 10 control animals.
2. Two treatment groups of 12 and 13 animals respectively.

Treatment took place during the period 7 to 10 October 1977 according to the programme outlined in Table 1. Thereafter the animals were kept on the farm until early January 1978 when they were transferred to the Veterinary Research Institute, Onderstepoort for slaughter and examination of the carcasses.

Table 1: PROGRAMME OF TREATMENT

Group No.	No. of Animals	Remedy	Dosage rate	No. of treatments	Method of treatment
1	10	Control	–	–	–
2	12	Nitro-xynil	10 mg/kg live mass	2 with 72 h interval	sub-cutaneous injection
3	13	Nitro-xynil	20 mg/kg live mass	2 with 72 h interval	sub-cutaneous injection

*'TRODOX' injection – Maybaker (S.A.) (Pty) Ltd. containing nitroxynil 34% m/v

(ii) Carcass Examinations

The control and teated animals were slaughtered during the period 4 to 11 January 1978. During the examination of the carcasses, the number of lesions was recorded and the dimensions of each measured directly on the carcass, while, after skinning, the total skin area of each animal was estimated.[2]

To differnetiate between typical parafilarial lesions and bruised areas, smears were taken from all lesions and stained by the Giemsa method to detect the presence or absence of eosinophiles. In the final calculations, only eosinophile positive areas were taken into account and lesions and lesion surface areas per animal in each group calculated.[1]

Grading of carcasses was undertaken by an approved government meat inspector of the Department of Agricultural Economics and Marketing.

In order to calculate the economic viability of the treatments, the following were taken into account: the cost of treatment; cold dressed mass; mass of trimmings due to lesions removed from the carcass; grading prior to trimming as if there were no lesions; grading carcass after trimming; financial loss due to trimming calculated on mass of trimming multiplied by carcass grading price prior to trimming; financial loss due to downgrading of carcass; sale of carcass calculated on the price per grade as at the Johannesburg abattoir on the date of slaughter.

RESULTS

Table 2 is a summary of the effects of each treatment regime on the number of lesions and eventual lesion area per carcass in each group. On a percentage basis, nitroxynil at 2 × 10 mg/kg decreased the eosinophile positive lesion areas by 55% and the regime of 2 × 20 mg/kg decreased the eosinophile positive lesion areas by 95%.

In the case of the animals treated at 2 × 10 mg/kg live mass, the results were shown by student's test to be statistically significant at $P<0,02$; and in the case of those animals treated at 2 × 20 mg/kg live mass at $P<0,01$.

The financial implications with reference to the cost of treatment, its efficacy and the financial saving with regard to the cost of this treatment as against the loss which would have been sustained if not treated, are shown in Table 3.

Table 3: FINANCIAL IMPLICATIONS

Goup	Mean dose of nitro-xynil per animal (ml)	Mean cost of treat-ment per animal R c	Mean live mass per animal (kg)	Mean cold dressed mass per carcass (kg)	Meat trimmed per carcass (kg)	Mean retum ex sale of carcass R c	Mean loss due to down-grading R c	Mean loss due to trimming R c	Total financial loss per animal R c	Saving per animal less cost of treatment R c
Control	–	–	287	153,2	2,5	120,21	6,10	2,10	8,20	
Treated animals 2 × 10 mg/kg	14,75	0,78	250	139,6	0,8	105,64	3,42	0,67	4,09	8,20 – 4,09 – 0,78 = R3,33
Treated animals 2 × 20 mg/kg	35	1,85	298	162,5	0,1	124,44	—	0,08	0,08	8,20 – 0,08 – 1,85 = R6,27

Table 2: EFFECT OF TREATMENT ON LESIONS FOR CARCASS AND PERCENTAGE CARCASS AREA AFFECTED

Treatment	Animals slaughtered	Mean number of carcass lesions		Percent decrease	Mean sub-cutaneous area per animal (cm²)	Mean lesion areas (cm²)		Mean percent affected area (cm²)		Percentage decrease in E+ lesion areas	Mean age of animal at treatment (months)
		Total	E+			Total	E+	Total	E+		
Untreated controls	10	5,8	5,4	–	30 711,2	3 229,4	3 060,2	10,5	10,0	–	19,5
Nitroxynil 2 × 10 mg/kg	12	4,25	3,8	29,6	30 163,3	1 407,1	1 372,2	4,7	4,5	55	17,6
Nitroxynil	13	1	0,5	90,7	30 931,4	357	164,5	1,2	0,5	95	18,6

E+ = Lesions or lesion areas positive for eosinophiles. Total = Visible lesions or lesion areas.

DISCUSSION

Nitroxynil injected subcutaneously at dosage rates of 10 mg/kg and 20 mg/kg, each repeated 72 hours later, resulted in a decrease in the eosinophile positive lesion areas of 55% and 95% respectively. The corresponding decrease in the number of eosinophile positive lesions at the two dosage rates was 29% and 90% respectively. Nitroxynil at the higher dosage regime is therefore effective in decreasing both eosinophile positive lesions and lesion areas by more than 50%, one of the criteria stipulated by Viljoen[2] which must be fulfilled before a drug may be regarded as having some effect. At the lower dosage rate nitroxynil did not fulfil these requirements entirely, carcass lesions were only reduced by 29%, and consequently the appearance of the carcasses suffered. This level of efficacy would also not be acceptable to the farmer.

Initially, 10 out of the 35 carcasses were grade 2 and the other 25 were grade 3. If these carcasses had had an initial overall higher grade, it is the opinion of the meat grading inspector that the financial gain per carcass would have been higher for the two treated groups than calculated in this experiment.

These results confirm the efficacy of nitroxynil at the 2 × 20 mg/kg dosage.

ACKNOWLEDGEMENTS

The author is indebted to the Director, Veterinary Research Institute, Onderstepoort, for permission to slaughter and examine the carcasses at the Onderstepoort abattoir and particularly to his staff: Dr. J.H. Viljoen for his help in checking the positive animals and lesions at the time of slaughter and Messrs. E.M. Nevill and G. van der Westhuizen for general assistance.

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COMMUNICATION MEDEDELING

ABOMASAL PHYTOBEZOARIASIS OF GOATS AND SHEEP

G.F. BATH*

Since 1973 a specific form of abomasal plant fibre ball or phytobezoar has been seen in goats and sheep from a large area of the Karoo. It has become clear that these bezoars can be an economical problem in some areas and under certain conditions. They can be easily differentiated from previously described forms of bezoars such as hair balls (zootrichobezoars) and grass seed balls (*Stipagrostis* spp).

The size of the bezoars may be 1 – 15 cm across, although most are 2 – 8 cm in section. Dry mass varies between 0,5 and 270 g, with a rough average of 5 – 10 g. The balls are dense and when waterlogged sink in water. Smaller bezoars, of which there may be over twenty in a single animal, are found mainly in young animals while large ones, often of bizarre shape, are found chiefly in adults. The shape can vary tremendously from the most common spheroid and ovoid forms to cuboid, trapezoid, or flattened forms, cylindrical shapes and several other forms which almost defy description. Occasionally several bezoars merge to form large lobulated structures. Generally the colour is buff to khaki, but may be almost black particularly where the surface is shiny. Usually the surface is regular and has a velvety texture, but sometimes deep irregular fissures may be present. On section numerous concentric layers can be seen, showing that the bezoars are probably formed by accretion. The central nucleus is not materially different to any part of the structure.

Investigations have revealed that the chemical composition of the bezoars is quite different to that of goat hair, but very similar in most important respects to plant fibre. Microscopic examination has shown a close physical similarity between the fibres making up the bezoars and the pappus hairs of the seeds of some common karoo shrubs.

In a small trial involving the feeding of the mature flowers of *Eriocephalus glaber, Arthrosolen (Gnidia) polycephalus,* and *Chrysocoma tenuifolia* to lambs and kids, morphologically similar bezoars have been formed from the latter two plants. Balls resembling the bezoars have also been formed *in vitro* from all these plant seeds using a felting techinque. From these results it appears that several karoo bushes may be involved in forming the bezoars, while the dominant plant species involved may vary from area to area.

The disease is encountered sporadically over a wide area, and has been reported from districts as far apart as Gordonia and Tarkastad. Several veld types are therefore involved. In the southern parts the disease is most common on mountainous farms, and there is some evidence that overgrazing increases the incidence of the disease.

It seems that goats are more readily affected than sheep, and that boer goats are more susceptible than Angoras. Both sexes are equally affected, but younger animals are more often clinically affected than adults. Older animals are often asymptomatic although one or more large bezoars may be present. Morbidity may be well below 1% on some farms while on others 20% or more of the flock may become affected. Mortality amongst affected animals is fairly high, though often bezoars are only found incidentally after the slaughter of apparently normal goats and sheep.

Generally, affected animals lose condition gradually, the abdomen becomes pendulous and watery on palpation, appetite decreases, and habitus is poor. The presence of the bezoars in the abomasum can readily be palpated through the abdominal wall. Sometimes diarrhoea may be present, and some animals die suddenly without previous sign of disease.

On post mortem examination the rumen and abomasum are often enlarged, thin – walled and contain watery, foul smelling blackish ingesta. The abomasum may rupture and partial or total obstruction is usually present. Below the site of obstruction, the intenstines are empty and atrophic. Emaciation is usually prominent.

Prevention of the disease by grazing practices is problematical since the bezoars are formed from the flowering parts of some very common karoo shrubs. Destruction of the bezoars by reagents not harmful to living tissues would be ideal but such a substance has not yet been found. Surgical intervention is quite possible but only justifiable in more valuable animals. However, since the presence of the bezoars may readily be detected by palpation, the farmer can at least limit his economic losses by immediately sending affected animals for slaughter.

Studies undertaken on various aspects of the disease are to be reported more fully in future publications.

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CASE REPORT **GEVALVERSLAG**

MENINGIOMA IN A DOG

J. BOOMKER*, P.E. KLOECK** and D. SCHAAP***

ABSTRACT: Boomker J.; Kloeck P.E.; Schaap D. **Meningioma in a dog.** *Journal of the South African Veterinary Association* (1978) **49** No. 2, 133–135, (En) Section. Pathology, Vet. Res. Inst., 0110 Onderstepoort, Rep. South Africa.

The history, clinical signs and pathological findings in a 9-year-old Labrador dog with an intracranial tumour, are described. The tumour conformed to the rare meningiothelial meningioma with focal ossification.

INTRODUCTION

Meningiomas have been reported in dogs, cats, horses, cattle and sheep[9 13]. They are said to be common in all animals[5], but are more frequent in cats[10], in which they occur intracranially and occasionally, spinally. Meningiomas are less often seen in dogs than in cats and are rare in sheep, horses and cattle[13].

In dogs, meningiomas arise both intracranially and intraorbitally from the meninges[15], and occasionally in the vertebral canal[14]. They are not primary tumours of the centrel nervous system since they are of mesodermal origin and are most often associated with the dura mater, but the pia mater and the tela chorioidea may also be involved[2 5].

Meningiomas grow by expansion rather than invasion, although the more anaplastic forms may demonstrate infiltration[4 13]. Ossification[2 8], invasion[3] and metastasis[7] have been reported to occur. Extensions from intracranial tumours can give rise to intra-orbital meningiomas, although the latter may also arise independently[6].

Clinical signs exhibited by dogs suffering from intracranial meningioma vary considerably and depend on the locality of the tumour.

The purpose of this paper is to report on the history, clinical signs and pathological findings of an intracranial meningioma with focal ossification in a dog, as few cases have beem described in the literature and the tumour is seldom diagnosed clinically.

HISTORY

The subject was a 9-year-old male Labrador. In August 1976 he had the first of a series of convulsions. During an attack the eyes were rolled back and there was excessive salivation. He was brought for examination about 30 minutes after the first convulsion and was found to be clinically normal. A few days later the dog had another convulsive attack, which was followed by heaving and retching. An intensive examination again failed to produce any indication as to the origin of the convulsions.

During the period December 1976 to March 1977 the dog had several similar attacks at irregular intervals. He had aged visibly, became very listless and suffered from general fatigue. His behavioural patterns had altered; he now sought company continouously and slept inside, whereas he had previously preferred sleeping in the open.

After a severe attack the animal was hospitalized but as his habitus was normal in all respects he was discharged after 4 days.

During April and May 1977 the convulsive attacks became more frequent and varied from 1–7 minutes in duration. Urinary and faecal incontinence usually followed an attack. Vision, hearing and balance were also subsequently impaired, and the dog walked around aimlessly with an inclination to move to the left. He frequently bumped into objects. The animal became more and more disorientated and often got lost within the confines of his own environment. On one occasion after an absence of a day, he was found in a dazed state.

Treatment with antibiotics, thiamine, corticosteriods and multi-vitamins resulted in a slight temprary improvement. Radiographical examination of the head showed no obnormalities.

Eventually the owners noticed that the left eye appeared glazed and it became apparent the he was blind in that eye. There was no response to commands and the dog showed no recognition of the owner's voice or scent. If, as a sign of affection, his head was patted, collapse and whining with pain resulted. He was humanely destroyed 10 months after the first convulsion occurred.

PATHOLOGICAL FINDINGS

The left cerebral hemisphere showed a depression 6 mm deep, 25 mm long and 20 mm wide on the dorsal surface, between the cruciate and ansate sulci, directly against the longitudinal fissure. The median parts of the posterior sigmoidal and postcruciate gyri were also involved. The depression was caused by a subdural tumour, 25 × 20 × 20 mm, projecting downwards from the roof of the cranium. The tumour was attached to the dura mater by its base and did not invade the surrounding tissues.

Grossly the tumour was greyish-white with an irregular almost nodular surface. Upon incision, it was firm and slightly gritty and the cut surfaces were smooth.

The tumour was fixed in 10% neutral buffered formulin prior to being processed, sectioned and stained with haematoxylin and eosin (HE), Luxol fast blue (LFB)[12] and Gomori's reticulum impregnation[11].

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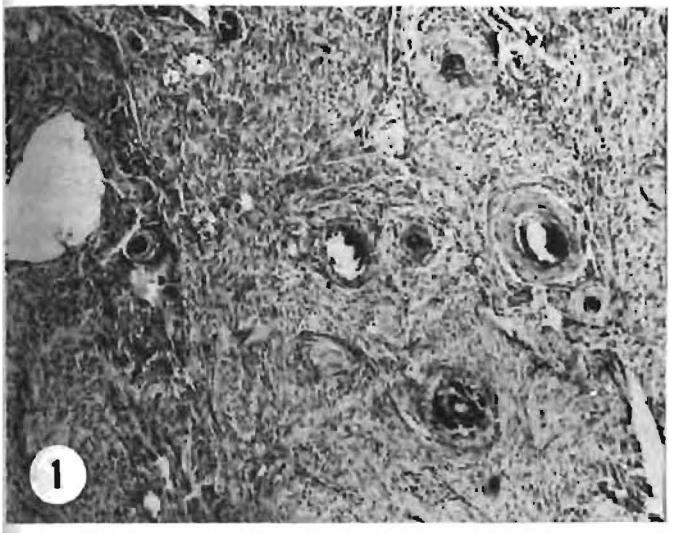

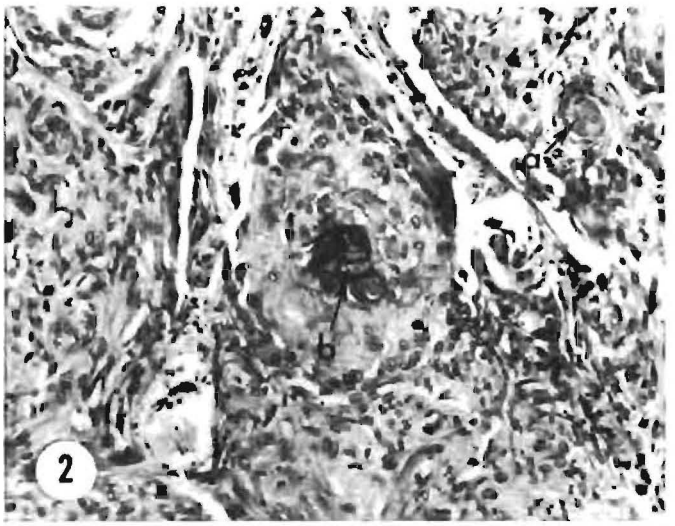

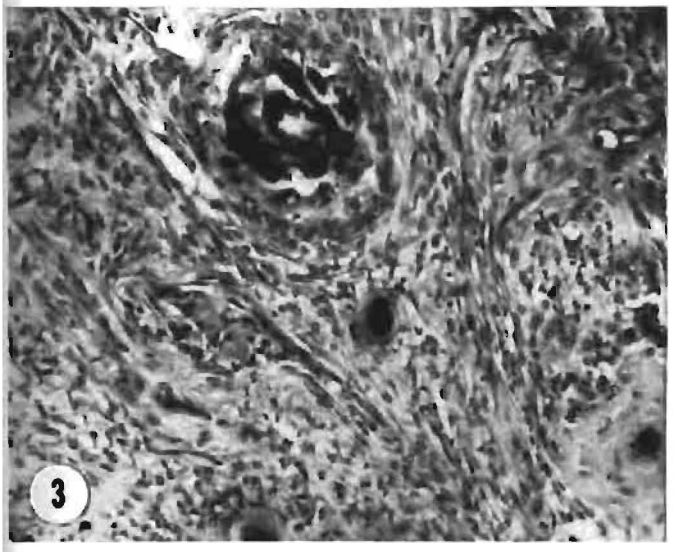

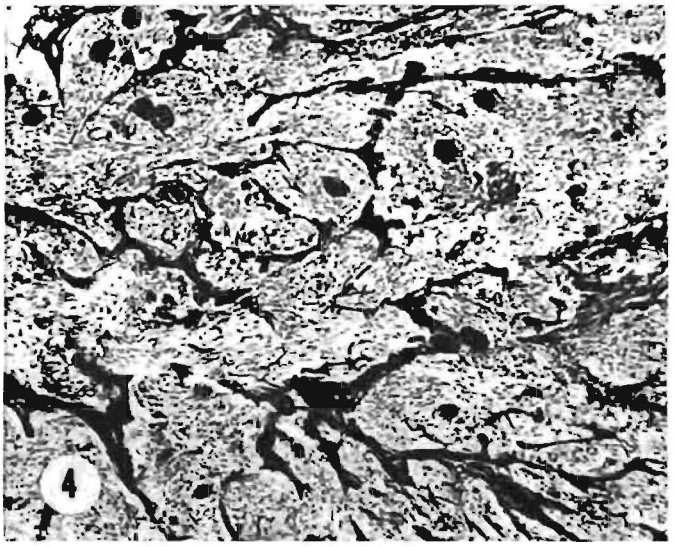

Fig. 1 Overall appearance of the tumour; HE, × 75.
Fig. 2 Part of the tumour showing cells containing eosinophilic globules (a) and cells surrounded by an osteoid-like substance (b); HE × 200.
Fig. 3 Mineralized spherules, one of which shows the trabecular arrangement of bone; HE, × 200.
Fig. 4 Overall appearance of the tumour, showing mineralized spherules, as well as cords and whorls of tumour cells separated by fibrous trabeculae; Reticulum stain; × 75.

Histopathology

The cells of the tumour were round to spindle shaped and of uniform size. The cell walls were indistinct and enclosed a lightly eosinophilic, slightly vacuolated cytoplasm. The nuclei were large and vesicular, round to oval in shape and contained a prominent nucleolus (Figs. 2 and 3). Mitotic figures were rare. Occasionally, cells with a more intensely eosinophilic staining cytoplasm and a pyknotic nucleus, were encountered.

The tumour cells were arranged in whorls and cords, separated by thin fibrous trabeculae, thereby giving it a lobulated appearance (Figs 1 and 4). In the centres of some whorls, 1 or 2 cells containing large eosinophilic globules and a pyknotic nucleus were occasionally seen (Fig. 2 a). In other areas a number of cells, surrounded by an osteoid-like substance arranged in concentric layers occurred (Fig. 2 b). Discrete mineralized spherules, some exhibiting the trabecular arrangement of bone, surrounded by concentric layers of tumour cells were often encountered (Figs. 1 and 3). Small foci of coagulative necrosis and karyorrhexis, accompanied by mild oedema, were also present irregularly throughout the tumour.

Blood vessels of the tumour were fairly large and conspicuous. No capilliaries were seen in the centres of whorls. The histologic appearance of this tumour closely resembles that of a meningiothelial meningioma, with focal ossification.

DISCUSSION

From the revision of the American and English literature, it is apparent that Boxers, Poodles, Shepherds, Collies and Terriers showed the highest prevalence of meningioma[1]. Sexes were equally affected and the mean age of afflicted dogs was 8,9 years (range 5–14 years)[1].

Meningiomas in animals often present a varied histologic appearance. Based on their histologic features they are classified as meningiothelial if the neoplastic cells are predominantly from meningeal origin, fibroblastic if fibroblasts are the predominant cell-type and psammomatous if mineralized spherules occur in the centres of whorls formed by neoplastic cells[1]. The pure angioblastic or angiomatous forms have, however, not been observed in animals[5].

Ossification of meningiomas has been described in dogs and the tumours were subsequently classified as osteogenic meningioma[2 7 15]. Histologically these tumours consisted of numerous irregular masses of bone and cartilage in a matrix of spindle cells[2].

In the present case, the deposition of osteoid-like substance and the formation of bony trabecula in the centres of whorls, indicated focal ossification. The growth therefore was interpreted as meningiothelial meningioma with focal ossification.

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GUIDE TO PRODUCTION OF RABBIT MEAT

A guide on the production of rabbit meat has been published by the Department of Agricultural Technical Services.

Rabbit farming for the meat market is gaining ground for various reasons. A relatively small area is necessary for an economic unit and a small-holding or even a fair-sized urban stand – where it is permitted – is thus suitable for rabbit farming. The initial capital outlay should not necessarily be high, depending upon the type of housing erected.

The rabbit compares favourably with other animals regarding the conversion of plantfoods into meat. To produce one kilogram live mass rabbit meat 3,5 kg is needed, compared with 2,2 kg for chicken, 3,5 kg for pork and 6,5 kg for beef. The advantage of the rabbit, however, is that he is able to effect this feed conversion on a ration which is less concentrated than that for either poultry of pigs.

The reproductive ability of a rabbit doe is remarkable in that out of a commercial herd, 40 small rabbits per doe per year can be marketed, which means that an animal of 3 kg can produce approximately 40 kg meat per year.

The guide also deals with the purchasing of breeding animals, housing, feeding, a breeding programme, marketing and disease control, and it can be ordered from the Division of Agricultural Information, Private Bag X144, Pretoria 0001.

Agricultural News, 5 May, 1978

GEVALVERSLAG **CASE REPORT**

PATOLOGIESE VERANDERINGS IN DIE PESE VAN DIE PEKTINEALE SPIERE MET HEUPDISPLASIE BY 'N HOND

W.S. BOTHA

ABSTRACT: Botha W.S. **Pathological changes in the tendons of the pectineal muscles in a case of canine hip dysplasia.** *Journal South African Veterinary Association* (1978) **49,** No. 2, 137–140 (Afr. en.) Dept. Path. Fac. Vet. Science, Univ. Pretoria, P.O. Box 12580, 0110 Onderstepoort Rep. South Africa.

This report gives radiological and pathological evidence of bilateral ossification of the aponeurotic tendons of insertion of the pectineal muscles in a dog. This was associated with a severe grade 3 hip dysplasia of which the pathological changes are described. The ossification of the tendons is considered to have resulted from excessive and continous tension of the pectineus muscles.

Various aetiological factors are discussed and some substantiation provided for the theory that pathology of the pectineal muscles and their tendons of insertion may be a contributing factor in the pathogenesis of hip dysplasia.

INLEIDING

Heupdisplasie is 'n welbesproke onderwerp in die veterinêre literatuur. Hierdie verslag handel oor die patologiese veranderings in die pektineale spiere en -pese in 'n 11-maand oue Boxer-reun met heupdisplasie, wat verwys is deur prof C.J. Roos (Afdeling Radiologie) en dr C. Button (Departement Geneeskunde) van die Fakulteit van Veeartsenykunde, Universiteit van Pretoria.

Twee toestande, vermoedelik nie met mekaar geassosieer nie, is klinies gediagnoseer. 'n Subaortiese stenose met linker-ventrikulêre hipertrofie was die rede vir primêre ondersoek en later ook genadedood van die dier. Die heupdisplasie onder bespreking was 'n bykomstige bevinding tydens die ondersoek. Radiologies was die heupdisplasie geklassifiseer as graad 3 as gevolg van die laterale verplasing van die femurkoppe en ernstige vervlakking van die asetabula. In Fig. 1 is hierdie heuppatologie radiologies sigbaar en word die verbening aan die mediale gedeeltes van beide femurs gesien.

PATOLOGIE

Met die nadoodse ondersoek is gevind dat beide femorale koppe na lateraal verplaas, en besonder beweeglik was met bilaterale subluksasie. In beide heupgewrigte was die gewrigkapsels en rondeligamente nog relatief ongeskonde hoewel laasgenoemde verrek en eersgenoemde verdik en fibreus voorgekom het.

Beide asetabulêre holtes was vlak en eksostoses is opgemerk in die aanhegtings van die gewrigkapsels op die pelvis en femurnekke. In Fig. 2 kan die benige eksostose sowel as 'n erosie aan die buiterand van die asetabulum gesien word. Dit is gevind dat 'n klein gedeelte van die femurkop aan die dorsale kant in kontak was met die buitenste gedeelte van die gewrigsvlak van die asetabulun. Aangesien die oppervlakte van kontak in die heupgewrigte deur die heupdisplasie aansienlik verminder was, was die liggaamsmassa nou onewerendig gekonsentreer op die gewrigsvlakke met gevolglike wanvorming en erosies op beide femorale en asetabulêre

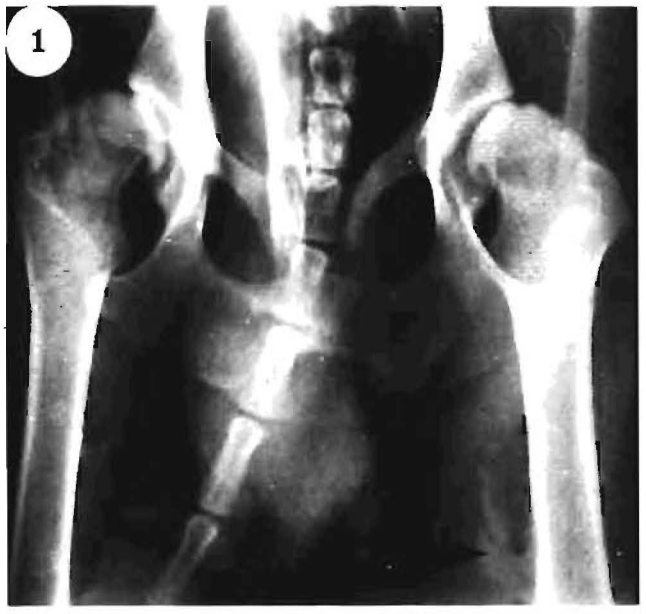

Fig. 1 Radiologies is die graad 3 heupdisplasie sigbaar. Ossifisering aan die mediale gedeelte van die regter femur word met 'n pyl aangedui.

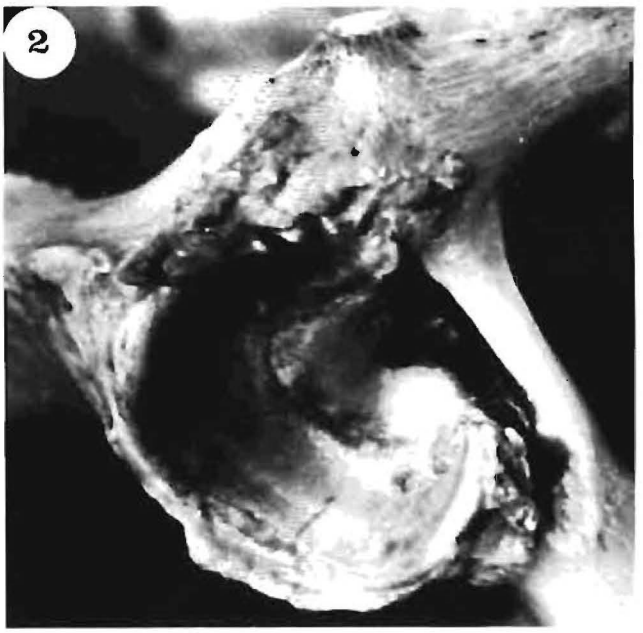

Fig. 2. Benige eksostoses aan die buiterand van die asetabulum. 'n Erosie van die gewrigskraakbeen is ook sigbaar.

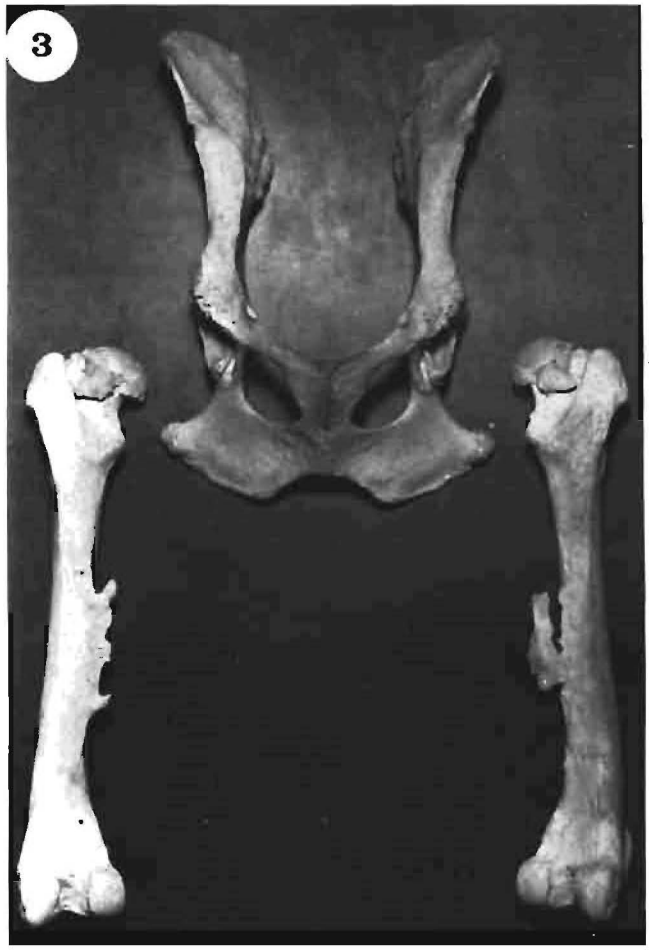

Fig. 3. Eksostoses by die plek van aanhegting van die aponeurotiese gedeelte van die pees van die pektineale spiere is duidelik sigbaar. Let ook op die veranderings in die heupgewrig.

oppervlaktes. Die femorale nekke het besonder kort voorgekom vanweë aansienlike eksostotiese been wat hier teenwoordig was.

Die pektineale spiere was besonder gespanne en 'n eksostose van 35 × 15 mm was in die regter pees van aanhegting by die femur teenwoordig. Die linker pektineuspees het 'n soortgelyke verbening van 40 × 10 mm getoon. Hierdie eksostoses (sigbaar in Fig. 3) was geleë aan die koudale verhewe rif, ongeveer halfpad tussen die mediale knie-gewrigsknobbel en die kleiner trokanterieseknobbel in die aponeurotiese gedeeltes van die pese van die pektineale spiere.

BESPREKING

Die beginsel, dat normale benige uitsteeksels in die liggaam ontwikkel in verhouding tot die spanningskragte daarop tydens embriogenese, is 'n anatomiese feit. Dit is ook bekend dat enige faktor wat abnormaal remodellering van been gedurende die neonatale periode veroorsaak 'n besonder uitgesproke patologiese verandering teweeg sal bring[14]. Met hierdie feite in gedagte is dit dus begryplik dat 'n besonder hoë spanning in die spiere en -pese teenwoordig was wat gelei het tot hierdie bilaterale eksostose.

Spasma en verkorting van die pektineale spiere word beweer 'n oorsaak van heupdisplasie in die hond te wees[1]. Die oormatige spanning veroorsaak 'n opwaartse defleksie van die femoralekoppe met geassosieerde koksofemorale gewrigslosheid en gevolglike heupdisplasie[1]. Dit word ook beweer dat met abduksie van die bene 'n besondere spanning in die pektineale spiere klinies waarneembaar is in honde met heupdisplasie[1 14].

Histopatologiese bewyse van die rol van die pektineale spiere in heupdisplasie word ook gegee deur die bevinding van spierveselatrofie[1], hipotrofie[5 13], en hipertrofie[5]. Hierdie pektineus-spierveranderings mag van primêre neurogene oorsprong[1] of 'n sekondêre spierverkorting agv die pyn van die arthrose wees [5]. Daar word beweer dat heupdisplasie 'n geneties-oorerflike toestand is wat nie dominant nog resessief voorkom maar as 'n poligenetiese toestand presenteer[3]. Hierdeur word bedoel dat dit deur die kumulatiewe aksie van 'n onbepaalde aantal gene veroorsaak word en dat die graad van uitdrukking deur 'n menigte van ander faktore beïnvloed mag word[3].

Behalwe die genoemde verkorting of spasma van die pektineale spiere word dit ook beweer dat abnormale metabolisme van estrogene[7], groei tempo[6 9], die massa van die pelviese spiere[11 13], rassomatotiepe[8 10] of die beperkte aksie van die *Muscularis capsularis coxae*[14] as bydraende etiologiese faktore mag wees. Heupdisplasie is 'n siektetoestand wat gewoonlik op 'n ouderdom van 3 tot 8 maande presenteer[6 12] terwyl daar geen morfologiese afwykings in die pelvis of koksofemorale gewrigte van neonatale honde met heupdisplasie voorkom nie[12]. Daar word ook na heupdisplasie verwys as 'n biomeganiese siekte waar beide patologiese afwykings en meganiese beginsels tydens beweging geld[13].

Heupdisplasie is dus 'n komplekse siektetoestand waartoe menigte faktore predisponeer en dit wil blyk dat patologie van die pektineale spiere en -pese 'n bydraende faktor in die patogenese van die toestand mag wees.

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CASE REPORT **GEVALVERSLAG**

GASTROSKOPIESE FOTOVERSLAG VAN ACHALASIEKARDIA IN DIE HOND

S.W. PETRICK

ABSTRACT: Petrick S.W. **Gastroscopic photo report of achalasia in the dog.** *Journal South African Veterinary Association* (1978) **49** No. 2, 139–140 (afr. en) Dept. Surgery, Faculty of Veterinary Science, Univ. of Pretoria, Box 12580, 0110 Onderstepoort, Rep. South Africa.

Five dogs with achalasia were examined pre- and post-operatively. Photos were taken of the diseased oesophagus, cardia, cardia with retroversion and the cardia following surgery. The normal structures are also shown.

INLEIDING

'n Gastroskoop is gebruik om achalasiekardia in honde te diagnoseer. Sigbare en funksionele veranderinge is waarneembaar in die esofagus en kardia. Die Olympus kamera, gekoppel aan die gastroskoop is gebruik om fotos te neem van die esofagus, kardia, kardia met retroversie en die kardia na kardiplastie. Fotos van die normale strukture word ook getoon. Sekere bevindings blyk van diagnostiese waarde te wees in geval van achalasiekardia in die hond.

MATERIAAL

In totaal is daar vyf honde ondersoek. Tabel 1 gee besonderhede van die diere wat ondersoek is en ook op watter stadium die ondersoek plaasgevind het.

Die Windhond en Saluki is dood of uitgesit kort na die operasie en was daar geen na-operatiewe ondersoeke gedoen nie. Die Doberman en Bulhond was eers na kardioplastie beskikbaar vir ondersoek.

METODE

Al die diere was onder algemene narkose tydens die ondersoek en is die gastroskoop oraal passeer. In elke geval was 'n mondsper gebruik om die instrument te beskerm. In enkele gevalle was dit nodig om speeksel, water en voedselreste uit die esofagus te suig om die ondersoek te vergemaklik.

RESULTATE

Die lumen van die esofagus is vergroot, met gas gevul en die esofagus omvou die tragea gedeeltelik. Hoeveelhede speeksel, water en voedselreste is teenwoordig. Vergelyk foto 1 van 'n normale esofagus met foto 2 geneem van die Saluki.

In gesonde honde is die kardia toe met ondersoek maar ontsluit simmetries wanneer lug ingeblaas word met behulp van die gastroskoop. Dit bly gesluit in gevalle van achalasiekardia. Fotor 3 toon 'n ontsluite kardia en foto 4 'n gesluite een.

Die gastroskoop gaan maklik deur die kardia in normale gevalle maar moeilik of gladnie in geval van achalasiekardia. In die Groot Deense hond kon die gastroskoop nie na die maag deurgestoot word nie.

Met retroversie vertoon die kardia glad of effens ingeduik in normale gevalle terwyl daar 'n duidelike prolaps van die esofageale mukosa in die Saluki aangetref is. Foto 5 toon 'n normale kardia met retroversie en foto 6 die prolaps wat in die Saluki gevind is.

Na kardioplastie is dit gevind dat die kardia permanent ontsluit bly en asimmetries vertoon. Foto 7 is die kardia van die Groot Deense hond en foto 8 die van die Bulhond.

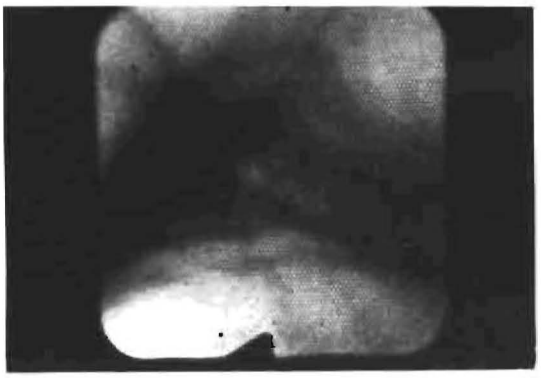
GEVOLGTREKKING

In geen van meer as 200 gesonde honde wat met die gastroskoop ondersoek was, is die bogenoemde afwykings opgemerk nie en kan die stelling dus gemaak word dat hierdie simptome eie is aan die toestand bekend as achalasiekardia in honde.

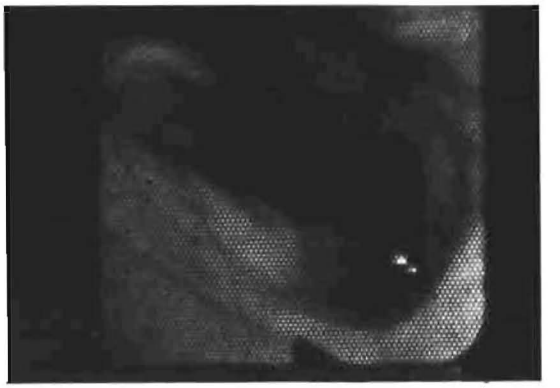
Dit blyk ook dat, as gevolg van die duidelike prolaps en die feit dat die gastroskoop selfs gladnie deurgestoot kan word nie, die weerstand wat ondervind word as gevolg van spasma moet wees en nie net weens die onvermoë van die kardia om op 'n prikkel te reageer nie.

Tabel 1: BESONDERHEDE VAN DIE GEVALLE WAT ONDERSOEK WAS EN OOK DIE STADIUM VAN ONDERSOEK

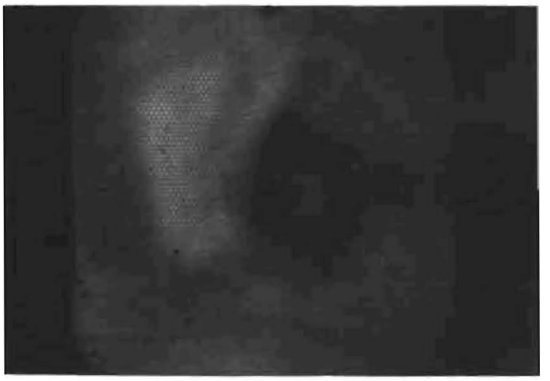
Ras	Groot Deense Hond	Windhond	Saluki	Doberman	Bulhond
Geslag	Manlik	Manlik	Manlik	—	Manlik
Ouderdom	2 maande	2,5 maande	5 maande	paar maande	6 jaar
Voor-operatief	.	.	.	—	—
Na-operatief	.	—	—	.	.



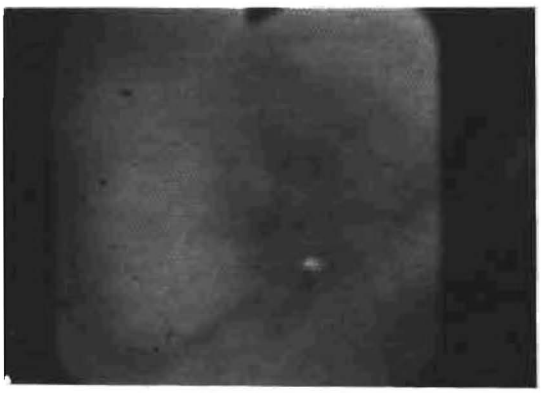
1 Normale esofagus



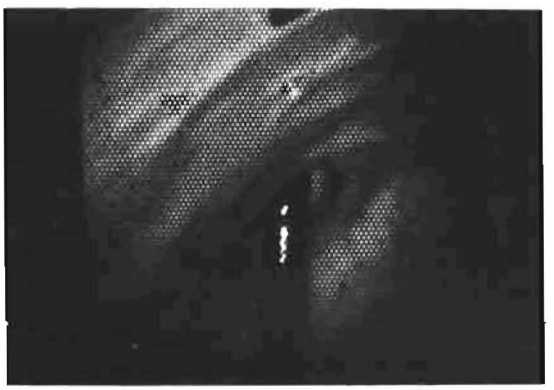
2 Vergrote esofagus



3 Ontsluite kardia



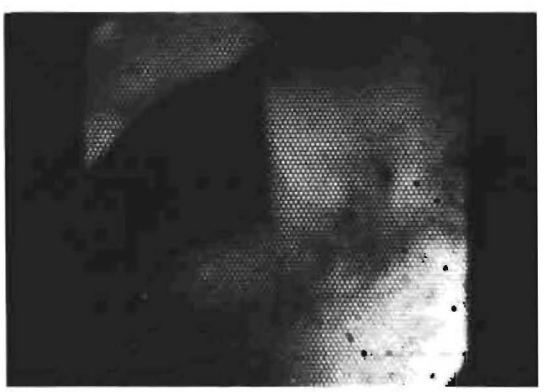
4 Gesluite kardia



5 Normale kardia met retroversie

6 Prolaps met retroversie

7 Kardia na kardioplastie



8 Kardia na kardioplastie

Fig. 1 Agt fotos geneem met die Olympus gastroskoop.

QUESTIONS & ANSWERS

VRAE & ANTWOORDE

Q. Could you give some information regarding *the inheritance of coat colour in Labrador dogs.* My client had his registered Golden Labrador Retriever bitch served by a registered Black Labrador male. The day after this service the female was served by a golden Labrador male, but not registered. Is there any possibility to deduct from the offspring which mating was successful?

A. Fortunately, the inheritance of coat colour in the Labrador Retriever has been clarified recently by Drs. Templeton, Stewart and Fletcher from the Texas A & M University based on results from a breeding colony established at the Health Science Centre, University of Oregon, Portland, Oregon.*

Labrador Retrievers have three basic coat colours, black, chocolate and yellow (also called golden). It is unnecessary to give all their breeding results, but a checkerboard scheme will be drawn on the basis of the above findings.

It has been shown that we deal here with a striking example of how non-allelic genes (borne on different chromosomes) can interact to produce an alternative effect in a single character. The gene interaction appears at two loci, the B-locus and the E-locus of the coat colour series in dogs:

The dominant B -allele determines black colour,
the recessive b -allele determines the chocolate colour, and
the recessive e -allele in homozygous condition the yellow colour, which is epistatic to the B-locus alleles.

The hypostatic B -alleles have limited expression in the homozygous e/e – individuals and are responsible for pigmentation of the iris, nose and lips.

In the following checkerboard scheme we start off with the breeding of a homozygous black to a homozygous yellow and take their offspring to the next mating *inter se:*

P:	Black BBEE	×	Yellow bbee			
F_1	Black BbEe					
F_1 dog (black)	Body cells BbEe					
	sperms	BE	Be	bE	be	
F_1 bitch (black) body cells BbEe	BE	BBEE	BBEe	BbEE	BbEe	
	Be	BBEe	BBee	BbEe	Bbee	
eggs	bE	BbEE	BbEe	bbEE	bbEe	
	be	BbEe	Bbee	bbEe	bbee	
F_2 Phenotypic ratio	9/16	Black				
	3/16	chocolate				
	4/16	yellow				

All possible genotypes are listed:

Black:	Chocolate:	Yellow:
BBEE	bbEE	bbee
BbEE	bbEe	Bbee
BBEe		BBee
BbEe		

In order to establish the genotype from the phenotype breeding tests have to be performed.

With all this information available, let us go back to the question above: Is there any possibility to find out from the offspring which mating was successful – the one with the registered black or the non-registered yellow male?

From the results shown in the checkerboard scheme it is obvious that the matings of yellows *inter se* only could give yellow offspring. But what is the probability of obtaining yellow offspring from the mating of the registered bitch with the registered dog?

Since no information is available of the genotype of the animals one must try to work out the average probability of obtaining yellow offspring.

POSSIBLE MATINGS OF BLACK AND YELLOW GENOTYPES

Parents		Offspring (%)		
Black	Yellow	Black	Chocolate	Yellow
BBEE	bbee	100		
BbEE	bbee	50	50	
BBEe	bbee	50		50
BbEe	bbee	25	25	50
BBEE	Bbee	100		
BbEE	Bbee	75	25	
BBEe	Bbee	50		50
BbEe	Bbee	37,5	12,5	50
BBEE	BBee	100		
BbEE	BBee	100		
BBEe	BBee	50		50
BbEe	BBee	50		50
Average possibility:		66	9	25

If all possible genotypes are considered 25% of the offspring would be yellows, but if the frequency of the different combinations is taken into account the probability of obtaining yellow offspring is increased to 35,3 percent. The frequency of the genotypes in a balanced population would be 1 BBEE, 2 BbEE, 2 BBEe, 4 BbEe, 1 bbee, 2 Bbee, and 1 BBee leading to a frequency of 58,8 percent black, 5,9 chocolate and 35,3 percent yellow offspring.

In conclusion it can be said that *if any black or chocolate offspring are produced in the litter it is obvious that the registered dog qualifies as sire.* If only yellow offspring are produced it can be said with a 65 percent certainty that the yellow dog has sired the yellow bitch in question. The number of offspring is of course another aspect for consideration. The average number of offspring in Labradors is 6,7 offspring per litter. In an abnormal small litter of 3 or 4 offspring we will only have one chance in two to give a correct answer but in a

*J.W. Templeton; A.P. Stewart and W.S. Fletcher: Coat colour genetics in the Labrador retriever The Journal of Heredity 68:134–135, 1977.

reasonable litter size of 6 to 8 the probability of a wrong conclusion would be decreased to about 20 percent, i.e. a certainty of 80 percent would be obtained that the yellow (golden) dog would qualify as the sire of the litter in question.

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EEN BEHEERSKEMA HOU VOORDELE VIR MELKBEDRYF IN

'n Enkele beheerskema vir melk impliseer nie strenger beheer of meer gesag oor of beperkings op die produsent nie, maar wel beter koördinering ten opsigte van die melkbedryf in sy geheel, sê die Adjunk-minister van Landbou, mnr. J.J. Malan.

Mnr. Malan het verlede week 'n boeredag in die distrik Bloemfontein toegespreek en verwys na die beoogde nuwe beheerskema vir melk en melkprodukte wat op 3 Maart ter inligting en vir kommentaar of beswaar afgekondig is en wat voorsiening maak vir gesamentlike beheer onder toesig van slegs een beheerraad.

Mnr. Malan het gesê dat die skema slegs groter stabiliteit en ordelikheid in bemarking tot voordeel van die produsent en in nasionale belang teweeg kan bring teenoor die huidige verdeelde beheer.

Afgesien van die verwagte kostebesparings ten opsigte van beheer hou die skema bepaalde voordele in met betrekking tot koördinering en bemarkingsreëlings en is die uiteindelike doelwit dat melk as melk beskou sal word. Die werklike gehalte van 'n produsent se melk sal bepaal vir welke doel dit aangewend sal word, met inagneming van die mark vir drinkmelk en produkte soos botter en kaas.

Die skema maak ook voorsiening vir landswye beheer sodat die huidige onbeheerde varsmelkgebiede ook daaronder ingesluit sou kan word indien regverdiging daarvoor bestaan. Die produsent wat tot dusver spesifiek vir die varsmelkmark geproduseer het en hoë kapitaalkoste aangegaan het om aan die vereistes te voldoen, hoef nie te vrees dat gesamentlike beheer tot sy nadeel sal wees nie.

Mnr. Malan sê daar is verskillende faktore wat die toekoms van die varsmelkprodusent beïnvloed. Markomstandighede en -behoeftes tel onder die bepalende faktore. Gesaghebbende vooruitskattings dui byvoorbeeld daarop dat aansienlike melktekorte reeds in 1980 in die vernaamste verbruiksentrums verwag kan word.

Alleen op grond hiervan kan aanvaar word dat daar 'n steeds toenemende vraag na varsmelk sal ontstaan en gevolglik 'n gunstige mark vir die produsent.

Origens kan die vraag na melk bykomend gestimuleer word deur onder meer markgerigte produksie op 'n gelykmatige vlak dwarsdeur die jaar. Dit behels onder meer die voorsiening van 'n produk van hoogstaande higiëniese gehalte, die beperking van prysstygings tot die absoluut noodsaaklikste en doelgerigte reklame.

Die ekonomie van varsmelkproduksie is 'n verdere faktor en waarskynlik die belangrikste wat die produsent se toekoms bepaal. Varsmelkproduksie kan een van die lonendste landboubedrywe wees, maar die voortdurende kostestygings soos in die afgelope tyd ondervind is, kan 'n uiters nadelige uitwerking hê. Ten einde sy posisie te kan handhaaf, moet die prys van die produsent dus vergoedend genoeg wees om sy koste te verhaal en sy onderneming en arbeid te loon. By die bepaling van die produsenteprys, sê mnr. Malan, moet egter ook gesorg word dat ondoeltreffendheid nie gesubsidieer word nie. Dit berus basies by die produsent om ekonomies te produseer en sy eenheidskoste so laag as wat moontlik is te hou. Dit kan bewerkstelling word deur die hoogs moontlike produksie per koei, goeie bestuur, kennis van die nuutste ontwikkelings en metodes, intensiewe arbeidbenutting, die lewering van gehalteproduksie, die bekamping van siektetoestande en behoorlike rekorthouding.

Varsmelkprodusente behaal reeds in 'n groot mate sukses in dié verband.

Landbounuus, 5 Mei 1978

TO THE EDITOR — AAN DIE REDAKSIE

THE LIFE-SPAN OF MAMMALS: ESTIMATES FOR THE DASSIE *(Procavia capensis)*

Dear Sir,

To determine the true life-span of any mammal, Petrick[10] developed the following formula: $y = c\sqrt{x}$; where y = life-span, c = 41,2 (constant), and x = first lifecycle. The first life-cycle equals twice the gestation period plus the period of puberty (i.e. the time it takes since birth to attain sexual maturity).

In the thesis[11] of which Dr. Petrick's subsequent paper[10] is an extract, the highest recorded age for *Procavia capensis* is quoted as approximately 6 years[4], the period of puberty as 24 months[3], and the gestation period as 7,5 months[1]. A first life-cycle of 39 months is thus calculated, giving an estimated life-span of 21 years. Because of this large difference between recorded and estimated life-spans, and the unresponsiveness of the data to various calculations, the dassie, and thereby a whole order, was excluded from the final list of 81 species (12 orders) on whose data the formula is based. Since this exclusion is purely the result of insufficient data utilized, suspicion in cast on the validity of Petrick's baseline data for other species, and consequently on the reliability of his formula-estimates. The dassie is here viewed in retrospect, especially as regards the acceptability of formula-estimates of life-span.

The highest recorded age for a dassie (a female *P. capensis syriaca;* regarded by Bothma[2] as *P. syriacus) is 12 years and 4 months*[7]. Females from two other species, *P. capensis* and *P. johnstoni mackinderi,* gave birth at 10 years[9] and 9 years[12] of age respectively. Both lived for some time after. Since dassie females usually do not live beyond their reproductive capacity[8], these ages represent true life-spans, i.e. ages at which reproduction is still possible. There can be little doubt that all dassies become sexually mature at 16 to 17 months of age[6 8]. Up to one third of *P. capensis* females and a few males may, however, already reach puberty 4 to 5 months after birth[8]. A gestation period of 7,5 months (230 days) is generally accepted[7 8 9 12]. The first life-cycle of the dassie therefore has its limits between 19,5 months (puberty period = 16,5 months). The formula life-span of the dassie accordingly varies between 15 and 19 years.

If the difference in years between the recorded and formula-estimated life-spans is expressed as a proportion of the recorded age, the dassie shows an 18% to 35% higher estimated life expectancy than the highest age reached. In Petrick's table 2[10], the life-spans of 32 species as calculated by means of the formula are listed against "authorative estimates" of life expectancy. A mean higher formula-estimated life-span of 15%, with a standard deviation of the mean of 27%, is shown by the summed data of the 32 species. The dassie compares well: 17 species have positive percentage differences (i.e. formula-estimates higher than recorded ages) above 18%. Of these, 8 fall inside the range of 20% to 33% and are comparable with the dassie, while 9 are above 35% (range 37% to 69%). If negative percentage differences are added, i.e. where recorded ages exceed the formula-estimates, the number of species showing departures of more than 18% either way from their respective recorded ages totals 20. These differences are not necessarily unacceptable or critical of the reliability of Petrick's formula. Interesting is the fact that all except two of the 7 domestic species listed show negative percentage differences, and it appears that the higher the degree of domestication, the higher is the negative percentage figure. The sequence and ratings are: sheep 7%, cattle 0%, pig −8%, donkey −14%, horse −23%, cat −25% and dog −33%. In the case of the other animals listed, a similar sequence can be stated whereby an increase in the positive percentage difference might suggest less chance of domestication or taming. It is, however, difficult to prove and can only be speculative. One wonders whether the Petrick formula tends to underestimate in the case of species which adapt well to captivity and to overestimate in the case of species which do not.

It is possible that the striking difference between recorded and estimated lifespan is not the result of an inherent fault created by the choice of functions in the formula. It could be attributable to external factors in the animal's environment which have a bearing on the life-span and which cannot be taken into account by the formula. The dassie, for example, utters characteristic sounds when it is exposed to extreme stress situations[5]. Such behaviour almost without exception preceded deaths without injury or disease in my captive colony. Given a slightly raised but sustained level of excitement in the dassie, the same toll could be taken over a longer period, resulting in a consistently lower life expectancy for the species. Could this be the reason for the dassie not living up to expectations, so to speak?

Yours sincerely,

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TO THE EDITOR — AAN DIE REDAKSIE

THE LIFE-SPAN OF MAMMALS: ESTIMATES FOR THE DASSIE *(Procavia capensis)*

Dear Sir,

I fully appreciate the fact that a formula of this kind is a very vulnerable one. Therefore any reaction is not only welcome but essential, because defence alone will bring proof to its reliability.

Dealing with the class Mammalia as a whole and its more than 5 000 species one is inclined to or even forced to collect such literature that will provide data on all or most of these species. This however does not necessarily result in "insufficient data utilisation", far from it because such literature includes data from all over the world and from many researches.

Being interested in a single order, or for that matter a single species one should undoubtedly be able to collect more literature and data. I do not believe that the exclusion of the dassie and its order from the final calculation has at all influenced the construction of the final formula. The reason, I hope, will be found in the rest of this paper.

Fourie correctly states that the reproductive life equals the true life-span of the dassie, however this is true for all mammals including *Homo sapiens.* He adds to my knowledge three more recorded ages for the dassie and the 12 years and 4 months is an improvement, almost living up to the expectation of the formula. However it is not yet good enough. Do we know if these recorded ages were the end of the line concerning their reproductive capacity? There is a marked difference between the 4,5 months and 16,5 months for the puberty period and one wonders which one is the natural one and which the acquired in captivity. The average life-span of 17 years according to the formula is rather promising.

Let me now explain why even the 12 years and 4 months is not the true life-span of the dassie. There can be little doubt that a correlation exists between the first life-cycle and the true lifespan of a mammal. (This was fully explained in the thesis). A change in either one can not take place without affecting the other. This type of change however is not easely or not at all accomplished and *Homo sapiens* provides us with all the answers. In spite of the fact that we have added many years to our true life-span it has not changed the onset of menopause in the woman, indicating the end of reproduction, neither has it affected the length of pregnancy or for that matter the length of the first life-cycle.

Although we do not agree on much about the dassie we both accept that the gestation period is 7.5 months. If for the next calculation we accept that the end of the dassie's reproduction period is 12 years and 4 months and we know the 7,5 months is the correct gestation period then let us calculate the duration of the puberty period. This is possible with the same formula that is used to calculate the true life-span.

$$P = \frac{Y^2}{C^2} - 2G$$

P = Puberty period
G = Gestation period (7,5 months)
Y = Life-span (148 months)
C = Constant (41,2)

$$P = \frac{148^2}{41,2^2} - 2(7,5)$$
$$= \frac{21904}{1697,44} - 15$$
$$= 12,904 - 15$$
$$= -2,096 \text{ months}$$

The negative puberty period means that a female dassie can be with young before it is born or else it proves that the dassie can not have such a short true life-span even though the correct one has not yet been recorded in captivity.

Positive and negative percentage differences makes the comparison between formula-estimated life-spans and the authorative estimates appear to be rather negative. The "striking difference" might well be explained in some other way.

The degree of domestication is certainly not the only factor responsible for the negative percentage differences in the domestic species. Veterinary Science has improved the life-spans of these species considerably and may be in the precise sequence Fourie has mentioned. For example the formula estimated life-span of the horse is 22 years and the authorative estimate is 27 years but in his studies on the longevity and mortality of English thoroughbred horses, Comfort (1959) came to the conclusion that "the model age of adult death is 22 years". We have added years to their true life-spans as has Medicine to that of *Homo sapiens.* It also indicates

that less is done to those species we consume and again the sequence shows remarkable accuracy.

With regard to the wild animals in captivity (there is no domestication in that) the positive percentage differences (i.e. formula estimates higher) clearly indicate that many species, including the dassie, have not yet and never will adapt themselves to captivity or for that matter the artificial care of *Homo sapiens*.

Now, after we have changed biology into mathematics, let us return to reality and consider the little creature itself. The dassie with a mass of 3–4 kg, an exceptionally long gestation period and the puberty period somewhere between 4,5 months and 24 months, is often refered to as being the nearest relative of the elephant. It is thus burdened with a relatively long first life-cycle and yet because of its poor performance in captivity we falsely underestimate its natural life-span.

In conclusion let us again consider the list of 32 species where a comparison is made between formula estimated-life-spans and authoritive estimates. Without percentage differences a remarkable correlation exists between these two estimates in at least 12 of the species. (We might also include the horse for that matter). Important is the fact that these 12 species include the elephant and the mouse giving it a wide range. Whether this is all pure coincidence I very much doubt and in favour of the formula one can state that it is based on physiological parameters whilst the authoritive estimates are based on recorded ages of mammals in captivity and domesticated species.

Your sincerely,
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BOOK REVIEW — BOEKRESENSIE

ATLAS OF RADIOGRAPHIC ANATOMY OF THE DOG AND CAT
ATLAS DER ROENTGENANATOMIE VON HUND UND KATZE

H. SCHEBITZ & H. WILKENS
Verlag Paul Parey, Berlin & Hamburg 1977 3rd Revised Ed. Pp 200, Figs. 274, Publ. Price DM180.

Accurate radiological diagnosis not only depends on good quality radiographs and an adequate knowledge of pathological conditions but very often also on the interpreter's ability to differentiate between abnormal and normal radiographic anatomy.

The Atlas under review is a comprehensive, and well-illustrated reference work on the normal radiographic anatomy of dogs and cats. It can be consulted with confidence when one is in doubt about the normality or otherwise of any anatomical structure on radiographs.

Included in the Atlas are brief descriptions and illustrations of the relevant methods of positioning for proper radiological examination of the various body regions. Technical data (e.g. exposure factors) necessary for obtaining good radiographs in each position is also supplied.

The various anatomical structures are identified on linedrawings accompanying large detailed reproductions of the radiographs. In the case of the carpus and tarsus identification of the small superimposed bones is facilitated by outlining them in different colours.

A number of more specialized procedures like myelography, pyelography, bronchography, pneumoperitoneum, etc are also described and illustrated.

The text is written in English and German. The nomenclature used throughout the Atlas is based on Nomina Anatomica Veterinaria (NAV 1973). A list of contents and an extensive bibliography are provided.

Judging from the frequency with which the previous edition of the Atlas has been consulted in the Radiology Section of our Veterinary Faculty, small animal practitioners will find this revised edition very helpful indeed.

It can be recommended unreservedly.

CJR

TREFFERBLAD FEATURE PAGE

SPIERATROFIE IN 'N PERD

Meegaande foto illustreer atrofie van die M. pectoralis superficialis in 'n perd. Adduksie van die voorbeen is hierdeur belemmer. Met beweging van die dier is daar 'n opvallende uitswaaiing van die voorbeen. 'n Binnespierse inspuiting in die betrokke spier was vier maande tevore toegedien. Kom dit moontlik vir die toestand verantwoordelik wees? Dit word aanbeveel dat in volwasse perde binnespierse inspuitings verkieslik in die laterale nekspiere toegedien word.

Ingestuur deur:

Dr J van Heerden
Departement Geneeskunde
Fakulteit Veeartsenykunde
Onderstepoort 0110

MUSCLE ATROPHY IN A HORSE

This foto illustrates atrophy of the M. pectoralis superficialis in a horse. Adduction of the fore limb is hindered as a consequence. During locomotion dishing of the front leg was marked. An intramuscular injection was given in this muscle four months previously. Could this have been the cause of the lesion? It is recommended that intramuscular injections in adult horses should be given into the lateral neck muscles.

Submitted by:

Dr J van Heerden
Department of Medicine
Faculty of Veterinary Science
Onderstepoort 0110

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Hookworm	A	A	A
Nodular worm	A	A	A
Large-mouthed bowelworm	A	A	A
Lungworm	A	A	A

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