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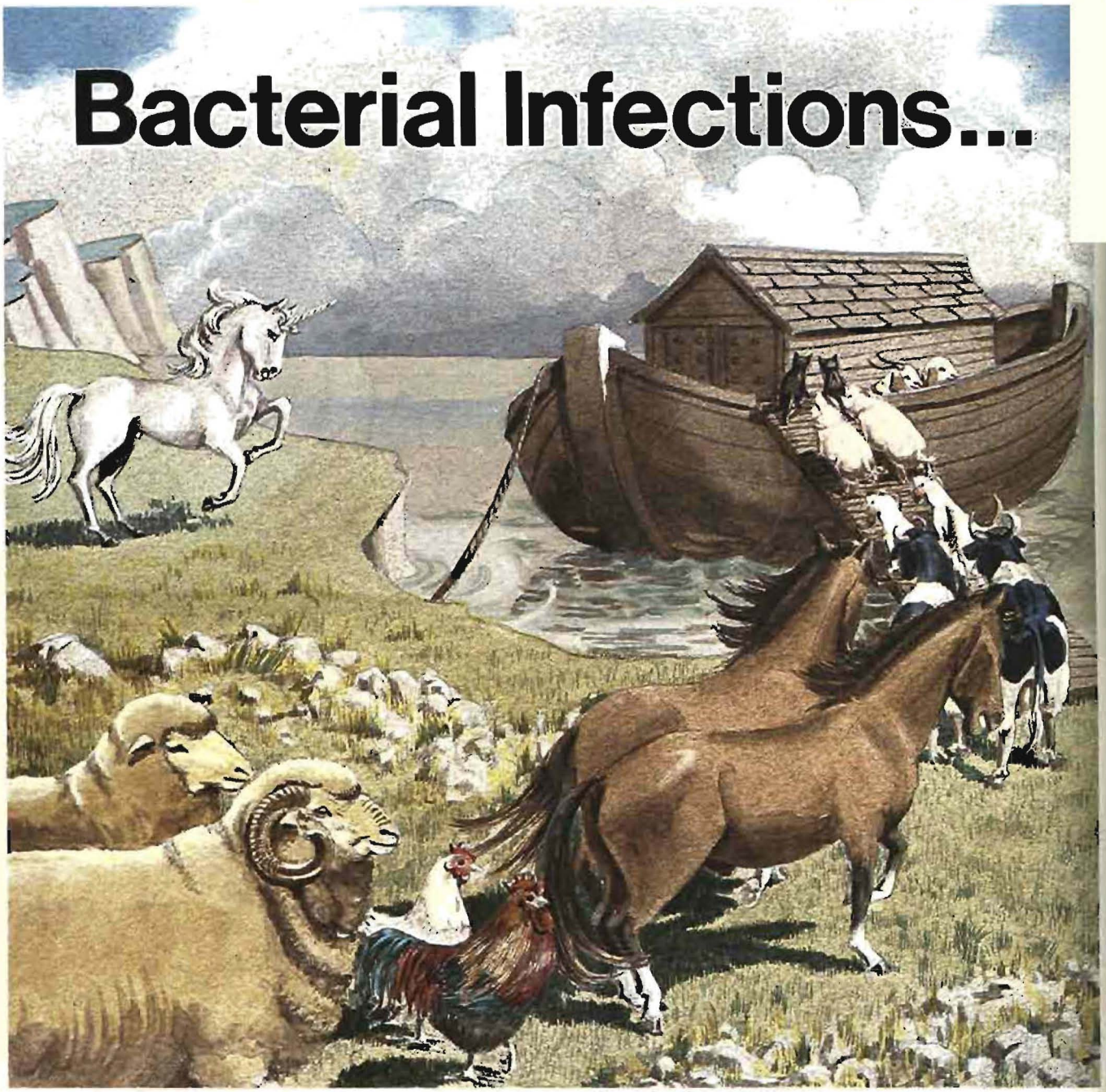

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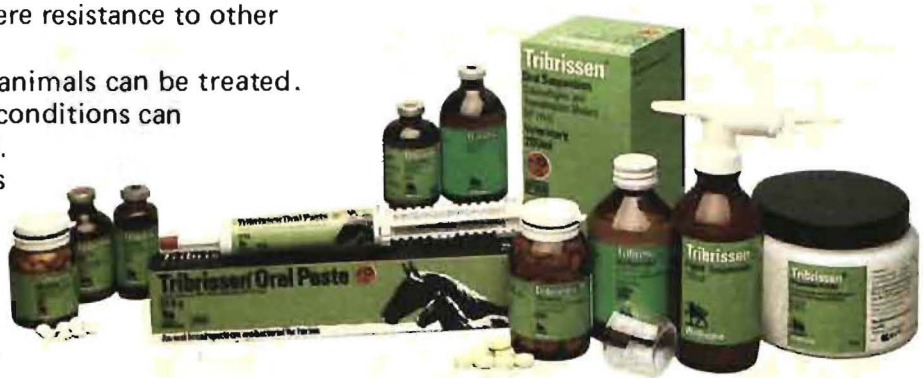

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JOURNAL OF THE SOUTH AFRICAN VETERINARY ASSOCIATION

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AN OUTBREAK OF OVINE LISTERIOSIS ASSOCIATED WITH POOR FLOCK MANAGEMENT PRACTICES

C.D. MEREDITH and D.J. SCHNEIDER*

ABSTRACT: Meredith C.D.; Schneider D.J. **An outbreak of ovine listeriosis associated with poor flock management practices.** *Journal of the South African Veterinary Association* (1984) 55 No. 2, 55-56 (En). Regional Veterinary Laboratory, Private Bag X5020, 7600 Stellenbosch, Republic of South Africa.

An outbreak of listerial meningo-encephalitis involving sheep in the Western Cape Province is recorded. Poor management practices which are described were thought to be principal precipitating cause.

Key words: *Listeria monocytogenes,* management, sheep.

INTRODUCTION

Du Toit[2] reported the first confirmed diagnosis of listeriosis in ruminants in South Africa. In that outbreak the flock of goats involved was maintained outdoors on natural veld grazing and no predisposing factors could be determined. This paper records an outbreak in the same winter rainfall area of this disease during winter and early spring in a small flock of Ille de France sheep kept under semi-intensive conditions.

CASE HISTORY

The first animal to be diagnosed as having listeriosis was a 2.5 month old lamb that died suddenly in late July. Prior to this case there had been 5 deaths over a period of 2 months which, in retrospect, were probably also listeriosis as the animals exhibited typical nervous signs, i.e circling with the head deviated to one side, nystagmus, inability to eat and a rapidly developing inability to stand, followed by coma and death. Ultimately 10 sheep out of approximately 275 died. These were mostly adult ewes.

Because of the value of some of the affected animals, antibiotic treatment (parental chloramphenicol) was tried in some cases by the farmer on the advice of his veterinarian, but was of no value as once clinical signs were established, no improvement occurred although life may have been prolonged. It was not always possible to establish with certainty which of the specimens delivered to this laboratory were from treated sheep and which were not.

Blood was collected in heparin from the first sick sheep and submitted for laboratory analysis, but not for bacterial isolation. Specimens from necropsies were collected in 10% buffered formalin, sectioned and stained with haemotoxylin and eosin. *Listeria* organisms were demonstrated using Gram-staining by the modified method of Brown and Brenn[5].

LABORATORY FINDINGS

Gross Pathology: A complete post mortem was performed on only 3 of the 5 sheep confirmed as having listeriosis. In the remaining 2 cases, only the brain was available for examination . No significant changes were found in organs other than the brain, and in the first confirmed case submitted alive the only abnormal blood chemistry figures were an elevated serum globulin concentration (56 g/ℓ) and glutathione peroxidase (13 min). This sheep also had glucose present in the urine.

The presence of ingesta in the mouths of some sheep indicated paralysis of masticatory and swallowing reflexes. One ewe had distinctly thickened meninges giving the membranes a slightly milky appearance and histopathologically this animal had marked mononuclear meningitis extending over the whole brain, whereas in the other 4 cases meningitis was confined to the brain stem and cerebellar regions.

Histopathology: Lesions characteristic of listeriosis were found to a variable degree in all 5 sheep examined. Lesions occurred in the brain stem between the midbrain and upper cervical spinal cord but were more severe and extensive in the medulla and/or pons in all animals. They consisted of single or multiple microabscesses in the grey and/or white matter with infiltration by neutrophiles and a few round cells. Depending on the degree of development of the microabscesses, reaction of the nervous tissue varied from mild perivascular mononuclear cell accumulations to massive cuffing and infiltration of quite large areas by many monoculear cells. Within the microabscesses necrotic and liquefactive changes were present.

In 3 animals that had definitely not received antibiotic, Gram-positive, short, rod-like bacteria, usually paired, were readily demonstrated in and around the microabscesses but were not seen where only relatively diffuse mononuclear cell infiltration was present. The lesions of the other 2 animals appeared to be sterile as either no bacteria or only small, mishappen, Gram-positive fragments could be found and it is assumed that these were the sheep that received treatment.

Bacterial isolation of *Listeria monocytogenes* was accomplished in only one case and it is probable that antibiotic treatment was mainly responsible for the failure to do so in the others.

A degree of mononuclear leptomeningitis was present in all cases.

ENVIRONMENTAL FACTORS

The winter during which this outbreak occurred was one of exceptionally heavy rain and low temperatures in the Western Cape, interspersed with brief clear periods when daytime temperatures sometimes reached as high as 28 °C. Similar conditions were associated with outbreaks of listeriosis reported from Australia[8] and Scotland[9].

On the farm in question the sheep were maintained on a deep-litter system to produce compost for the vine-

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yards. Because of pasture limitations, grazing was restricted to two, two-hour periods early and late in the day and consisted of lucerne paddocks supplemented by occasional access to natural winter grazing in the vineyard and guava orchard. The latter also provided some supplementation in the form of fallen guava fruit. Chopped wheat straw constituted the balance of the diet and was fed in troughs in the sheep pens.

For the remainder of the day and at night the sheep were confined in a walled structure about 3 m high and approximately 15 m square with no wall openings other than the entrance gate. This enclosure was roofed on all 4 sides with an open courtyard in the centre.

The sheep were further confined by wooden railings to an L-shaped area approximately 4 m wide under the roof on 2 sides of this building. As, for various reasons mostly concerned with the unfavourable weather pattern, the litter had been allowed to accumalate to a depth of almost a metre, the sheep had access to a very restricted vertical air space, open for ventilation purposes only along one side, and with no openings to permit cross-ventilation.

Inspection *in loco* revealed that the sheep were badly overcrowded, their nutritional plane low, with high levels of free ammonia gas from decomposing urine, etc. being present; sufficient to severely irritate the human upper respiratory tract. In addition, accumulated body heat and lack of airflow produced an uncomfortably high temperature and these various factors were reflected in noticeably abnormal breathing patterns in the sheep.

CONTROL

It was obvious that the sheep were being subjected to severe stress under the prevailing conditions and the farmer was advised immediately-

1. to break down the internal fences thus alleviating the overcrowding and allowing the animals access to the open centre courtyard of the enclosure where ventilation was better,
2. to remove the deep litter as soon as possible to limit the ammonia production and provide more overhead air space,
3. to reduce flock numbers to permit the remaining animals to spend a greater portion of the day on the available grazing, and
4. to improve the quality of the supplemented feed.

The first 2 of these recommendations were in fact implemented without delay and no further losses from listeriosis were reported.

DISCUSSION

L. monocytogenes is considered a common soil saprophyte capable of persisting under suitably favourable conditions for many years[4,10]. As outbreaks in other countries have often been associated with the feeding of silage[9,6], attempts to link the two together by isolating *Listeria* from the silage during outbreaks have been made[3,9]. Although limited success has been achieved in one or two instances it would seem that wet, muddy conditions may well be more important[8,9], with perhaps stress factors playing the deciding role in precipitating an outbreak[4]. If it is assumed that *Listeria* is very commonly present in the environment, outbreaks, such as the one reported here, can be related to the quality of the management and thus the stress endured by the animals rather then seeking a simple feed contamination explanation. Under wet, muddy, overcrowded conditions, exacerbated by extreme cold, animals are constantly exposed to infection orally, nasally or conjunctivally. These are the most likely routes of introduction of the bacteria in the encephalitic form of listeriosis and are likely to become infected when their natural defence mechanisms become overwhelmed[8,9]. In the outbreak described here, further stress and insult can be attributed to toxic ammonia fumes, extreme humidity and high carbon dioxide and low oxygen tension. It is perhaps surprising that *Pasteurella* pneumonia did not occur as well as it often does in this region at this time of year.

While the probable damage done to the nasal mucosae by ammonia gas tempts one to claim this as an explanation for this particular outbreak on the grounds that a damaged mucosa would more readily permit ingress of bacteria, there is practically no evidence, especially in this particular instance, for aerogenous transmission, and transmucosal oral infection from mud-soiled feed seems far more likely as the work of Asahi et al. would seem to indicate[1].

It is, however, worth mentioning in passing that if a virus-like agent were to be incriminated in the aetiology of listeriosis, as suggested by Olson & Segre[7], then a more favourable environment than the one described here for sheep to sheep transmission of such an agent would be hard to find.

It may be concluded that the prompt cessation of mortality in this outbreak was linked to the measures taken to reduce the highly stressful situation to which the sheep were subjected and not to any significant reduction in the infectivity of the environment.

ACKNOWLEDGEMENT

Our thanks to Dr D.T. Longland for referring this outbreak to the laboratory.

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THE NUMBER AND LOCATION OF AIR SACS IN BROILER CHICKENS AND THE IMPLICATION IN *ESCHERICHIA COLI* INFECTION

J.R. MITCHELL*

ABSTRACT: Mitchell J.R. **The number and location of air sacs in broiler chickens and the implication in *Escherichia coli* infection.** *Journal of the South African Veterinary Association* (1984) 55 No. 2, 57-60 (En). Department of Anatomy, Faculty of Veterinary Science, Medical University of Southern Africa, P.O. Box 217, 0204 Medunsa, Republic of South Africa.

Broiler chicken carcasses were injected with latex to determine the number and location of the air sacs and the presence of diverticula. The adverse affect of *Escherichia coli* air sacculitis spreading into the diverticula is discussed.

Key words: Broiler chicken, air sacs, *Escherichia coli*.

INTRODUCTION

The existence of air sacs in birds has been known for a long time, the first description being attributed to Harvey[7] in 1657. Hunter[8] in 1774 moved a step forward when he experimentally cut the humerus of a living fowl and ligated the trachea, and found that respiration could continue, the air would pass to and from the lungs via a canal in bone. Campana[4] in 1875 was the first to measure the capacity of an air sac which he called "interclavicular". His findings that on inspiration the capacity of the interclavicular air sac was 20 ml was accepted until 1942 when Zeuthen[12] contradicted the findings of Campana by stating that the capacity of the interclavicular sac was 9 ml. The multitude of investigations which followed have failed to bring about agreement on the capacity and the number of air sacs in various species. The differences in findings could probably be attributed to the variety of species used for research. The matter was further complicated by the innumerable terminologies used until Baumel et al.[2] in 1979 standardised some of the terms.

Whilst the recognised functions of the air sacs in birds are acknowledged, it will also be true to say that in broiler chickens such functions will obviously be somewhat limited. In terms of commercial production and their susceptability to infection, the air sacs in them may even be regarded as a handicap. The present work is therefore confined to establishing the number and the location of the air sacs in broiler chickens and the implication of *Escherichia coli* affecting them.

MATERIALS AND METHODS

The work was conducted on 30 carcasses of broiler chickens 45-49 days old with an average body mass of 1,5 kg, supplied by the industry as dead on arrival. The carcasses were injected with latex and examined in groups of 10 over a period of a few months. In addition 4 adult fowls, 2 males and 2 females were used as controls. For the recording of results the sex of the 30 broiler chickens was disregarded.

The commercial latex injected, was coloured by the addition of red dye and a quantity of ammonia. The technique of injection and the amount of latex used were established by trial and error. The incision was made on the right side of the neck, cranial to the crop, the trachea exposed and freed from the oesophagus. A loop of string was applied and a cut of about 1 cm in the trachea was made cranially to the loop. A nozzle of a 50 ml syringe filled with latex was inserted into the trachea and the latex injected without unnecessary pressure. During the refilling of the syringe the cut was kept closed with an artery forceps. Upon completion of injection, the trachea was ligated with the string already in place.

The injected carcasses were marked and stored at a temperature of 1°C for periods varying from 4-7 d before dissection was carried out. The amount of latex injected varied from 50-150 ml; the optimum was found to be 100 ml. Excessive amount of latex required application of pressure during injection which frequently resulted in rupture of some air sacs. The latex did not solidify at the same time in all the air sacs. There appeared to be some factors which may delay the action; moisture of the viscera in the abdominal air sacs is suspected as being one of the delaying factors.

The examination consisted of exposing all latex filled air sacs and diverticula by careful dissection.

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RESULTS AND DISCUSSION

Number of air sacs and denomination

The observation by King[10] that there are 8 air sacs in domestic fowl was confirmed in the broiler chickens but the precise location and diverticular extensions found did not always agree with that previously described.

The air sacs were named in accordance with the terminology suggested by Baumel et al.[2] and are set out in Column I, previously used terms are listed in Column II of Table 1.

Table 1: TERMINOLOGY OF AIR SACS

Column I	Column II
Single *saccus cervicalis*	superior anterior[4] subbronchial[9]
Single *saccus clavicularis*	superior anterior[4] anterior intermediate[9] interclavicular[1]
Paired *sacci thoracici craniales*	middle superior[3] anterior intermediate[8] anterior thoracic[1]
Paired *sacci thoracici caudales*	middle inferior[4] posterior intermediate[9] posterior thoracic[1]
Paired *sacci abdominales*	inferior[4] posterior[6]

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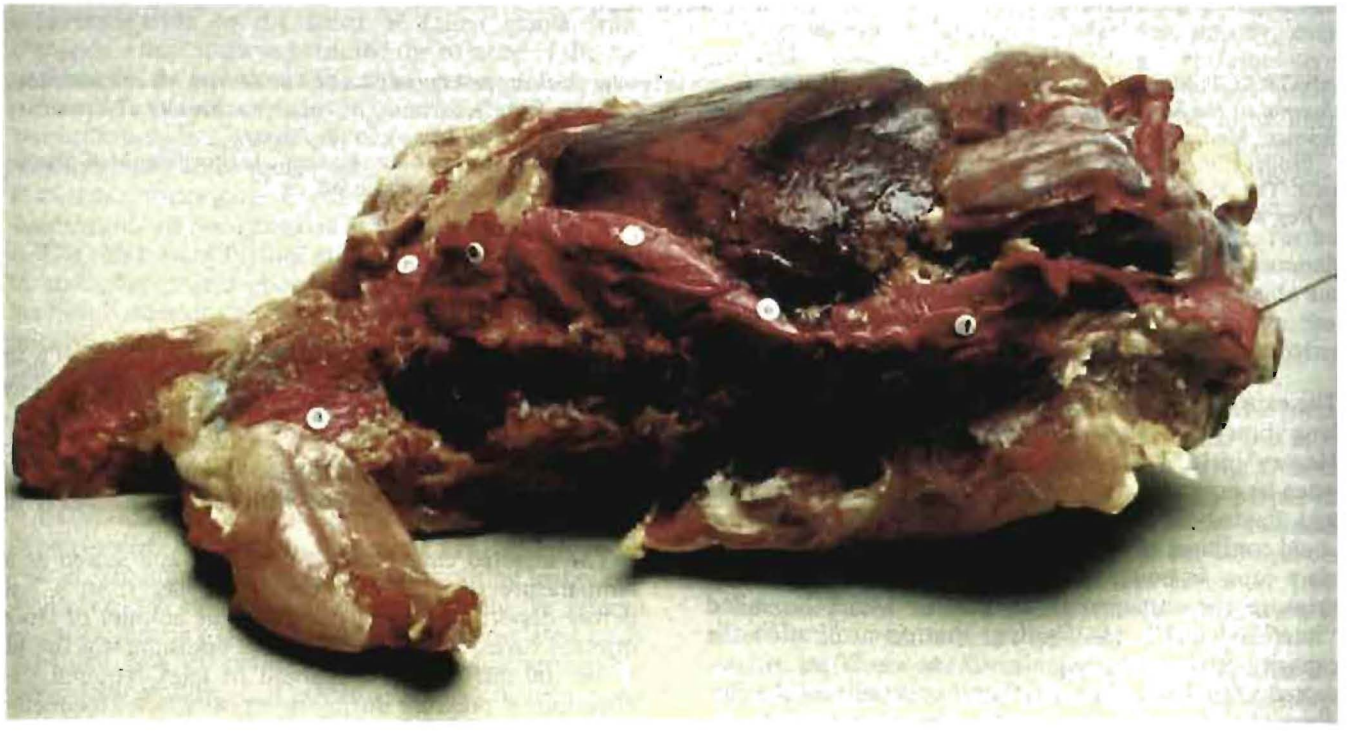

Fig 1 Lateroventral view of the air sacs in a broiler chicken: (a) humeral diverticulum, (b) sterno-cardiac or main part of *saccus clavicularis*, (c) costal diverticulum, (d) right *saccus thoracicus* cranialis, (e) right *saccus thoracicus caudalis*, (f) *saccus abdominalis*.

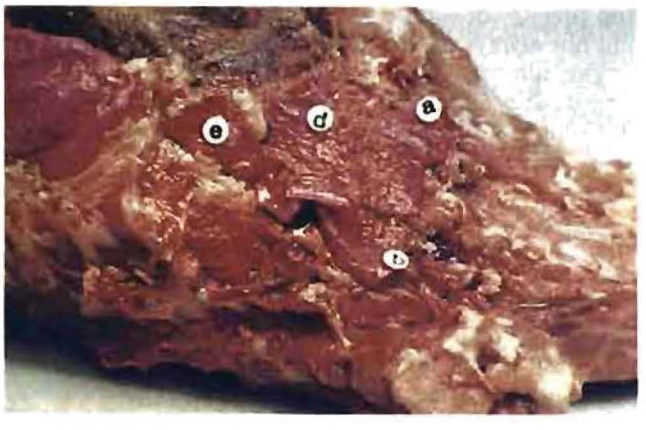

Fig 2 Lateral view of the thoraco-cervical region, (left side): (a) *saccus cervicalis (pars mediana)* (b) humeral diverticulum, (d) *saccus clavicularis* (sterno-cardiac part), (e) costal diverticulum.

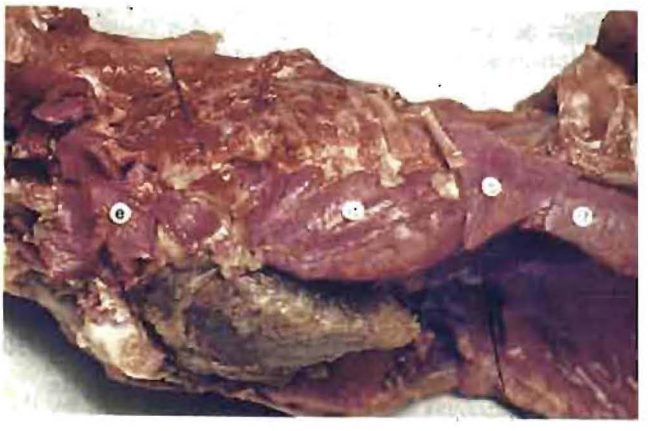

Fig 3 Lateral view of the thoracic region, (left side): (a) *saccus abdominalis* (b) *saccus thoracicus caudalis*, (d) *saccus thoracicus cranialis*, (e) costal diverticulum of *saccus clavicularis*. (The heart showing fibrinous pericarditis due to *E. coli*.)

Location of air sacs and diverticula

The location of the air sacs and their relative positions are described below. The air sacs being fully distended with latex may vary somewhat from the position in live birds.

The *saccus cervicalis* is located in the neck region, extending dorsally along the muscles of the vertebral column. Ventrally it lies upon the clavicular air sac, caudally it extends beyond the crop and between the lungs and laterally it is in contact with the lungs and cervical muscles. Cranially it appears to extend to the 6th or 7th cervical vertebrae corresponding to the bend in the neck. Regarding its cranial extention, it was not possible to confirm that all the cervical vertebrae, except for the first 2, are pneumatised. This could be as a result of an inadequate technique employed.

The diverticula of the cervical air sac have been referred to as the *diverticula vertebralia*[6], *diverticula intermuscularia*[5] and the *diverticula subcutanea*[10].

King[11] also refers to interspinal and extraspinal diverticula. As all the above do not necessarily refer to *Gallus domesticus* nor to any specified species, the present work recognises 3 parts of the *saccus cervicalis* in broiler chickens. The *pars mediana* or median part located as described above, the *diverticula transversales vertebrales* passing on each side through a *foramen transversarium* and the *diverticula canales vertebrales* within the vertebral canal.

In order to reach the transverse foramen and the vertebral canal, the diverticula from the *pars mediana* must pass along the muscles and any other structures; to give those parts special names is considered superfluous.

The *saccus clavicularis* is located at the thoracic inlet. Dorsally it is in contact with the cervical air sac and the muscles of the vertebral column. Ventrally it is in contact with the crop and lungs. Caudally it extends to the sternal plate, heart and hilus of the lung. Laterally it is bound by the bones of pectoral girdle and ribs. The oesophagus, trachea, blood vessels and nerves lie between the cervical and clavicular air sacs and do not penetrate the air sacs but lie within an invagination of the clavicular air sac.

For the sake of simplicity it is considered unnecessary to recognise the intrathoracic and extrathoracic parts as described in the literature. The cardiac part seems descriptive enough to indicate that it lies intrathoracically in contrast to the humeral diverticulum which lies extrathoracically.

The following parts and diverticula of the clavicular air sacs are recognised: sterno-cardiac or median part; humeral diverticulum which on leaving the sterno-cardiac part, runs cranioventrally towards the *foramen triosseum*. A branch enters the humerous through *foramen pneumaticum* located in the *fossa pneumatica* on the medioventral surface of the proximal extremity of the humerus. The latex penetrated the proximal third of the humerus; and the costal diverticulum often referred to as axillary diverticulum which pneumatises ribs and coracoid.

The *sacci thoracici craniales* have no diverticula and are related on each side to the ribs laterally, to the lungs and *septum horizontale* dorsally, to the heart medioventrally and to the proventriculus caudally. The cranial thoracic air sacs are overlapped cranially by the clavicular air sac and caudally by the caudal thoracic air sacs.

The *sacci thoracici caudales* have no diverticula and are attached to the body wall by their whole lateral surface. They are in contact cranially with the cranial thoracic air sacs, medially with the liver, laterally with ribs and caudally with the abdominal air sacs. Because the caudal thoracic air sacs overlap the cranial thoracic and abnormal air sacs, they have no contact with the viscera.

Each *saccus abdominalis* has a body and diverticula, the right one being slightly larger than the left one. They are located in the abdominal cavity. Cranially the air sacs are in contact with the liver, dorsally they adhere to the body wall, laterally to the caudal thoracic air sacs and the abdominal muscles, caudally they reach the pelvis. Medially they are in contact with the viscera and the mesentery, as well as with the gonads. They balloon between the intestines. The following parts of the abdominal air sacs are recognised: *saccus abdominalis* (main body); *diverticula perirenalia*; and *diverticula femoralia*.

Only the femoral diverticulum is considered to be of importance from a disease point of view. It leaves the main abdominal air sac through the rim of the *tuberculum preacetabulare*[2] (syn. *processus pectinealis)* and then almost surrounds the acetabulum, extending to the neck of the femur. The *tuberculum preacetabulare* is formed by the ilium and lies cranially to the acetabulum. The synonym *processus pectinealis* probably derived from the insertion of the *musculus pectineus* in mammals, which is equivalent to the *musculus ambiens* in birds.

Implication in *Escherichia coli infection*

Among the organisms affecting the air sacs, the ubiquitous *E. coli* appears to be most common. It produces a fibrinopurulent exudate which cannot be removed from the air sacs by the body defence mechanisms but it is removed during the commercial process of evisceration. Within certain diverticula, however, this exudate becomes imprisoned and cannot be removed, even with the aid of instruments such as the lung gun. The diverticula considered as most important in this respect are the humeral diverticulum which actually enters the humerus, and the femoral diverticulum around the acetabulum. These diverticula are not exposed during routine commercial processing and form part of the recognised poultry cuts. There are eight recognised air sacs in broiler poultry, two are single and three are paired. Although the aim of this work was to establish the number and location of the air sacs, the brief reference to the function of the air sacs becomes inevitable in the light of their implication in diseases. The air sacs in broiler poultry have probably retained the nominal functions only, forming protective air pockets around the viscera thus preventing the loss of heat. In the case of the abdominal air sacs they exert pressure upon the intestines thus helping to evacuate the faeces.

From the industry point of view the disadvantage of the presence of air sacs in broiler poultry is manifested by the undesirable spread of infection into commercially valuable parts. The presence of exudate within the clavicular and abdominal air sacs of broilers will render such carcasses unsuitable for those cuts. The frequently used argument that *E. coli* in this instance is non-pathogenic to man contradicts the basic principle of food hygiene and should not be entertained. There is an urgent need for further research in this field and this can only be

achieved by the combined efforts of the fast growing broiler industry and the guardians of our public health. A sound code of practice for the broiler processing industry based on scientific data rather than on the legacies inherited from the red meat industry could do nothing but good.

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BOOK REVIEW — BOEKRESENSIE

GENERAL VETERINARY PATHOLOGY

R.G. THOMSON

2nd Edn. W.B. Saunders Company, Philadelphia. 1984 pp xii and 463, Figures 571, Tables 22
ISBN 0-7216-8851-9 R72,24

As a textbook intended for introduction of pathology to the veterinary undergraduate student, this book fulfills the criterion admirably. A standardised format, similar to the first edition, has been adopted comprising seven chapters and four appendices. An introductory chapter is followed by chapters dealing with Degeneration and Necrosis, Circulatory Disturbances, Inflammation and Repair, Disturbances of Growth, Neoplasia and Host-parasite Relationships. The appendices include aspects which cover the processing of tissues, examination and naming of lesions, historical perspectives of pathology, Greek and Latin roots and affixes and lastly, the learning requirements and objectives for general pathology within the context of the veterinary curriculum.

The text is clear and concise. Indeed, the ruthless omission of all unnecessary detail is a noteworthy feature of this textbook. If any criticism may be levelled it is with regard to the lack of detail. Not insofar as the students being expected to know the detail but because greater detail, by means of explanation, may permit a better understanding of the dynamic processes of disease states. This minor failing is offset to a large extent by the selected list of references provided at the end of each chapter for the more enquiring mind. The outstanding feature of this book, however, is the photographic series employed to illustrate almost every facet in the text. Both the author and the publisher are to be congratulated on this aspect; the former for their selection and the latter for the high standard of their reproduction. Factual errors are few and minor. (It was learnt with some dismay that facial eczema of sheep follows ingestion of spores from *Sporidesmium bakeri*.) Although dominated by a no-nonsese approach, the langauge is, in general, easy to follow and understand.

Whilst obviously reflecting the personal concerns of the author, the appendices provide an interesting adjunct to the text. The last appendix, in particular, will be most useful to the undergraduate.

In spite of a price which may appear prohibitive this book is recommended without hesitation as a standard text book in general veterinary pathology.

J.W. Nesbit

PREVALENCE AND TYPES OF BACTERIA ASSOCIATED WITH SUBCLINICAL MASTITIS IN BLOEMFONTEIN DAIRY HERDS

RIANA SWARTZ*, P.J. JOOSTE* and J.C. NOVELLO*

ABSTRACT: Swartz Riana; Jooste P.J.; Novello J.C. **Prevalence and types of bacteria associated with subclinical mastitis in Bloemfontein dairy herds.** *Journal of the South African Veterinary Association* (1984) 55 No. 2, 61-64 (En). Department of Dairy Science, Faculty of Agriculture, University of the Orange Free State, P.O. Box 339, 9300 Bloemfontein, Republic of South Africa.

Bacteria associated with subclinical mastitis were isolated from machine-milked dairy herds in the Bloemfontein area supplying fresh milk during the period July to December 1980. The 151 quarter milk samples examined, were also subjected to somatic cell counts. Identification of the isolated bacterial strains showed that *Staphylococcus aureus* was the dominant mastitis-associated organism, constituting 66,4 % of all bacteria isolated. Compared with other recent mastitis surveys a low prevalence of classical mastitis streptococci (0,7 %) and of Gram-negative bacterial infections (6,3 %) was encountered. The Gram-negative bacteria were almost invariably isolated from neglected herds in which the cows were generally in poor condition and the hygienic measures employed were totally inadequate. Other bacterial strains isolated included *Corynebacterium bovis* (6,3 %) and the coagulase negative staphylococci (11,0 %). The high somatic cell counts of the quarter milk samples yielding *S. aureus*, the mastitis streptococci and the Gram-negative bacteria suggested a major pathogenic role for these isolates. The frequent occurrence of *C. bovis* strains and coagulase negative staphylococci in samples with high somatic cell counts similarly suggested that these organisms were more pathogenic than is generally assumed.

Key words: bovine mastitis, dairy herds, mastitis associated bacteria, *Staphylococcus aureus*, somatic cell counts.

INTRODUCTION

Despite the large amount of world literature on bovine mastitis, information on many aspects of this important disease in South Africa is sorely lacking. Very little, for example, has been published recently on the bacteriology of mastitis, especially subclinical mastitis, in the various geographical areas of South Africa. This situation, however, exists worldwide and is not limited to this country. Dodd[6] is of the opinion that there are few countries that have reliable information on the proportion of cows with udders infected by the various major mastitis pathogens.

Studies relating to mastitis in Southern Africa include that of Crewe[4] who examined the prevalence and types of mastitis pathogens in Pretoria herds. Van den Heever & Giesecke[18] and Giesecke et al.[9] published information relating to mastitis in various machine-milked and hand-milked dairy herds. More recently a survey on clinical mastitis was conducted in dairy herds around Bulawayo[3]. With a view to supplementing existing data on the aetiology of mastitis in Southern Africa it was decided to initiate a survey in the Bloemfontein area. During the period July to December 1980 various aspects of mastitis were investigated in fresh milk dairy herds in the Bloemfontein district. The main objective, however, was to determine the types of bacteria associated with mastitis in udder quarters.

MATERIALS AND METHODS

Collection of milk samples

At the time of the survey there were 105 fresh milk producers in the Bloemfontein district. Twenty herds, i.e. 19 %, were included in a statistically representative test sample. The test sample was distributed on a geographical basis to include all farming and climatological conditions in the area. All the herds investigated were machine-milked.

All cows in the herds were subjected to preliminary testing with the California mastitis test (CMT). Milk samples were aseptically drawn from 151 CMT positive udder quarters into sterile containers according to the following procedure: Udders were washed in running water and dried with disposable paper towels. After the teat ends had been thoroughly rubbed with pledgets moistened with 70 % ethanol, they were immersed in the same concentration of ethanol for approximately 1 min. The milk sample was taken after evaporation of the alcohol and was subsequently stored at 4°C. Somatic cell counts and bacteriological examinations were performed within 24 h.

Bacteriological examination and identification of isolates

A 0,1 ml aliquot of each milk sample was plated aseptically, in duplicate, on Difco tryptose blood agar containing 5 % sheep blood. One plate was incubated aerobically and the other anaerobically at 37°C for 24 to 48 h. Anaerobic conditions were produced in a glass anaerobic jar using an Oxoid gas generating kit (Protea Laboratory Services, P O Box 5598, Johannesburg, 2000).

Representative colonies of the dominant bacterial flora that developed on the plates were isolated and purified by streaking on fresh blood agar plates. Bacterial isolates were identified according to Harrigan & McCance[10]. The staphylococci were divided into two groups viz. coagulase negative and coagulase postive, using human plasma[15 16], those in the coagulase positive group were regarded as *Staphylococcus aureus*. Enterobacteria were identified using the commercial API 20 E system (Path-Ident, P.O. Box 27202, Benrose, 2011). *Streptococcus* spp. were indentified using the following tests:

(i) API Strep system (Path-Ident, P.O. Box 27202, Benrose, 2011)
(ii) Lancefield grouping using the Streptex system (Wellcome Reagents, P.O. Box 653, Kempton Park, 1620).
(ii) The CAMP test[5].
(iv) Colonial morphology on sheep blood and Oxoid Edward's agar plates.

Coryneform rods were identified by means of catalase and urease production, growth in Tween 80 agar and the presence or absence of haemolysis on sheep blood agar[10].

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0038-2809/84/02/0061-0064

The identified isolates were subsequently lyophilized and stored.

Somatic cell counting

Somatic cell counting was done in triplicate using a Model FN Coulter Electronic Particle Counter with a 70 μm aperture. Milk samples were prepared as described by Tolle et al.[17], diluted with a Coulter Dual Diluter and analyzed using the standard commercial reagents and methods.

RESULTS

Bacteria were isolated from a total of 138 of the 151 milk samples tested. Five of these yielded mixed cultures containing 2 types of organisms per sample resulting in a total of 143 bacterial isolates. No bacteria could be cultured from the remaining 12 samples. No anaerobic organisms were isolated under the cultural conditions applied in this study. One sample yielded 6 different bacterial isolates. The samples was however regarded as being contaminated from external sources and no isolates were studied.

The main primary udder pathogens[14] (Table 1) were responsible for 73,4 % of all organisms isolated. This group consisted of *S. aureus* (66,4 %) and the mastitis streptococci (7,0 %), the latter group consisting of *S. agalactiae, S. dysgalactiae* and *S. uberis*. The secondary udder pathogens[14], i.e. *Corynebacterium bovis* (6,3 %) and the coagulase negative staphylococci (11,9 %) totalled 18,2 %. Gram-negative organisms (Enterobacteria and *Pseudomonas* spp. etc.) made up 6,3 % of the bacteria isolated.

In Table 2 an attempt has been made to relate somatic cell counts of individual quarter milk samples to specific pathogens isolated from the same samples. A clear relationship exists between high cell counts and the presence of specific pathogens. More than 80 % of the samples yielding *S. aureus* and streptococci had cell counts exceeding 10^6/ml. All milk samples yielding *P. aeruginosa* and Enterobacteria also fell into this somatic cell count category. In the case of the coagulase negative staphylococci and *C. bovis*, 55 % and 33 % of the samples respectively fell into this category.

Somatic cell counts and bacteriological composition of quarters yielding mixed infections are shown in Table 3. *S. aureus* was also present in all 5 mixed infections. In all but one sample the cell counts were high, having more than 10^6 cells/ml. Two of these samples were taken from the same herd, i.e. samples 2 and 3.

Table 3: BACTERIA INVOLVED IN "MIXED UDDER INFECTIONS"

Quarter milk sample no.	Bacteria identified	Somatic cell count/ml
1	*S. aureus* Coagulase negative staphylococci	96 00
2	*S. aureus* *S. agalactiae*	7 362 000
3	*S. aureus* *C. bovis*	1 534 000
4	*S. aureus* *S. uberis*	4 781 000
5	*S. aureus* *S. dysgalactiae*	6 069 000

The somatic cell counts of 10 of the 12 samples from which no bacteria could be isolated, are given in Table 4. Seven of the samples had counts exceeding 10^6/ml. Two of the remaining samples had counts exceeding 500 000/ml. It is of interest to note that 5 of the above samples were taken from the same herd i.e. samples 4-8.

Table 4: SOMATIC CELL COUNTS OF CMT POSITIVE SAMPLES FROM WHICH NO BACTERIA WERE CULTURED

Quarter milk sample no.	Somatic cell count/ml
1	2 417 000
2	4 112 000
3	71 000
4	1 078 000
5	519 000
6	263 000
7	711 000
8	1 149 000
9	1 377 000
10	16 785 000
Average	2 848 200

Table 1: BACTERIA ASSOCIATED WITH SUBCLINICAL MASTITIS

Bacterial species	Number of strains isolated	% of total
Staphylococcus aureus	95	66,4
Coagulase negative staphylococci	17	11,9
Streptococcus dysgalactiae	5	3,5
S. agalactiae	4	2,8
S. bovis	2	1,4
S. uberis	1	0,7
Corynebacterium bovis	9	6,3
Serratia marcescens	3	2,1
Klebsiella oxytoca	1	0,7
Pseudomonas aeruginosa	5	3,5
Acinetobacter sp.	1	0,7

Table 2: DISTRIBUTION OF SOMATIC CELL COUNTS IN UDDER QUARTER MILK YIELDING POSSIBLE PATHOGENS

Somatic cell count (thousands/ml)	*S. aureus* % (90 strains)	Coagulase negative staphylococci % (20 strains)	Streptococci % (10 strains)	*C. bovis* % (9 strains)	Enterobacteria % (4 strains)	*P. aeruginosa* % (4 strains)
0 – 300	7	20	0	44	0	0
301 – 500	4	15	0	11	0	0
501 – 750	4	10	0	11	0	0
751 – 1000	3	0	10	0	0	0
1001 – 5000	48	20	40	33	50	25
5001 – 10000	12	25	20	0	0	25
>10000	21	10	30	0	50	50

DISCUSSION

The survey carried out in the Bloemfontein area was aimed mainly at determining the types of bacteria associated with subclinical mastitis. Somatic cell counts of all milk samples were carried out in order to allow more meaningful interpretation of bacteriological results.

It is of interest to compare the bacteriological results (Table 1) with those obtained by the more recent and applicable South African masititis surveys[3 4 9 18] and the recent national British survey[19]. The incidence of *S. aureus* strains in the current investigation was in agreement with the above mentioned local surveys and the British survey. Bryson & Hobbs[2] similarly found that 63 % of mastitis pathogens isolated from fresh milk herds in the Natal area were strains of *S. aureus*.

The high incidence of *S. aureus* infections found in Bloemfontein herds is undesirable since *S. aureus* infections can be notoriously resistant to antibiotic treatment. One reason for this resistance is the secretion of toxins by many *S. aureus* strains which enable the organisms to penetrate the duct walls of the udder. They become established in numerous foci that are walled off with fibrous tissue, thus creating a habitat virtually impenetrable to drugs[7]. This may lead to widespread therapeutic failures in mastitis control programmes.

The incidence figures for *Streptococcus* infections, excluding *S. bovis*, in the Bloemfontein herds (7 %) and the Pretoria herds (7 %)[4] are in good agreement with the British figure of 6 %[19]. *S. dysgalactiae* (3,5 %) followed by *S. agalactiae* (2,8 %) were the streptococcal species most frequently isolated (see Table 1). In the pre-antibiotic era *S. agalactiae* was the most dominant mastitis pathogen, but the incidence of *Streptococcus* infections declined drastically after the advent of antibiotic therapy.

There appeared to be a relatively low incidence of Gram-negative bacteria in the Bloemfontein area as indicated by the fact that only 6,3 % of all organisms isolated were Gram-negative. In the Pretoria survey of the machine-milked fresh milk herds[4] the corresponding figure was 36 % and in the hand-milked herds around Bulawayo, 33 %[3]. In the British survey the incidence was extremely low[19] as was the case in the survey by Van den Heever & Giesecke[18]. Most of the Gram-negative bacteria in the current survey were isolated from cows and herds in poor condition. The milking shed was likewise poorly tended and hygienic measures were inadequate. Three of the 4 Enterobacteria and one of the 5 *P. aeruginosa* strains were recovered from such herds. An additional 3 *P. aeruginosa* strains were isolated from a herd producing industrial milk. The cows in this herd were underfed and generally in a poor state of health. According to Jain[13] herds in which streptococcal and straphylococcal infections have been successfully reduced by means of effective antibiotic therapy and hygienic measures, become increasingly more susceptible to Gram-negative bacterial mastitis. Futhermore, teat dipping and dry cow therapy are ineffective against Gram-negative bacteria. In the current survey, however, staphylocci dominated and Gram-negatative infections did not present a significant problem.

Important points of similarity between the Bloemfontein and British surveys were the relatively high incidence of coagulase negative staphylococci and *C. bovis* strains (18,2 % in Bloemfontein herds and 43,5 % in British herds). Certain authors[8] are of the opinion that these organisms mostly arise from teat canal infections. From the results of the current study the impression was gained that the majority of these secondary pathogens were responsible for udder and not teat canal infections for the following reasons: (i) Most of the bacteria were isolated from high somatic cell count quarters; (ii) These organisms were only rarely involved in mixed infections; (iii) Milk samples were taken with extreme care. Neave[14] pointed out that contamination of samples by teat duct colonizing *C. bovis* and coagulase negative staphylococci can be greatly reduced by extended scrubbing of the teat end with disinfectant. This practice was strictly adhered to in the present study.

The relationship between high individual quarter milk somatic cell counts and the presence of specific pathogens is strongest in the case of quarters infected with *S. aureus*, streptococci, Enterobacteria and *P. aeruginosa* (Table 2). The level of increase of the somatic cell counts due to udder infections caused by these organisms emphasizes their strong pathogenic role in susceptible udders.

The International Dairy Federation[12] definition states that where the milk and udder are macroscopically normal a cell count of more than 50 000 cells/ml together with the presence of pathogenic bacteria signifies subclinical mastitis. In the current survey, however, it was noted that 65,0 % of coagulase negative staphylococci and 44 % of *C. bovis* were isolated from quarters with cell counts exceeding 50 000/ml. Although it is widely assumed[1] that *C. bovis* and coagulase negative staphylococci are rarely associated with clinical disease or a marked reduction in milk yield, results of this survey clearly indicate a more pathogenic role for these secondary udder pathogens than is generally assumed. This conclusion is in agreement with the new awareness of the important role that coagulase negative staphylococci play in human infections[11]. Very little information is available on the importance of secondary pathogens in mastitis and additional research into this aspect would be useful.

The 5 samples with "mixed infections" all contained *S. aureus* (Table 3). It can be speculated that these quarters were primarily infected by *S. aureus* and that these bacteria rendered the quarters more susceptible to infection by secondary pathogens. The low somatic cell count of sample 1, however, indicated that no active infection was present. The presence of *S. aureus* as well as coagulase negative staphylococci may indicate accidental contamination of the sample with common skin bacteria or a low grade test canal infection.

A total of 12 samples (8,4 %) failed to yield any bacteria in culture. It is widely reported[14] that pathogens are usually not recovered from 10 – 15 % sporadic clinical mastitis cases. The reasons for this phenomenon are however not clear.

CONCLUSIONS

S. aureus is by far the most dominant subclinical pathogen in Bloemfontein dairy herds. A surprisingly low incidence of streptococcal and Gram-negative infections were found. Somatic cell counts indicated a major pathogenic role for *S. aureus* streptococci and Gram-negative bacteria. The high incidence of *C. bovis* and coagulase negative staphylococci in quarters with high cell counts suggested that these organisms may be more

pathogenic than is generally assumed.

It is essential that more mastitis surveys be carried out in South African dairy herds to supplement existing information on the proportion of cows with udders infected by major mastitis pathogens. This information is necessary for assessing the mastitis position and future progress in mastitis control in this country.

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INFORMATION INLIGTING

RINDERPEST FIGHT IN AFRICA

Dr Brendan Halpin, a British specialist in animal diseases of the tropics who arrived in Nairobi at the beginning of January for a one-year tour sponsored by Britian's Overseas Development Administration, expects to travel in East, Central and West Africa during the current campaign against rinderpest. Together with 2 other veterinary officers from other countries, who are to work with him on the FAO and the Pan African Rinderpest Campaign, he will be collating information on control methods and future eradication plans.

A spokesman for the ODA in London said that Dr Halpin, who will be based in Nairobi, would help to identify areas which required concentrated local effort when the campaign gets underway.

He was also expected to contact government and other interested parties to explain the extent of the rinderpest threat to Africa and the potential danger it poses to the rest of the world.

Rinderpest, the "cattle plague" which reached Africa about a century ago, is transmitted by virus and claims many victims within 8 days of infection. Prevention centres on quarantine, slaughter, the use of disinfectants and immunisation with vaccines derived from viruses grown in hens' eggs.

Dr Halphin, who is a member of the Royal College of Veterinary Surgeons, has served in Nigeria and until recently he was an animal health adviser to the Overseas Development Administration.

ON THE USE OF OXYTETRACYCLINE IN REDUCING THE INCIDENCE OF METRITIS IN DAIRY COWS

C.W. MOORE*, J.J. MARNEWICK** and A.C. HENNING**

ABSTRACT: Moore C.W.; Marnewick J.J.; Henning A.C. **On the use of oxytetracycline in reducing the incidence of metritis in dairy cows.** *Journal of the South African Veterinary Association* (1984) 55 No. 2, 65-69 (En). P.O. Box 783720, 2146 Sandton, Republic of South Africa.

250 dairy cows were alternately either given no perinatal treatment or were given an intramuscular injection of oxytetracycline in a 2-pyrrolidone base at a dosage rate of 20mg/kg body mass.

The incidence of metritis in the untreated (control) group was 46 out of 120 cows (38,3%), while in the treated group there were 30 metritic cows out of 130 (23%) – a statistically significant difference.

Key words: metritis, oxytetracycline, dairy cows.

INTRODUCTION

There has in the past been some disagreement as to the bacterial content of the normal uterus[2 5 24] but recently consensus has been reached that the uterus postpartus is colonized by a variety of organisms[2 6 10 23 24].

Blood, foetal fluids and tissue debris remaining in the uterus shortly after birth serve as a favourable medium for bacterial growth, and this, coupled with the relaxation of the cervix allows the colonization of the uterus to take place[2 6].

Most individuals are able to "clean themselves" of these infections to a greater or lesser degree[2 6 10]. Metritis occurs, however, when this cleansing process does not take place and the "normal" colonization becomes a pathogenic process.

The reproductive efficiency of the South African dairy herd leaves much to be desired; with low conception rates, a high number of unnecessary inseminations or services to achieve conception and a long intercalving interval (Animal and Dairy Science Research Institute, personal communication). The cost of this management inefficiency is enormous to the individual farmer, not to mention the South African economy as a whole. One of the major problems contributing to this poor reproductive efficiency is the entity commonly known as "metritis".

Following various assertions in the literature[1 6 8 10 13 17 20] and the recommendations of colleagues in general practice, it was decided to examine the prophylactic effects of a perinatal injection of a long-acting oxytetracycline (OTC) on the occurrence of postpartum metritis in dairy cows.

MATERIALS AND METHODS

Four commercial dairy farms with grade Friesian cows in the Johannesburg area were selected for this study. These farms were chosen as being representative of dairy farming in the area and differed widely with regard to number of cows in milk, incidence of disease and standard of management.

Upon calving cows were alternatively assigned to either a treatment or control group. Cows in the treatment group received a deep intramuscular injection, into the *M. gluteus* of oxytetracycline in a 2-pyrrolidone base (Liquamycin^R LA, 200 mg OTC base per ml, Pfizer Laboratories) at a dosage rate of 20 mg OTC base/kg. The injection was administered by the farmer at partus or as soon thereafter as possible. The control group received no peripartum medication.

Individual animals with chronic recurrent mastitis or with a previous history of obstetrical problems were not allowed into the trial – no further selection constraints were imposed on the animals used in this study. On his routine monthly visit the veterinarian rectally examined the genitalia of all cows having calved since his last visit as well as those not yet confirmed pregnant. Any cow with a suspected metritis was examined by vaginal speculum. The criteria for identification of potential metritis cases were size and tone of uterus and cervix, presence or absence of lochia and if present the volume, colour and smell thereof. The size and tone of uterus and cervix were graded on the basis of the method described by Studer & Morrow[18]. However, their examinations occurred at 28 or 35 days post calving and those described in this trial from 7 days to 35 days post calving. To avoid subjective bias the veterinarian was unaware at each examination to which group (treated or control) the cow belonged.

No lochia or swabs were taken for culture or typing of any organisms present. Any animal which fell ill during the course of the trial (either treated or control groups) was treated according to the discretion of the veterinarian. Thus the only difference between the 2 groups of cows was the perinatal injection of OTC. A total of 250 cows were involved in this metritis study, divided between the 4 farms as set out in Table 1.

RESULTS

A multi-way frequency table analysis was done with treatments, mastitis, metritis and farms as the factors. Since no 3 factor associations were present any 2 factor relationships could be sought. However, no significant data were found with regard to the incidence of mastitis or its correlation to the treatment or control groups.

Table 1 shows the total incidence of metritis on each of the farms used in the study, irrespective of whether the cows were in the treatment or control groups. There was a marked difference in the incidence of metritis between the treatment and control groups. This is shown in Fig. 1 – the combined graph shows 30 out of 130 treated animals developed metritis (23,1%) while 46 out of 120 control animals developed metritis (38,3%). This difference in the incidence of metritis between treated and untreated animals is significant ($p = 0,0135$) based on the chi-square test.

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Table 1: INCIDENCE OF METRITIS ON 4 FARMS, AND NUMBER OF COWS INVOLVED (PERCENTAGE IN BRACKETS)

Farm	1	2	3	4	Combined
Metritis	20 (62,5)	8 (18,6)	26 (27,4)	22 (27,5)	76 (30,4)
No Metritis	12 (37,5)	35 (81,4)	69 (72,6)	58 (72,5)	174 (69,6)
Total	32 (100,0)	43 (100,0)	95 (100,0)	80 (100,0)	250 (100,0)

DISCUSSION

Uterine involution is considered to be almost complete by 21-30 days postpartum[2 6 11 19 21]. Uterine colonization by pathogenic bacteria can delay the return of the uterus to normal[2 6 19]. Tennant & Pedicord[19] quote Buch *et al.* that 'involution of the uterus is necessary for a cow to conceive readily after parturition'. They further cite Roberts, and Foote *et al.* who have shown that conception rates in cows with involuted uteri are higher than in cows with incompletely involuted uteri. Johanns, *et al.*[6] agree with these findings.

The majority of authors are in agreement that the metritis complex will have a deleterious effect on:

- ovarian activity and the development of follicles[3 6 11 14],
- conception and early embryonic survival[2 4 10 13 18 19],
- the number of services per conception[16 18 19],
- "days open" or intercalving interval[6 9 11 16 18 19 23], and thereby resulting in the term" repeat breeders"[5 20].

Various workers have isolated organisms from cases of septic bovine metritis and it seems that the most common causes thereof are *Corynebacterium pyogenes, Corynebacterium haemolyticum, Pasteurella* spp., *Proteus vulgaris,* streptococci, *E. coli, Staphylococcus aureus,* and *Micrococcus* spp., with *C. pyogenes* causing by far the most gross pathology and leading to the most serious metritis[2 4 5 10 18 21 23].

The susceptibility of these organisms to various antibiotics has been investigated. Miller *et al.*[10] found all organisms isolated to be susceptible to oxytetracycline and penicillin and that based on their susceptibility data, nitrofurazone and dihydrostreptomycin were not to be recommended for intrauterine infusion.

Their finding as to the susceptibility of *Staphylococcus aureus* to OTC differs from that given by Ziv[24] quoting Panagala & Barnum who determined that the only 2 *Staphylococcus aureus* found in 237 isolates of various bacterial species were both resistant to OTC. However, Morse *et al.*[12] determined that OTC was very effective in vitro against a wide range of organisms. Wetherill[21] also found that the above endometritic organisms were susceptible to OTC.

The present method of treating uterine infections is by flushing with various antimicrobial agents including antibiotics[8 10 13 17]. This local intrauterine (I U) therapy has been investigated by many authors. Masera *et al*[8]. used I U administration of OTC at 4-8 mg/kg and found high levels of OTC in the endometrium and uterine secretions. They and others[9 15] found the normal uterus to absorb OTC moderately to poorly but that absorption across the diseased endometrium was further impaired[15].

They did not find OTC in other parts of the genital

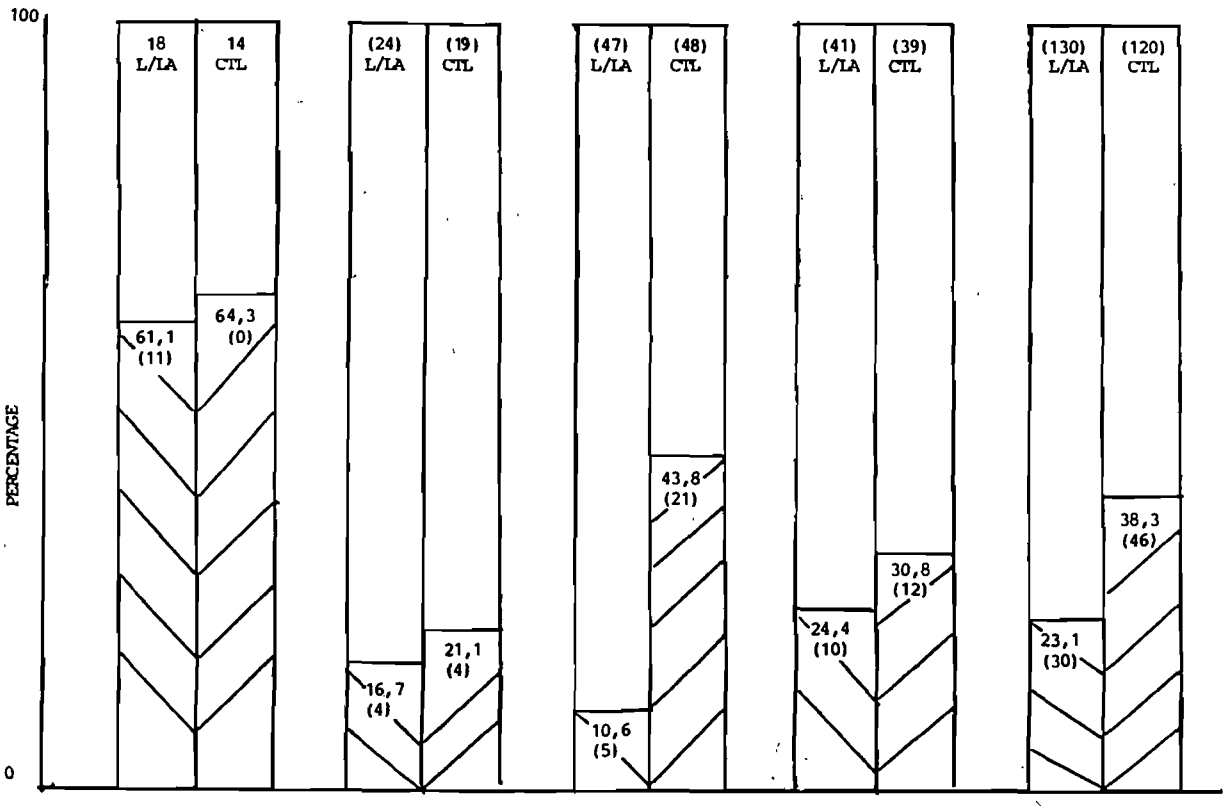

Fig. 1: Incidence of metritis in treated (L/LA) and control (CTL) animals (absolute numbers in brackets)

tract. Righter *et al.*[15] also showed that absorption immediately postpartum is poorest. Miller *et al.*[10] using 600 mg infusions could not detect OTC in the endometrium. They decided that OTC must be infused every 24 hours for a number of days to have a good effect. It is important to note the levels use in Masera *et al.*[8] (4 and 8 mg/kg) whereas the usual I U administration of oblets is 500 or 1 000 mg. In a dairy cow of 500 kg this is equivalent to a dose of 1-2 mg/kg.

It is further important to note that these workers[8 9 15] used propylene glycol based OTC, which is known to be irritant[15 17] (data on file, Pfizer Labs). It is not known to what extent this may affect these findings.

Although Ulberg *et al.*[20] found no advantage to I U infusions, this mode of treatment is common and is regarded as efficient and useful[10 13 17 21 24].

In the light of the above findings, in particular:

- that OTC has proved effective against the organisms causing metritis;
- that intra-uterine therapy is not always effective or warranted because of the possible introduction of contamination especially in the hands of the layman;
- that other parts of the female genital tract (besides the uterine lumen or endometrium) are often involved[8];
- that levels are not obtained in the endometrium after I.U. therapy except at very high dose;
- that levels are not maintained long enough[1 8];
- that in practical terms it is very seldom that the organism is isolated and a specific antibiogram performed; it was decided to investigate the use of a broad spectrum injectable for the prophylaxis of metritis in dairy cows.

Table 2: CONCENTRATIONS OF OTC IN COWS 24 HOURS AFTER INTRAMUSCULAR (IM) (8 mg/kg) AND INTRAUTERINE (IU) (4 mg/kg) ADMINISTRATION (MASERA ET AL.[8])

Tissue		I M (n = 3)	I U (n = 3)
Ovaries	(mcg/g)	1,50 ± 0,55	ND
Oviducts	(mcg/g)	1,53 ± 0,55	ND
Endometrium	(mcg/g)	1,87 ± 0,45	2,31 ± 2,66
Myometrium	(mcg/g)	1,30 ± 0,76	ND
Serosa	(mcg/g)	1,28 ± 0,62	ND
Cervix	(mcg/g)	1,81 ± 0,48	ND
Vagina	(mcg/g)	1,53 ± 0,36	ND
Uterine Secretion	(mcg/ml)	1,83 ± 1,47	4
Udder Secretion	(mcg/ml)	1,65 ± 0,04	ND
Pectoral Muscle	(mcg/g)	2,25 ± 0,07	ND
Thigh Muscle	(mcg/g)	2,60 ± 0,14	ND
Plasma	(mcg/ml)	0,72 ± 0,03	ND

Data expressed as mean ± SD
ND = Not Detected

Zemjanis[23] sees the problem as follows: "Furthermore, it must be understood that success in the treatment of uterine infection can be expected only if and when:

1. the causative agent is susceptible to the drug used,
2. the drug is used in effective concentration, and
3. the entire endometrium and the rest of the internal tubular tract are exposed to the drug."

Numerous workers have determined OTC in bovine serum after intravenous and intramuscular injection[1 8 22] (data on file Pfizer Laboratories). Yoder &

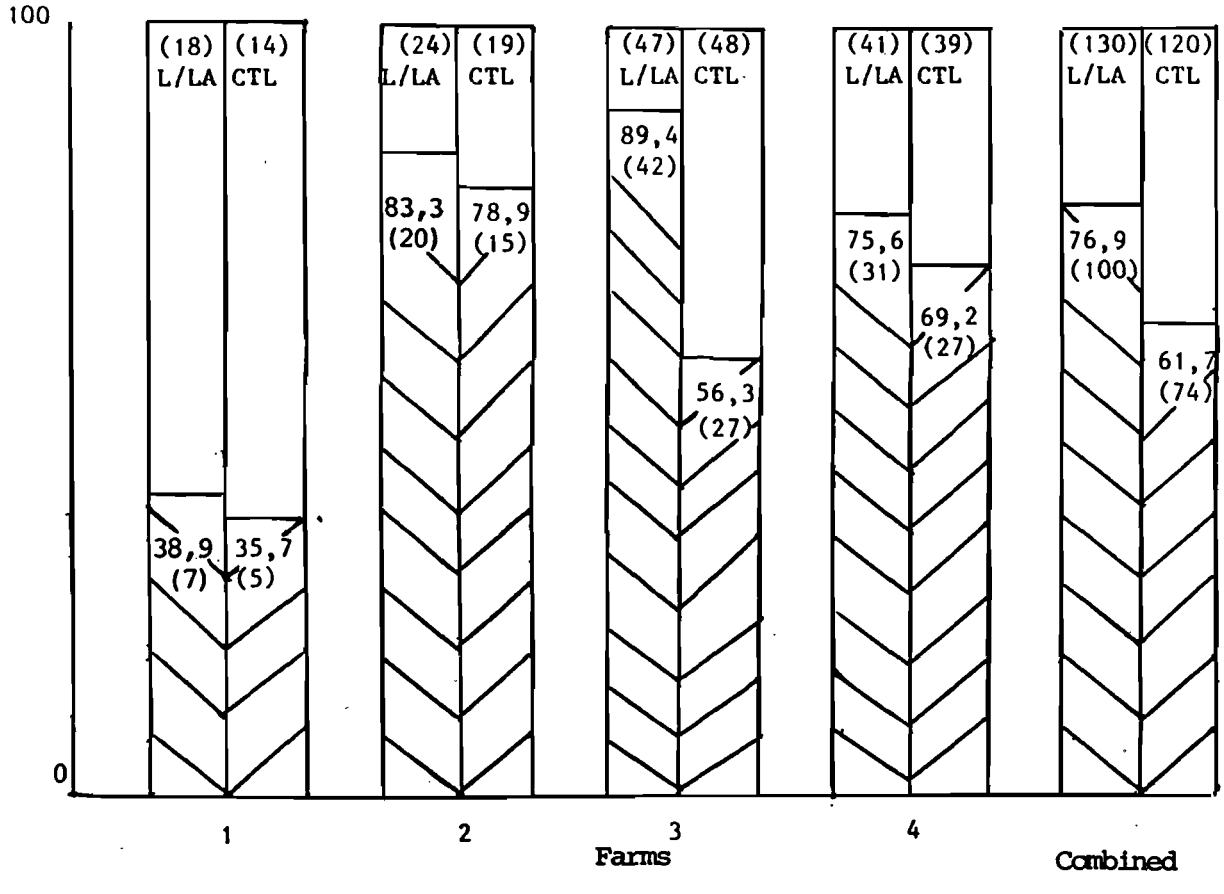

Fig. 2: Percentage of animals with no metritis in treated (L/LA) and control (CTL) groups (absolute numbers in brackets)

Packer[22] reported that blood levels in excess of 0,5 g/ml were maintained for longer than 24 hours after intramuscular dosage of approximately 2,2-4,4 mg/kg (probably using OTC powder in water, but not stated).

Masera *et al*[8], using a dose of 8 mg/kg of OTC intramuscularly, obtained serum levels of greater than 0,5 g/ml and tissue levels of greater than 1 g/g at all levels of the genital tract, in cows 24 hours after administration (see Table 2)

No work has thus far been performed to determine OTC levels in the genital tract of cows using the 2-pyrrolidone based product, Liquamycin^R Long-Acting. However, the drug consistently gives serum levels of greater than 0,5 g/ml for 72 hours with levels of greater than 0,2 g/ml in some animals to 120 hours and therefore it is reasonable to assume that at least equivalent levels of OTC would be maintained in the genital tract. Furthermore it has been shown that high blood levels are obtained within 30 minutes of intramuscular administration (Data on file Pfizer Laboratories).

An injectable administration route for OTC seems to have a good rationale as it has been shown that all parts of the female genital tract are involved in metritis (and not only the uterine lumen), and further that none or low levels of OTC are found even in the endometrium after intrauterine administration[10]. Similar results have been obtained using penicillin parenterally, but in this case levels disappeared within 12 hours post-injection[8].

As opposed to the opinion of Ziv[24], Masera et al.[8] and Bretzlaff *et al.*[1] found that OTC concentrations in the genital tract can exceed the concentrations in plasma following intramuscular and intravenous injections respectively.

The above clearly shows that a problem exists in the prevention of endometritis, and that the present mode of treatment can be improved upon.

This study showed an incidence of metritis of 38 % in "normal" dairy cows – the national average can be considered to be the same. Marnewick[7] found an average incidence of 56,6 % on 3 commercial diary farms. It has been shown by many workers that the metritis complex leads to an increased intercalving interval[6 9 11 16 18 19 23]. The average intercalving interval for 25 000-30 000 dairy cows in the Eastern Transvaal is given as 403 days (H. Lombard 1983 OTK, P.O. Box 100, Bethal, 2310, personal communication). The national average can be considered to be a similar figure.

It has not, as yet, been determined what the cost of this lengthened intercalving interval is to the country – however it seems certain that the cost would be enormous[3 14 19]. The costs would include: diminished milk production; fewer calves being produced, increased drug, costs, and most expensive of all – cow "losses" or replacement costs[3 14].

A further problem is that chronic endometritis is not palpable rectally[12], and thus prevents the timely diagnosis of the chronically endometritic cow which becomes a repeat breeder. Futhermore as cows become more productive they become more prone to disease and thus dairymen can expect to have an increased incidence of the metritis complex[12].

In the words of Morrow[11] when discussing the effects of postpartum disease – 'these results emphasize the importance of preventing postpartum diseases and of providing prompt treatment of those that occur. Additional examinations and treatment are especially helpful following retained foetal membranes and metritis in order to reduce infertility and to achieve satisfactory calving intervals'.

CONCLUSION

The findings of this study suggest that a peripartum injection of Liquamycin^R LA will reduce the incidence of metritis in a dairy herd.

Opinion from the literature is that systemic administration of antibacterials is superior to local application, provided the drug is found in sufficient concentrations in the female genital tract.

The authors would therefore suggest a peripartum injection of Liquamycin^R LA to all cows at a dosage rate of 20 mg/kg, and that in any cow developing a uterine bacterial infection, a combination of systemic and local (intrauterine) treatment be employed.

ACKNOWLEDGEMENTS

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BOOK REVIEW BOEKRESENSIE

DISEASES OF EXOTIC ANIMALS

JOEL D. WALLACH and WILLIAM J. BOEVER

1st Edn. W.B. Saunders Company, Philadelphia. 1983 ppxii and 1159. Price R248,00 (ISBN 0-7216-9105-6).

This book is comprehenisve and the objective of the authors obviously was to inlcude as many species as possible. This is verified by their own words – "It is our wish that this book provide a widespread understanding of the anatomy, physiology, diagnosis and treatment of the rainbow of species with which we share this planet". It has 1159 pages, many tables and hundreds of photos.

The authors are veterinarians who are well qualified to write on the subject as they both have gained extensive experience during their careers in game parks, zoos, aquaria, research institutes and laboratory animal centres.

Section I covers the mammals which *inter alia* includes the primates, rodents, lagomorphs, ruminants, felidae, canidae, viverridae, wild swine and bats. Section II deals with the game birds, water fowl, ratites, birds of prey and companion birds while section III concerns itself with the reptiles, amphibians and tropical fish.

The species featured in this book can be grouped into those that are less exotic and which are frequent inhabitants of zoos, laboratory animal centres or kept as unusual pets. They are the baboons, monkeys, rats, mice, gerbils, hamsters, fish, snakes, tortoises and the companion birds. The second category contains the real exotic species such as leopards, wolves, foxes, meerkats, kangeroos, bears and dolphins to name but a few.

This book deals with more than just the diseases of the exotic animals. Each section also contains taxonomic information and data on the sizes and weights of the animals. Tables with normal haematological, blood chemistry, blood gas and pH values are also included. The data given on housing, recommended cage sizes, anaesthesia, restraint, behaviour, training, longevity, nutrition and surgery are most valuable and those of us concerned with laboratory and zoo animals can benefit from the sections on abnormal behaviour psychopathology of animals in captivity. Also included is normal biological data such as rectal temperatures, heart and respiratory rates and tables indicating reproductive characteristics.

In each section on diseases the infectious, parasitic, neoplastic and non-infectious conditions are dealt with. It is unfortunate that only the most essential information on diseases is given and it will be necessary to consult other sources when more detail is needed. As an example I would like to point out that ectromelia (mouse pox) which is an important desease is dealt with in only 4 sentences. This criticism must not however be allowed to take too much merit from the book as a publication with its scope cannot posibly deal with everything in detail.

A further criticism is that some of the photos don't really justify insertion because they are indistinct while some can't convey the message that they are meant to because they are in black and white but are inserted nevertheless.

The appendix of this book contains very useful illustrated information on equipment for restraint, methods and equipment for chemical immobilization of animals as well as data on the composition of commercial diets available for various species.

This book will be most useful to veterinarians, biologists and research workers who have to care for exotic animals in zoos, laboratory animal centres, aquaria, research institutes and game parks. Veterinarians who are called upon to tend to exotic pets kept by hobbyists will also find it most useful.

W.A. De Klerk

IONIZED CALCIUM VERSUS TOTAL CALCIUM IN DAIRY COWS

J. DAUTH, M.J. DREYER and J.P. de CONING

ABSTRACT: Dauth J: Dreyer M.J. de Coning, J.P. **Ionized calcium versus total calcium in dairy cows.** *Journal of the South African Veterinary Association* (1984) 55 No. 2, 71-72 (En). Department of Chemical Pathology, Medical University of Southern Africa, Private Box 136, 0204 Medunsa, Republic of South Africa.

Ionized and total calcium levels were determined in 29 Friesian and 5 Drakensberger cows. One Friesian cow had parturient paresis and specimens were taken before and after treatment. It is suggested that due to the easy reliable way in which ionized calcium levels can now be determined this investigation should replace total calcium estimations in dairy cows presenting with parturient paresis. Ionized calcium levels serve as a valuable diagnostic aid but are also important to assess the effectivity of treatment for this condition.

Key words: Ionized calcium, total calcium, dairy cows, parturient hypocalciumia, milk fever.

INTRODUCTION

The biologically active portion of calcium in plasma is the ionized or free form.[3] The recent development of reliable calcium-selective electrodes now makes it possible to estimate this fraction in a semi-automated way. Due to an estimated overall incidence of 5 % of parturient hypocalcaemia or milk fever in dairy cows[2], 34 cows were examined and both the total calcium (ct Ca) and ionized calcium levels (cCa^{2+}) were established. A linear regression was calculated between the 2 parameters.

MATERIALS AND METHODS

An ICA1 ionized calcium analyzer (Radiometer A/S Copenhagen) was used to determine cCa^{2+} in fresh heparinized venous whole blood (VWB). Special heparin supplied with this instrument was used. The ICA1 employs 2 electrodes viz. one for determination of cCa^{2+} and one for pH. A microprocessor then converts the cCa^{2+} to cCa^{2+} at normal body pH (7,40).

Venous blood was also drawn into an unheparinized syringe and transferred to intergrated serum separator tubes (Corvac, St. Louis, Mo, USA). After separation these serum specimens were taken to the laboratory for ctCa estimations done on a Varian A.A. 1475 Series atomic obsorption spectrophotometer (AAS). The estimation of total protein and albumin levels in serum was carried out on a SMA II (Technicon Instruments Corporation, Tarrytown, N. York 10591).

Linear regression values were calculated by a Hewlett-Packard 9845 B computer (Hewlett-Packard, Covallis, Oregon 97330) to examine the possibility of a linear correlation between ctCa and cCa^{2+}.

RESULTS

The ICA1 which was previously evaluated under laboratory conditions[1] performed satisfactorily on location and 20 specimens per hour were analysed using 120 $\mu\ell$ of VWB.

cCa^{2+} normal ranges for cows (Drakensbergers and Friesians) were previously established and are between 1,18 – 1,35 mmol/ℓ for VWB. Serum ctCa levels are between 2,16 – 2,62 mmol/ℓ. Table 1 shows the cCa^{2+} and ctCa values of one Friesian cow with milk fever before and after treatment. As can be seen the initial cCa^{2+} level was dangerously low and it started falling again 8h after treatment even before the ctCa.

All the cows, including the one suffering from milk fever, had normal serum total protein and albumin levels.

A linear correlation between ctCa and cCa^{2+} was present (Fig. I) and the coefficient of correlation between the two parameters is 0,93.

DISCUSSION

The development of a reliable ionized calcium analyzer like the ICA1 may mark the end of total calcium estimations because:

1. There is an excellent correlation between the ionized and total calcium levels.
2. cCa^{2+} levels are only influenced by pH changes and to a lesser extent s-albumin levels.
3. A VWB pH is available with the cCa^{2+} levels which could also be of great value in detecting disturbances of the acid-base balance.
4. The success or failure of treatment for parturient paresis can be ascertained within 3 minutes of submitting VWB.
5. No special training is needed to operate this instrument.
6. Low or falling pre-partum cCa^{2+} levels could be detected in time especially in old cows or cows who previously developed milk fever.
7. The cost of the instrument and operational costs are not prohibitive.
8. The ICA1 is a sturdy instrument that can be transported without deleterious effects.

CONCLUSIONS

The establishment of cCa^{2+} in dairy cows can now be done reliably and with ease and it is suggested that it could be of great benefit to veterinary surgeons and other people involved in the diagnosis and treatment of milk fever, a disease with an extremely high incidence especially in certain breeds of dairy cows.

In contrast to this the determination of ctCa is a laborious procedure for which trained laboratory personnel and expensive equipment are mandatory. Futhermore, instananteous results cannot be obtained and the ctCa is dependant on a wide variety of factors of which s-albumin levels are extremely important. Also, the ctCa level alone is not of great help to calculate cCa^{2+} if s-albumin values and the plasma pH are not available.

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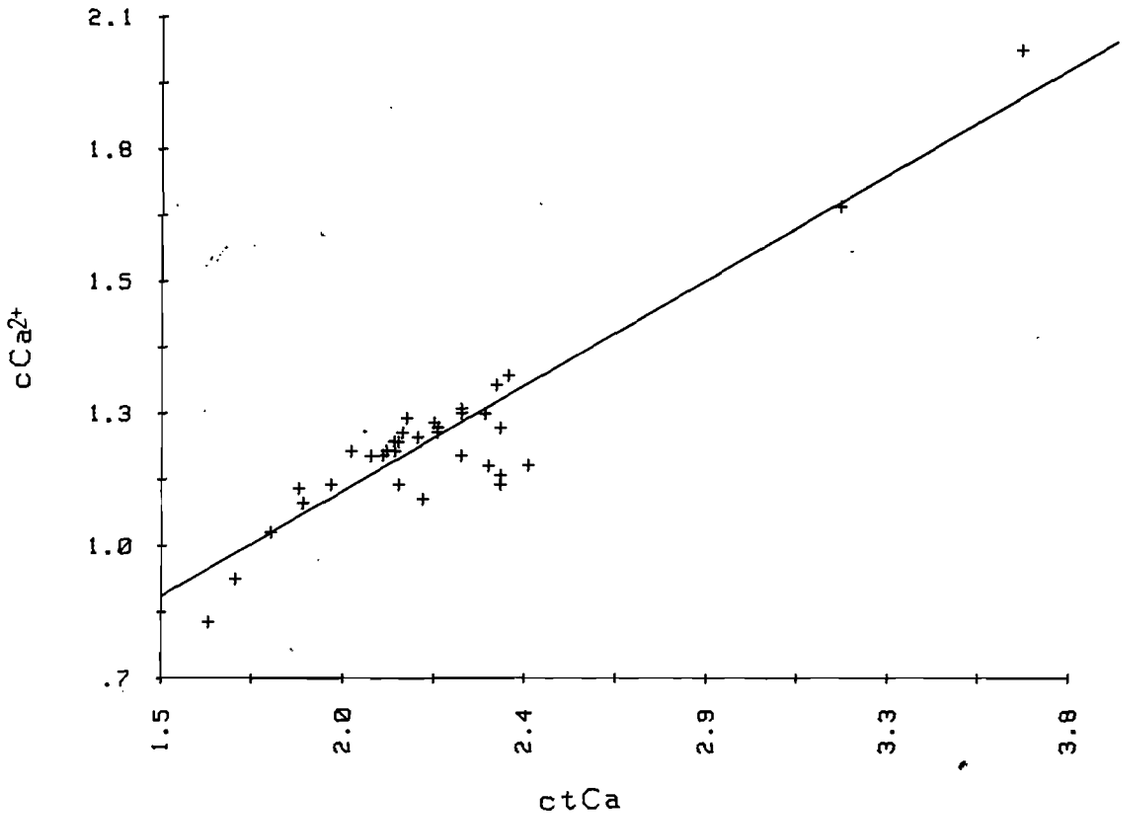

Fig. 1: Correlation between ctCa and cCa^{2+} in dairy cows

Table 1: ctCa AND cCa^{2+} VALUES OF A FRIESLAND COW WITH MILK FEVER BEFORE AND AFTER TREATMENT

	ct Ca mmol/l	c Ca^{2+} mmol/l	pH	c Ca^{2+} at pH 7.4 mmol/l
BEFORE TREATMENT	1.69	0.91	7.50	0.95
10min AFTER TREATMENT	3.68	2.03	7.48	2.11
4h AFTER TREATMENT	2.36	1.23	7.65	1.40
8h AFTER TREATMENT	2.26	1.17	7.62	1.31
12h AFTER TREATMENT	2.10	1.11	7.60	1.22
16h AFTER TREATMENT	2.06	1.17	7.49	1.23

ACKNOWLEDGEMENTS

We would like to thank Messrs. Radiometer A/S (Copenhagen) and Messrs. Medical Distributors (Johannesburg) for having made this study possible.

We would also like to thank Dr. Jan Marnewick for collecting the specimens.

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HELMINTH PARASITES OF GAME IN TRANSKEI

R.C. MARES, L. AMARAL and LURDES C. FACHADA*

ABSTRACT: Mares R.G.; Amaral L.; Fachada Lurdes C. **Helminth parasites of game in Transkei.** *Journal of the South African Veterinary Association* (1984) **55** No. 2, 73-74 (En). Umtata Veterinary Laboratory, Umtata, Republic of Transkei.

Opportunities have been taken to examine sundry game animals for parasites over the past 2 years. A host parasite check list is presented from which it may be noted that *Oesophagostomum columbianum* is recorded for the first time in the red hartebeest *Alcelaphus buselaphus* and *Haemochus bedfordi* for the first time in the eland *Taurotragus oryx.*

Key words: Game, eland, redhartebees, *Haemonchus bedfordi, Oesophagostomum columbianum,* Transkei

INTRODUCTION

The Republic of The Transkei, hereinafter referred to as Transkei, is a small territory bounded by Natal, Eastern Cape, the Drakensberg and the sea. Until 1976 the Veterinary Services were run from Pretoria, Republic of South Africa through a State Veterinarian usually resident in Umtata. With a view to encouraging tourism, small game reserves, or nature reserves, have been established. Although, like most of Africa, game once abounded, larger game had been hunted out so that animals listed in this communication had been re-introduced, mainly from South West Africa, in 1979 and 1980. Except for the bushbuck *Tragelaphus scriptus* and the zebra *Equus burchelli* the other game was culled by helicopter in the Mkambati Game Reserve on the Transkei coast at latitude 31°15′. On these 8 000 hectares of coastal sour veld the game has done so well that periodic culling is necessary. It was on the invitation of the management of the reserve that the opportunity was taken to make collections of parasites.

A search of departmental records in Umtata and of annual reports at Onderstepoort has not revealed any host parasite list for game, or indeed for domestic animals in Transkei. Any study of internal parasites in Africa, no matter how modest, owes a great deal to Mönnig, and his standard text book, now edited and expanded by Soulsby[10], is probably the most useful general reference. Mönnig's 1928 check list[4] already lists many species from the zebra but has few references to other game animals. Mönnig[6] was always active in seeking and recording new species in game. More recently, Horak[3] has listed the internal parasites of blesbok *Damaliscus dorcas phillipsi* and impala *Aepyceros melampus* and given their distribution in Southern Africa according to climate.

Because Round's check list[8] does not give *Oesophagostomum columbianum* as a parasite of the red hartebeest or *Haemonchus bedfordi* as a parasite of the eland it is thought that a presentation of these results may be of interest.

MATERIALS AND METHODS

The animals became available for examination mainly as the result of aerial culling from a helicopter. All animals were in good condition except the old red hartebeest and the Burchell's zebra. This latter died of old age. The following animals were examined: Blesbok, 8 young and 8 adults; Blue wildebeest *Connochaetes taurinus* 5 adults; Bushbuck, 1 adult; Eland, 2 adults; Gemsbok, *Oryx gazella* 3 adults; Red hartebeest, 8 adults; Burchell's zebra, 1 adult; Black-backed jackal *Canis mesomelas* bitch, 1 adult.

The material from the zebra and bushbuck was sent in by game wardens but that at Mkambati was obtained by one of us (L.A.) following the course of the helicopter on a motorbike, identifying the kill and directing selected specimens to the base camp for examination with limited facilities in the open air. The ingesta were washed from the intestines and examined in black painted trays from which the parasites were picked out with forceps and placed into physiological saline and thence into 70 % alcohol for subsequent identification in the laboratory. No attempt was made to count parasites so that the estimation of a heavy infestation was subjective only.

RESULTS

Haemonchus contortus infection in young blesbok and in the single old red hartebeest was considered heavy as were the ascarids in the zebra and the tapeworms in the jackal. Otherwise parasites were few and quite difficult to find.

Host-parasite Check List

Blesbok *(D. dorcas phillipsi)*
- *Haemonchus contortus*
- *Ostertagia ostertagi*
- *Cooperia* sp.
- *Monezia expansa*

Blue Wildebeest *(C. taurinus)*
- *Haemonchus contortus*
- *Avitellina* sp.

Bushbuck *(T. scriptus)*
- *Oesophagostomum radiatum*

Eland *(T. oryx)*
- *Haemonchus bedfordi*
- *Avitellina* sp.
- *Monezia* sp.

Gemsbok *(Oryx gazella)*
- *Haemonchus contortus*
- *Fasciola hepatica*

Red Hartebeest *(A. buselaphus)*
- *Haemonchus contortus*
- *Ostertagia circumcincta*
- *Trichostrongylus* sp.
- *Cooperia* sp.
- *Agriostomum cursoni*
- *Oesophagostomum columbianum*
- *Trichuris ovis*
- *Avitellina* sp.
- *Monezia* sp.

Burchell's zebra *(E. burchelli)*

*Umtata Veterinary Laboratory, Umtata, Republic of Transkei

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Parascaris equorum
Crossocephalus viviparus
Cyathostomum (Trichonema) sp.
Cylicocyclus (Trichonema) sp.
Cylindropharynx sp.
Triodontophorus sp.
Anoplocephala magna
Anaplocephala perfoliata
Black-backed jackal *(C. mesomelas)*
Dipylidium caninum
Taenia sp.

DISCUSSION

Sachs and his co-workers[9] gave a useful table of the spicule lengths and the distance from barb to tip of the spicules of eight species of *Haemonchus* found in domestic animals and game in Tanzania. The male *Haemonchus* from the eland gave spicule measurements corresponding to those given by these workers and Gibbons[2] for *Haemonchus bedfordi.* They considered that *H. bedfordi* was the commonest species after *H. contortus* and found it in wildebeest and Lichtenstein's hartebeest *A. lichtenstenii* but not in the eland. Only female *Agriostomum* sp. were found in the red hartebeest, so identification had to be based on the marked vulval protruberance, and absence of cervical groove as described by Mönnig[5] for *Agriostomum cursoni* and the fact that Round[8] does not list this for the red hartebeest. *Oesophagostomum columbianum* is easily recognised by the cervical alae and has not been recorded previously in this host (Round q.v.). Young[11], however, states that it infests 14 species of antelope, but does not list them. The differentiation of the species of *Trichuris* is difficult[1]. Accurate measurement of the long curving spicule and its bulb is almost impossible. *Trichuris ovis* have been previously recorded in the red hartebeest and this fact, along with vulval extrusion and some, admittedly very approximate, spicule measurement, led us to conclude that it was this species we had.

The question arises as to whether the parasites identified are original to Transkei or have been imported along with their hosts from South West Africa. One of us (R.G.M.) is presently engaged on a helminth survey of the sheep and cattle of the country and has found all the parasites listed with the exception of *Agriostomum* and *H. bedfordi.* All land now being used for game areas has, so far as is known, been used at some time or other for grazing domestic livestock.

Neitz[7] found *H. contortus* in the red hartebeest, blue wildebeest, blesbok, gemsbok and eland in South and South West Africa. The gemsbok he examined was a specimen from a zoo. Horak[3] lists *Haemonchus* for the blesbok but does not give it as distributed on the south coastal areas. He does not list *Ostertagia* or *Monezia* in blesbok.

The animals at Mkambati had been given "Thibenzole" impregnated mineral blocks as routine, but in spite of this the subjective impression of the worm burdens was that it was high in young and poor conditioned animals, and very low in good conditioned animals of mature age of all species. Old cattle examined at post mortem by one of us (R.G.M.) in Transkei have uniformly had a very low burden of parasites so it would appear as if the red hartebeest, like the sheep, does not have the ability to develop an immunity to helminths.

ACKNOWLEDGEMENTS

We thank the management and staff of Mkambati Game Reserve (Pty) Ltd. for the help and hospitality provided for most of the work.

Dr. R.D. Bigalke, Director of the Veterinary Research Institute, Onderstepoort and Professor R.K Reinecke of the Faculty of Veterinary Science, University of Pretoria are thanked for providing one of us (R.G.M.) with the facilities, reference works and technical asistance to confirm our provisional identificatons. Dr Anna Verster was particularly helpful and drew attention to the fact that *Oesophagostomum columbianum* had not been recorded previously in the red hartebeest. Dr Linda Gibbons of the Commonwealth Institute of Parasitology kindly checked the literature to confirm this. We are indebted to Mrs R.C. Scialdo-Krecek of the Faculty of Veterinary Science, University of Pretoria for identifying the zebra parasites.

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CASE REPORT GEVALVERSLAG

INANITION IN A DERBY ELAND DUE TO FOREIGN BODY ABOMASITIS

I.B.J.VAN RENSBURG * and H. EBEDES**

ABSTRACT: Van Rensburg I.B.J.; Ebedes H. **Inanition in a Derby eland due to foreign body abomasitis.** *Journal of the South African Veterinary Association* (1984) **55** No. 2, 75-76 (En). Department of Pathology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

An adult Derby eland bull, *Taurotragus derbianus* (Grey) kept in captivity showed wasting over a period of 3 months. It died from acute inhalation pneumonia while being transported in an immobilized state. Post mortem examination revealed a chronic ulcerative abomasitis. Histopathologically it was established that the abomasitis resulted from penetration of the mucosa by awns of plant material.

Key words: Derby eland, abomasitis, wasting, inanition.

INTRODUCTION

A 5-year old Derby eland bull, *Taurotragus derbianus* (Grey) in the National Zoological Gardens of Pretoria South Africa, was kept on a regular zoo antelope diet consisting of dried and fresh lucerne, antelope cubes (Epol Antelope Cubes, Epol Feeds, Pretoria) and sliced pumpkin.

Over a period of 3 months it was noticed that the animal was steadily losing condition. It was suspected that the animal was either not deriving adequate nourishment from the food provided or that it might be suffering from an erosion disease like tuberculosis. An intradermal tuberculin test for tuberculosis was performed under tranquillization with negative results. It was therefore decided to capture and remove him to the hospital for observation. For this purpose he was immobilized in 15 minutes by intramuscular injection via a dart containing a mixture of 4 mg Etorphine HCl + 100 mg Xylazine HCl and 50 mg Azaperone. This procedure initially went very well, but he died of asphyxiation caused by inhalation of rumen contents shortly after being loaded on a trailer while still prostrate.

A post mortem examination was carried out within 2 hours of the time of death.

Macroscopical findings

The eland was in a fairly good condition. It was cyanotic and showed moderate generalised venous congestion. The trachea and bronchial tree up to the level of the small bronchi were filled with rumen contents while the lungs showed generalised emphysema. The rumen was filled with a fine soft and sloppy almost watery content with a pH of 5,3. There was a marked chronic abomasitis evidenced by thickening of the mucosa with several large scars in the mucosa creating smooth fibrosed areas over large parts of the abomasal wall. Four small inspissated and calcified abscesses were found in the abomasal wall and rumenal lymph node. A small quantity of sand was present in the abomasum which was well filled with otherwise normal fairly dryish contents. There was a mild ascites, hydrothorax and hydropericardium, moderate nephrosis and a few jejunal ecchymoses present. A few *Rhipicephalus evertsi* ticks were found on the skin. The incisor teeth were overgrown and very sharp, and did not bite properly onto the dental pad. No internal parasites were found.

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**National Zoological Gardens, Pretoria.

Microscopical findings

Haematoxylin and eosin stained sections were prepared from several tissues and sections from the abscesses in the rumenal lymph node were also stained by the Ziehl-Neelsen method.

The most significant lesions were found in the abomasum where large areas of chronic ulcerative abomasitis were found. These areas were completely denuded of epithelium which was replaced by granulation tissue. In areas of scarring as well as in those where the abomasal mucosa was more intact, bits of plant material, probably grass awns, were embedded in the abomasal wall. Many of them were sharply pointed and some were surrounded by granulation, while around others not much of a reaction had been provoked.

The cause of the abscessation in the rumenal lumph node could not be determined, but no acid fast organisms could be demonstrated.

DISCUSSION

The inhalation of rumen contents during transport while under immobilization was probably predisposed to by the watery nature of the rumenal contents.

It is postulated that the reason for the gradual deterioration in the animal's condition was the chronic abomasitis. This apparently was the result of the awns penetrating the abomasal mucosa or lodging in the gastric pits and probably serving as portals of entry for bacterial and/or fungal organisms. These then resulted in foci of abomasitis which progressed to large areas of chronic ulceration and scarring.

Derby eland are very rare animals in Zoological Gardens. The Royal Zoological Society of Antwerp is the only other known zoo in the world that until recently maintained a group of these animals. Since the deaths of the last survivors in Antwerp from various causes including tuberculosis (W. De Meurichy 1983 Zoological Society of Antwerp, personal communication), the animals in the National Zoological Gardens, Pretoria have become unique. Derby eland do not seem to do well in captivity, one reason being possibly that they do not adapt well to the artificial diets.

ACKNOWLEDGEMENTS

The authors wish to thank Prof. R.C. Tustin for critisism of the manuscript, Mrs. V. Käber for the typing thereof and the staff of Pathology and Photography, Faculty of Veterinary Science, University of Pretoria for preparing the histological sections and photographs.

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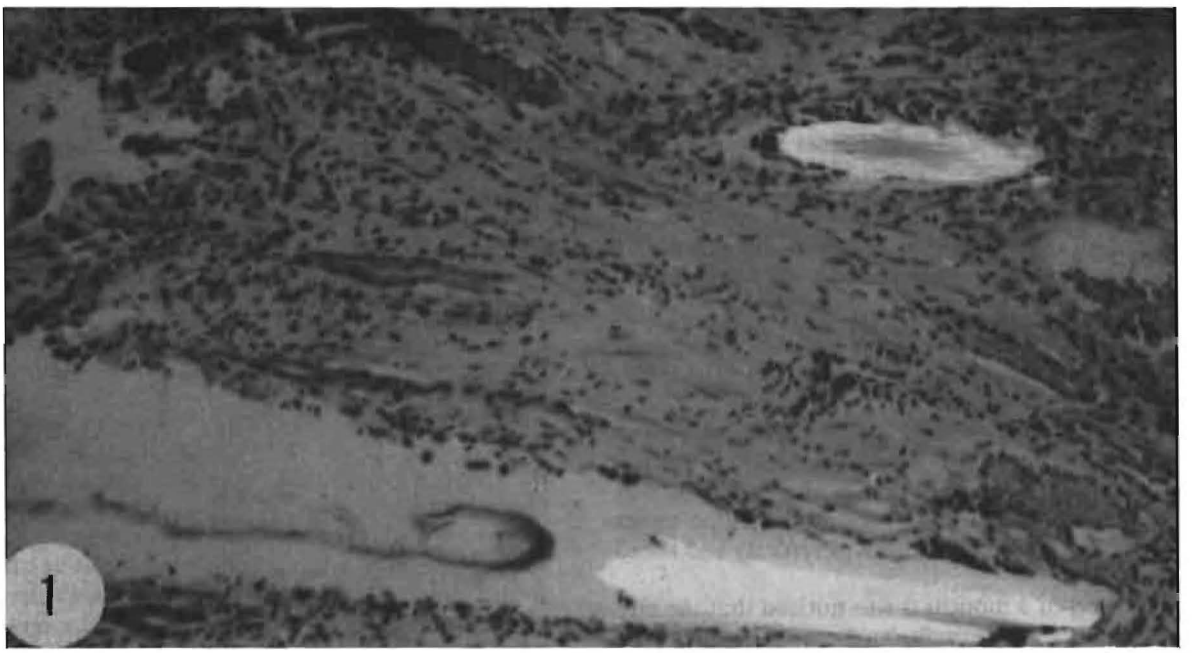

Fig. 1: Fragments of plant embedded in abomasal mucosa. Plant material highlighted by partial polarisation of the light.

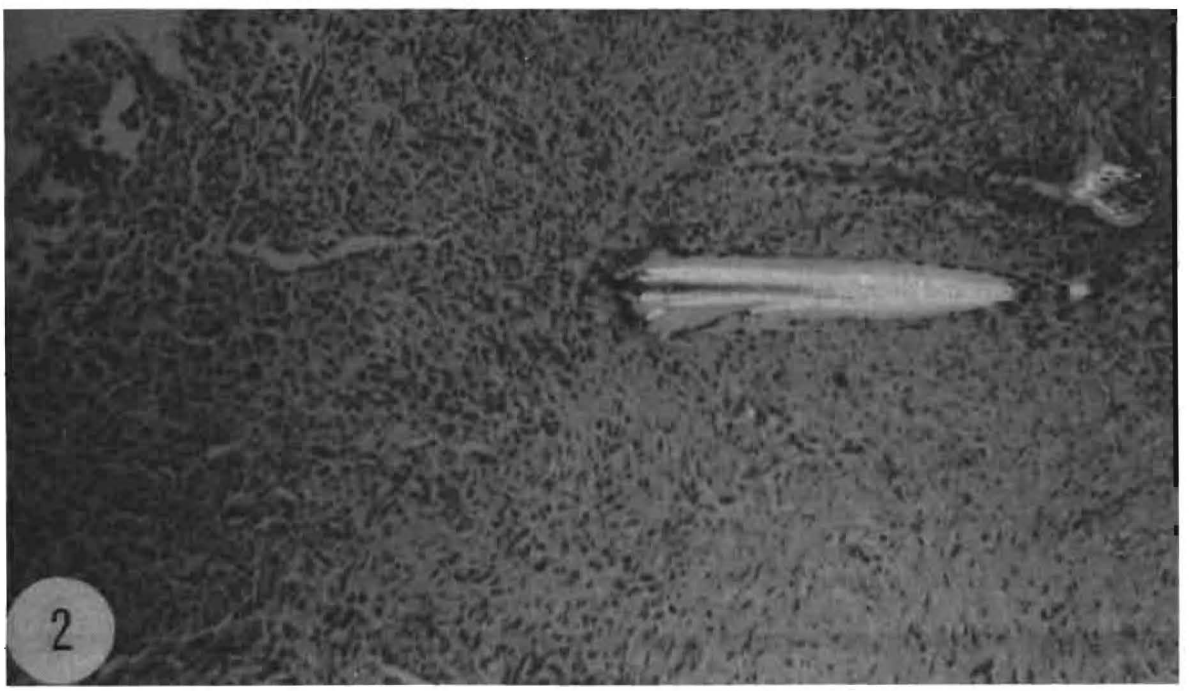

Fig. 2: As above but plant material surrounded by a prominent granulomatous reaction.

CASE REPORT **GEVALVERSLAG**

INTUSSUSCEPTION IN AN OSTRICH CHICK

R.H. KEFFEN*

ABSTRACT: Keffen R.H. **Intussusception in an ostrich chick.** *Journal of the South African Veterinary Association* (1984) **55** No. 2, 77 (En). Oasis Ostrich Farm, P.O. Box 232, Sun City, Bophuthatswana, Southern Africa.

Intussusception of the distal small intestine was observed in a 5-week old ostrich chick. The intussusception occurred at the point of attachment of the yolk sac and was speculated as being a predisposing cause to the problem, as a result of localised infection. Other predisposing causes seen in poultry such as enteritis, tumours, parasites and diet, were not evident in this case.

Key words: Intussusception, yolk sac, ostrich chick.

A male ostrich chick *(Struthio camelus)*, 5 weeks of age, was observed to show signs of general weakness, dehydration, and reluctance to eat. The bird was in good physical condition, and on clinical examination the presenting signs included ventral recumbency, an outstretched neck, closed eyelids, and palor of the mucous membranes of the mouth. Even with considerable prodding the bird was unable to stand even to raise his head. It succcumbed to the ailment before a complete examination could be performed.

The most striking feature on autopsy was the large amount of serosanguinous fluid present in the abdominal cavity. Approximately 20 ml of the fluid was recovered from the body cavity. The body organs showed various degrees of palor. A 200 mm long intussusception occurred in the ileum. The invaginating portion enclosed an 80 mm length of proximal ileum and its mesentery. The affected bowel was twice its normal width and deep purple in colour. An incision through the wall of the ileum revealed a large amount of clotted blood and sanguinous fluid. The mucosa was severely congested. Mesentery veins entrapped by the constricting bowel were also very engorged with blood. The area of constriction was identified as being the point of attachment of the yolk sac.

In a normal resorbed yolk sac, the remnants of blood vessels in the umbilicus form a ligamentous structure which completely envelopes the free lateral borders of the bowel and attaches to the mesenteric vasculature. This forms a ring of tissue around the small intestine. It could be conceivable that, should there be an infection in the yolk sac, the inflammation and fibrous tissue response to the infection could create an obstruction of the bowel or predispose it to the formation of an intussusception.

Intussusception in poultry commonly occurs at the bifurcation of the cecae. They occur in poultry as a result of enteritis[1], parasites, tumours, or hyperperistalsis due to high fibre diets (N S van Blerk 1983, personal communication). Since such predisposing factors were not evident in this case, the aetiology presented is only a speculation.

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*Oasis Ostrich Farm, P.O. Box 232, Sun City, Bophuthatswana.

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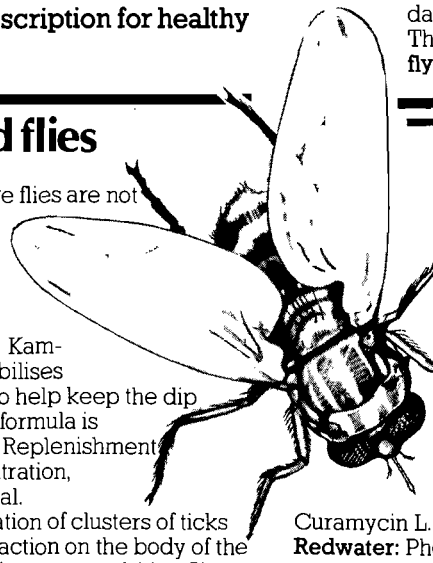

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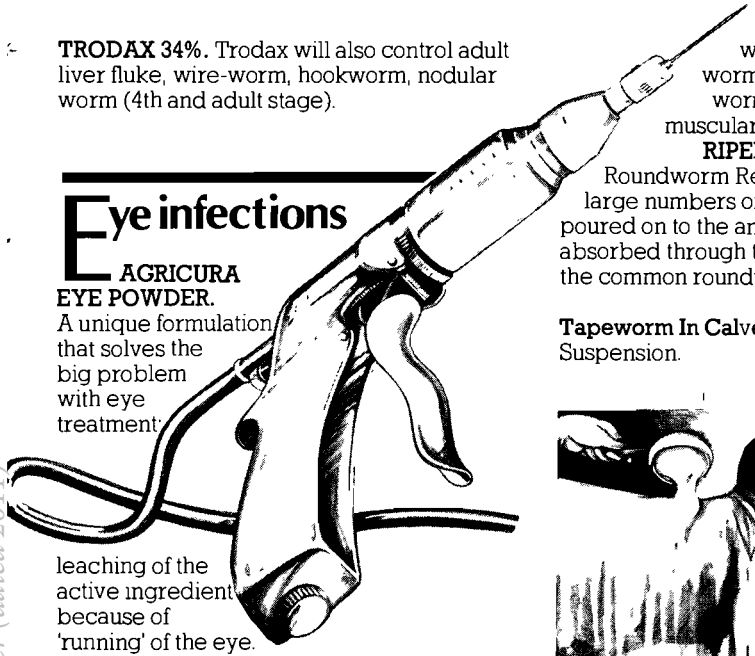

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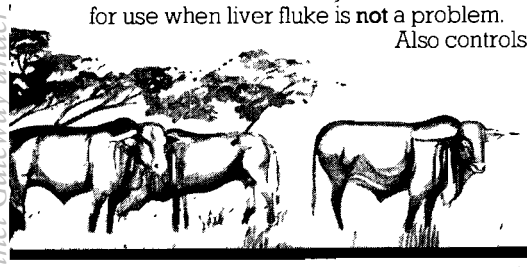

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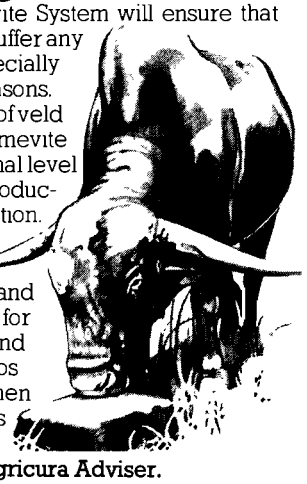

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CASE REPORT GEVALVERSLAG

MADUROMYCOSIS (*MADURELLA MYCETOMATIS*) IN A HORSE

S.R. VAN AMSTEL*, M. ROSS* and S.S. VAN DEN BERGH*

ABSTRACT: Van Amstel S.R., Ross M., van den Bergh S.S. **Maduromycosis *(Madurella mycetomatis)* in a horse.** *Journal of the South African Veterinary Association* (1984) **55** No. 2 81-83 (En). Department of Medicine, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

A case of maduromycosis mycetoma caused by *Madurella mycetomatis* is reported. The horse presented with multiple subcutaneous swelling over the right scapula. There were no discharging fistulae present. Macroscopically the lesion contained a black granular material embedded in a granulomatous mass. Microscopiclly large numbers of microcolonies were present containing segmented hyphae. No typical chlamydospores were observed. Cultures yielded a fungus identified as *Madurella mycetomatis.* Treatment consisted of surgical excision and local treatment with thiabendazole powder.

Key words: Maduromycosis, mycetoma, *Madurella mycetomatis,* horse.

INTRODUCTION

Maduromycotic mycetomas have only been reported on a few occasions in the horse[1-6]. Fungi implicated in these mycetomas identified on histopathological appearance and/or cultural characteristics include *Allescheria boydii, Brachycladium spiciferum* Bainier *(Curvula spicifera* (Bainier) Boedijn), *Curvularia geniculata* and *Helminthosporium spp.*[1-6]. This is believed to be the first reported case of a mycetoma in the horse caused by *Madurella mycetomatis.*

HISTORY

A 11-year-old Thoroughbred gelding was referred to the Department of Surgery, Faculty of Veterinary Science, University of Pretoria with 3 tumour-like subcutaneous swellings over the right scapula. The history indicated that the horse had been operated on twice before to remove similar lesions from the same site, but without success as they had regrown on both occasions. The horse at no stage showed any systemic signs attributable to these lesions. No history of any injury in this area could be obtained.

CLINICAL SIGNS

A clinical examination on the horse revealed no abnormality other than the 3 swellings. They were each about 30 mm in diameter, had a firm consistency, were movable in the subcutaneous tissue and did not appear to be painful. None of these swellings had any discharging openings.

DIAGNOSIS AND TREATMENT

For diagnostic purpose one of the swellings was surgically removed. This proved to be difficult as the lesion was infiltrative and had penetrated between the muscle fibres. On cut surface the lesion consisted of a thick walled granulomatous tissue mass, the central part of which was seperated into smaller divisions by white fibrous septa. Scattered throughout the tissue were black foci of granular material (Fig. 1). Histopathological examination revealed large numbers of fungal microcolonies which under low power had a yellow-brown amorphous appearance (Fig. 2). Under high power these mircocolonies contained segmented hyphae with pigmented cell walls (Fig. 3). No chlamydospores could be identified. The microcolonies were surrounded by a zone consisting mainly of neutrophils with a succeeding predominance of macrophages towards the periphery many of which contained yellow-brown pigment granules in their cytoplasm. Some gaint cells were also present (Fig. 4). On culture the colonial morphology included a white to greyish-white granular appearance with radiating grooves from the centre of the colony (Fig. 5). After some days the colonies changed to a mixture of a brown and dark green colour. The fungus was identified as *Madurella mycetomatis* by Dr C.N. Young of the South African Institute of Medical Research.

Treatment included surgical removal of the remaining 2 lesions. Due to the invasiveness of the mycetoma and the likelihood of regrowth the surgical wound was dusted with autoclaved thiabendazole powder which in vitro significantly suppressed the growth of this fungus (Fig. 6). At the time of discharge which was 30 days after surgical removal, no regrowth of the lesion has occurred.

DISCUSSION

Maduromycotic mycetomas affecting the horse may differ in location, clinical appearance, causative fungal agent, microscopic appearance and course. Anatomical locations described have involved the coronary band[3]; different areas of the skin of the body, neck and limbs[3,4]; ventral thoracic muscles[2]; uterus[6] and nasal septum[1]. The clinial appearance of the lesion has been described as an ulcerated swelling[3], wart-like growths and girth gall[2] and subcutaneous swellings some of which have discharging fistulae[4].

Madurella mycetomatis is the most common cause of maduromycotic mycetoma in South Africa (C N Young, South African Institute of Medical Research, personal communication) and in other tropical areas of Africa and India where it is endemic[7]. A survey done in 1963 covering a 20 year period between 1940 and 1960 showed that at least 464 cases of maduromycotic mycetoma occurred in humans in Africa and Madagascar[7]. The ap-

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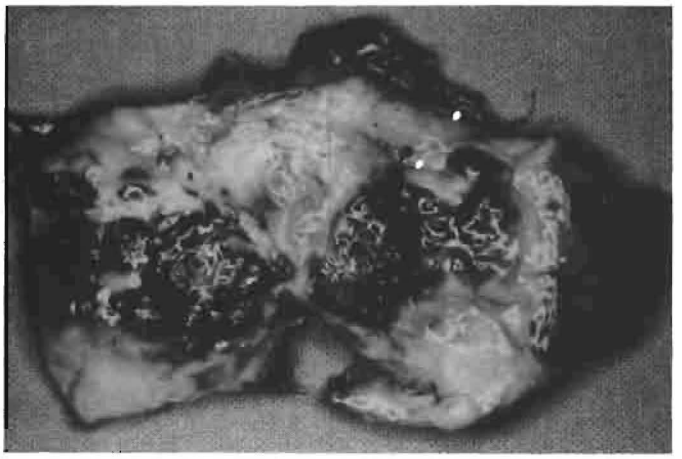

Fig. 1: Macroscopic appearance of the lesion on cut section.

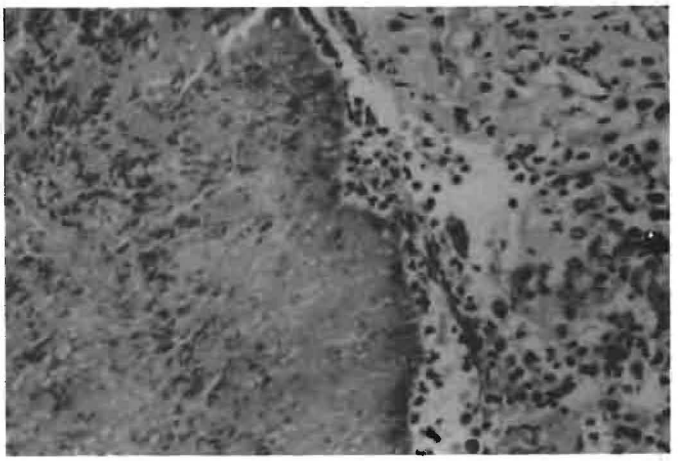

Fig. 4: Reaction zone containing neutrophils, macrophages and giant cells.

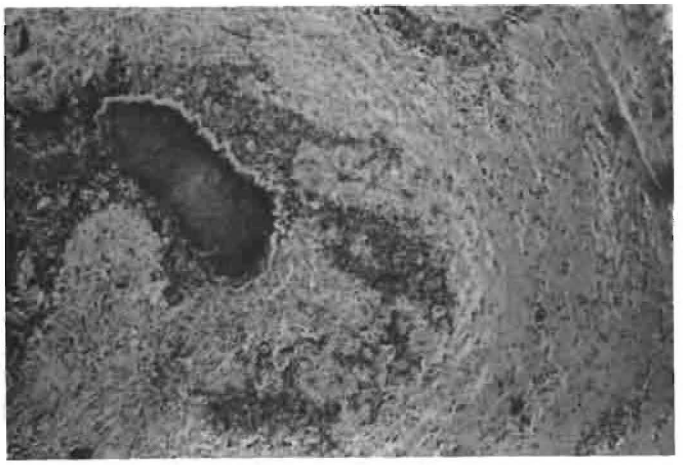

Fig. 2: Histopathological appearance of a microcolony viewed under low power

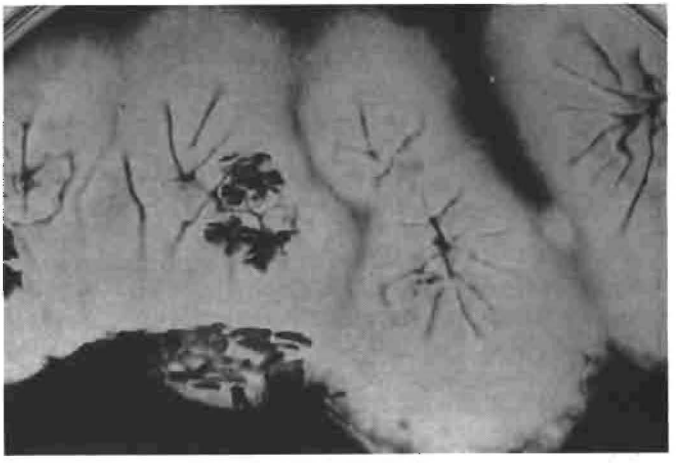

Fig. 5: Colonial morphology showing greyish-white granular appearance with radiating grooves from the centre of the colony.

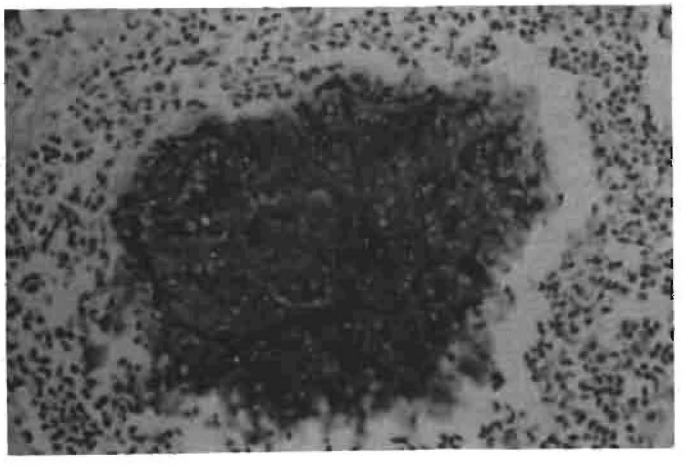

Fig. 3: Segmented hyphae with pigmented cell walls present in the microcolony.

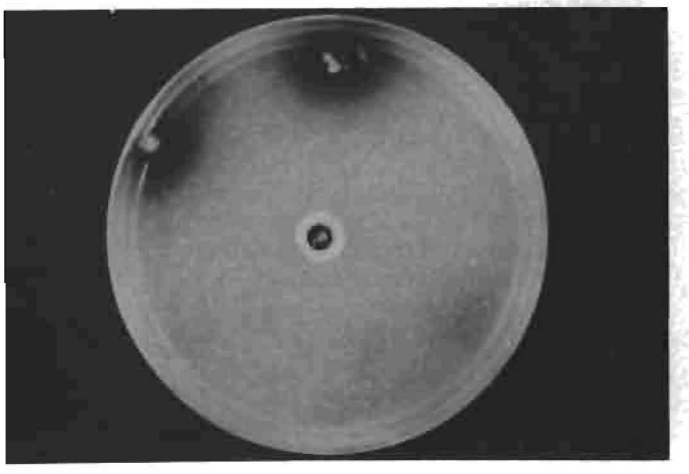

Fig. 6: In vitro suppression of fungal growth with thiabendazole disc.

parent low incidence in lower animals may indicate that the latters tissues are more resistant to these fungi or that the general conditions which favour the initiation of infection occur more commonly in the human. Repeated minor trauma seems to play an important part in the pathogenesis of the disease[7].

Variations in the microscopic appearance include the size of the microcolonies and their contents. Variation in size may be related to host resistance as well as the duration of the infection[4]. The presence of chlamydospores without mycelia are regarded as basic faetures of maduromycotic mycetomas[4]. In this case however no typical chlamydospores could be recognised. The presence of pigment although very characteristic of maduromycotic mycetomas, may not always be present[4]. The course of the infection is usually chronic and treatment often ineffective but spontaneous recovery have occurred in the horse[4].

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BOOK REVIEW

BOEKRESENSIE

FROM THE HORSE'S MOUTH

THE STORY OF SOUTH AFRICA'S VETERAN VET

S.W.J. VAN RENSBURG

1st edition, J.L. van Schaik, Pretoria 1983 pp 330 + Index 7 pp ISBN 0 627 01309 0

For those who know the author, either as colleague or friend, the nature of this autobiography of his can be summed up very briefly: true to the writer's character, simple, straightforward, unassuming, with bubbles of good humour rising and bursting every now and again, with charity to all and malice to none-verily "from the horse's mouth". With prodigious memory, events are recalled from earliest childhood days and described so vividly and in detail that the events of yesteryear seem to have happened yesterday. Reading this book is like sitting comfortably in an arm-chair before a cosy fire and listening to the author telling a fascinating story full of anecdotes and historical references. The interest lies therein that his life-span coincided with momentous developments in South Africa on all fronts. Almost half the book is devoted to the writer's childhood and student days, the latter part of which was spent at the Royal Veterinary College, London, and seeing practice in various parts of England.

To the younger members of the profession the most interesting part undoubtedly will be the overview afforded of the advances made in combatting reproductive disturbances in South Africa. And to the older amongst us it comes as a good reminder of what has been achieved. The third last chapter is devoted to the constitution and functioning of the South African Veterinary Association, in which Professor van Rensburg played a prominent part, whereas the penultimate chapter deals with the Veterinary Faculty at Onderstepoort, Herein he makes a fervent plea for another faculty (apart from the one at Medunsa). Not everyone will agree that the taking over of the Onderstepoort Faculty by the University of Pretoria was a "misfortune", seeing that it at least removed the Faculty from the parsimony of the Department of Agriculture! An important financial issue, which few people realise and which the author leaves unmentioned, is the fact that the hospital set-up associated with every medical faculty is provided for by the Department of Health, whereas the veterinary faculties have to provide such a set-up and services from faculty funds. With the escalation of costs of equipment and drugs, as well as the provision of technical staff, an impasse will soon be reached. In all fairness it has to be pointed out that Government Departments are exposed to a barrage of demands from taxpayers and opposition spokesmen to lessen government spending.

In the final chapter the writer deals out a tried recipe for longevity, which one could well follow.

Although somewhat of a paradox that a person who, in his own words, is not very talkative, should come out with a book like this, it is all to the good. For too long the veterinary fraternity in this country has been too busy fighting "front-line battles" to worry about its own image and overhead strategy and to put their case to the man in the street. To the reviewer's knowledge, this is only the second one of its kind in this country.

H.P.A. de Boom

SOME MONITORING AND TREATMENT EQUIPMENT FOR SMALL ANIMALS

LEA STOGDALE*

ABSTRACT: Stogdale L. **Some monitoring and treatment equipment for small animals.** *Journal of the South African Veterinary Association* (1984) **55** No. 2, 85-88 (En). Department of Veterinary Internal Medicine, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Saskatchewan, S7N OWO, Canada.

Previously used and sterilized fluid administration tubes, bottles and bags were utilized to replace expensive equipment. Urine output and central venous pressure can be readily monitored and thoracic or abdominal fluid can be easily drained using the equipment described.

Key words: Urine output, central venous pressure, pleural drainage.

INTRODUCTION

Intensive care is being increasingly used in small animal practice[4]. This is especially true in South Africa with the high proportion of animals that become severely sick as a result of babesiosis, parvovirus enteritis and trauma. In these, as well as other critically ill animals, it is very useful to be able to monitor urine output and central venous pressure[8]. Dogs or cats suffering from pneumothorax or thoracic effusions must have the air or fluid removed rapidly but without stress[2,3,5]. Using the equipment commercially available for performing these frequently life-saving procedures, often adds considerably to the cost of treatment. This paper describes methods of utilizing previously used and resterilized fluid administration tubes, bottles and bags to perform these procedures.

MATERIALS AND METHODS

Only equipment available in the majority of veterinary clinics was used. For the 3 techniques described the equipment required was a urinary catheter (Dog Catheter, Arnolds Veterinary Products Ltd.), Feeding Tube (Argyle), Jackson Cat Catheter (Arnolds Veterinary Products Ltd.), an intravenous through-the-needle catheter (Venocath, Abbott Laboratories S.A. (Pty.) Ltd.), intravenous fluid (Baxter Travenol Laboratories, Inc.), a fluid administration stand (a drip stand), a linear measure marked in centimeters, adhesive tape, a collecting bowl, disposable needles, empty intravenous fluid bottles and bags (Baxter Travenol Laboratories, Inc.) and fluid administration tubes (Solution Administration Set, Baxter Travenol Laboratories, Inc.).

In preparation for resterilizing the fluid administration tubes, thoroughly wash, clean and flush them with tap water immediately after they have been used. If you have an autoclave, seal each tube in a separate container and autoclave.

Alternatively, if you do not have access to an autoclave, flush and immerse the tubes in a chemical disinfectant, such as 70 % ethyl alcohol, 10 % povidone-iodine (Betadine, Salphar), or 4 % chlorhexidine (Hibitane, I.C.I.) for 24 hours[6]. Following this soaking, flush the tubes with sterile water or saline and hang them up to dry. Once the tubes are completely dry, inside and out, place them in an airtight container with 2 formaldehyde tablets. This sterilizes the ends[6] and keeps the tubes clean and dust free. The tubes are then safely stored where everyone in the practice knows their location and they are ready for immediate use.

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URINE OUTPUT MONITOR

Urine output monitoring is necessary for dogs or cats which are in shock, traumatized, undergoing prolonged surgery or are suffering from uraemia of any cause[3,8]. The following technique is very useful for draining the urine from recumbent dogs, particularly those receiving intravenous fluids, diuretics or glucocorticoid therapy, as it prevents urine scalding. You should not use this method in animals with bladder paralysis: manual bladder expression is preferable to chronic catheterization.

Catheterize the patient's bladder[3,7] and connect the catheter to a fluid administration tube. Insert the end of this tube into an empty fluid bottle or bag. If you use a bottle, insert a needle into the rubber cap so allowing air to escape. With the bottle in the upright position, the needle should not be inserted into the conventional air vent as this will prevent the air from escaping (Fig. 1). Tape the tube to the animal's tail. This reduces the likelihood of the catheter being accidentally pulled out.

Place the fluid bottle or bag on the floor; this is out of everyone's way and the siphon improves drainage. Drain the bladder. Save a sample of the urine for analysis and discard the rest. You can then easily monitor the urine output for as long as you consider it necessary. The normal rate of urine production in dogs is 1-2 ml/kg/h and in cats is 0.5-1 ml/kg/h[3]. However, a urine production level of 0.25-0.5 ml/kg/h indicates adequate tissue perfusion[8].

Urine output monitoring has several advantages. Assessing urine production is easy and it can be measured over a short (one hour) or long (overnight) period. Urine is immediately available for analysis. Urine is kept off the patient, preventing scalding, and out of the way of veterinarians and technicians. The major disadvantage is the possibility of an iatrogenic bacterial cystitis.

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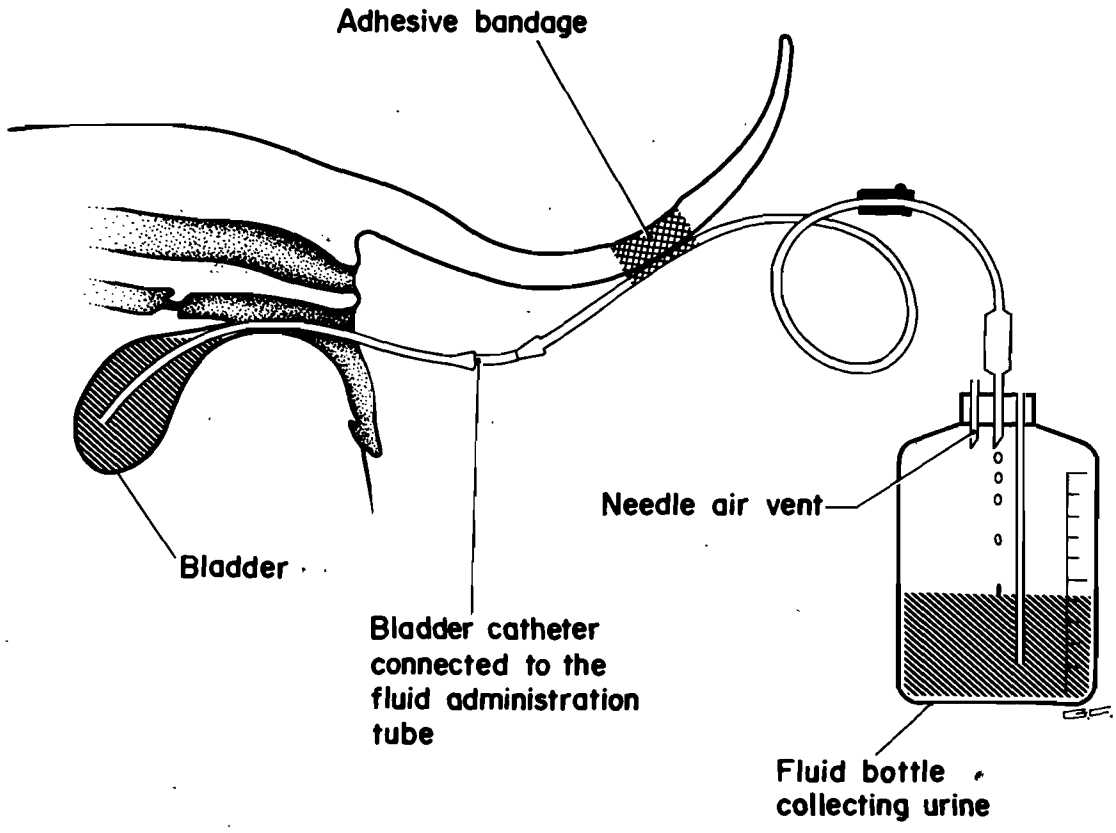

Fig. 1: Urine output monitor in operation (not to scale).

CENTRAL VENOUS PRESSURE MONITOR

The central venous pressure provides a good indication of the venous return and the cardiac competence of the patient. It is a very useful parameter when an animal is in shock, or is receiving large volumes of intravenous fluid for any reason. It is essential in such patients that, additionally, have heart valve incompetence[2,3,8].

The intravenous (IV) fluid therapy is set up intially by inserting an indwelling, through-the-needle catheter into one of the jugular veins. Prepare the injection site by clipping off the hair and disinfecting the skin as you would for a surgical procedure. This is important to prevent the introduction of bacteria to the subcutaneous tissue or the vein. Because you may be leaving the catheter in place for up to a maximum of three days, a local abscess, phlebitis or septicaemia can occur as a result of careless preparation[2,3]. The end of the indwelling catheter should be situated in the thoracic anterior vena cava or in the right atrium. Bandage the catheter and tube securely to the animal's neck. Give any additional emergency therapy, such as fluids, blood or other specific, supportive or symptomatic drugs. The central venous pressure monitor can now be set up easily. Connect a second fluid administration tube to the fluid line with an 18 guage needle. This will become the central venous pressure (CVP) tube. Insert the needle into the rubber section of the fluid administration tube near the catheter connection (Fig. 2). Tape the CVP tube to the drip stand from the level of the table to about 30 cm above the patient. Also tape the linear measure to the drip stand alongside the CVP tube. This scale is marked from -10 to $+20$ cm. Place the O horizontal to the patient's right atrium. With a dog or cat in lateral recumbency, this is approximately at the level of the sternum. Open the valve on the CVP tube and so fill it with the intravenous fluid. The fluid moves through the needle, along the CVP tube past the linear measure and towards the bottle or bag end, which is open to the air.

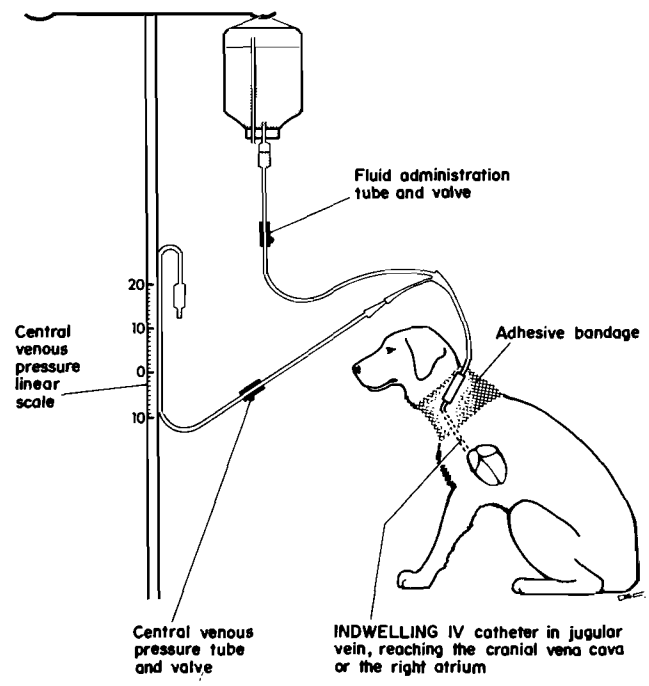

Fig. 2: Central venous pressure monitor in operation (not to scale).

You can now quickly and easily measure the patient's CVP by closing the valve on the fluid administration tube and opening the valve on the CVP tube. This allows the fluid in the CVP tube to fall to a level equal to the pressure at the end of the jugular vein catheter, namely to equilibrate with patient's CVP. Wait a few moments to make sure that the CVP tube fluid level has reached a stable level. The fluid meniscus will move vertically a small amount in time with the heart beat[2]. Read the CVP from the linear measure.

The normal CVP in dogs is -1 to $+5$ cm[3]. A level over 10 cm indicates either excessive fluid administration or a failing right heart. A level less than -1 occurs with inadequate venous return, and so indicates hypovolaemia or peripheral vasodilation. This results in hypotension and the need for more vigorous fluid and supportive therapy. The direction and magnitude of change in the patient's CVP is more important that the absolute value[2,3,8].

When the CVP has been recorded, open the valve on the fluid administration tube. When the level of fluid in the CVP tube rises about 5 cm, close the CVP tube valve. This prevents any back flow of blood into the IV catheter and CVP tube the next time you measure the CVP. Fluid therapy is continued as indicated by the CVP just measured and the other parameters being monitored.

Monitoring central venous pressure has many advantages. It's easy to set up and read this simple piece of equipment. Because large volumes of fluid are usually administered via a jugular vein catheter, much of the equipment is already in place. The two valves on the fluid administration and CVP tubes are easy to operate; much easier than 3 or 4-way valves. The CVP rapidly and reasonably accurately indicates the efficacy of fluid therapy.

ASPIRATION APPARATUS

A hydrostatic pressure aspiration apparatus is very useful and sometimes life-saving, for removing fluid or air from the pleural cavity[2,3,5]. Occasionally, a massive accumulation of transudate in the peritoneal cavity may require drainage. This equipment can readily be set up utilizing fluid administration bottles and tubes. Remove the metal seals from the top of the fluid bottles, and take the long air vent out of one bottle. Fill the other bottle with tap water and connect the fluid administration tubes to the bottles as shown in Figure 3.

Insert a catheter or needle into the thoracic cavity in the appropriate position[2,3,5]. I use either an 18 gauge needle or a 14 gauge through-the-needle intravenous catheter, attached to a 10-20 ml syringe. Usually I place the needle or catheter through a surgically-prepared site on the thoracic wall at the 7th intercostal space at mid-thorax. The site selected depends on the radiographic findings. If I think that the pleural space will require drainage for longer than the immediate period, I insert a catheter. Either a chest drain or a long intravenous catheter is appropriate. The chest will drain faster if the catheter has multiple holes in it. You can make extra holes in the intravenous catheter by using sterilized curved surgical scissors. The holes should be at most one-third of the diameter of the tube, and should be restricted to the distal half of the tube. After inserting the catheter, suture and bandage it in place. Use a screw

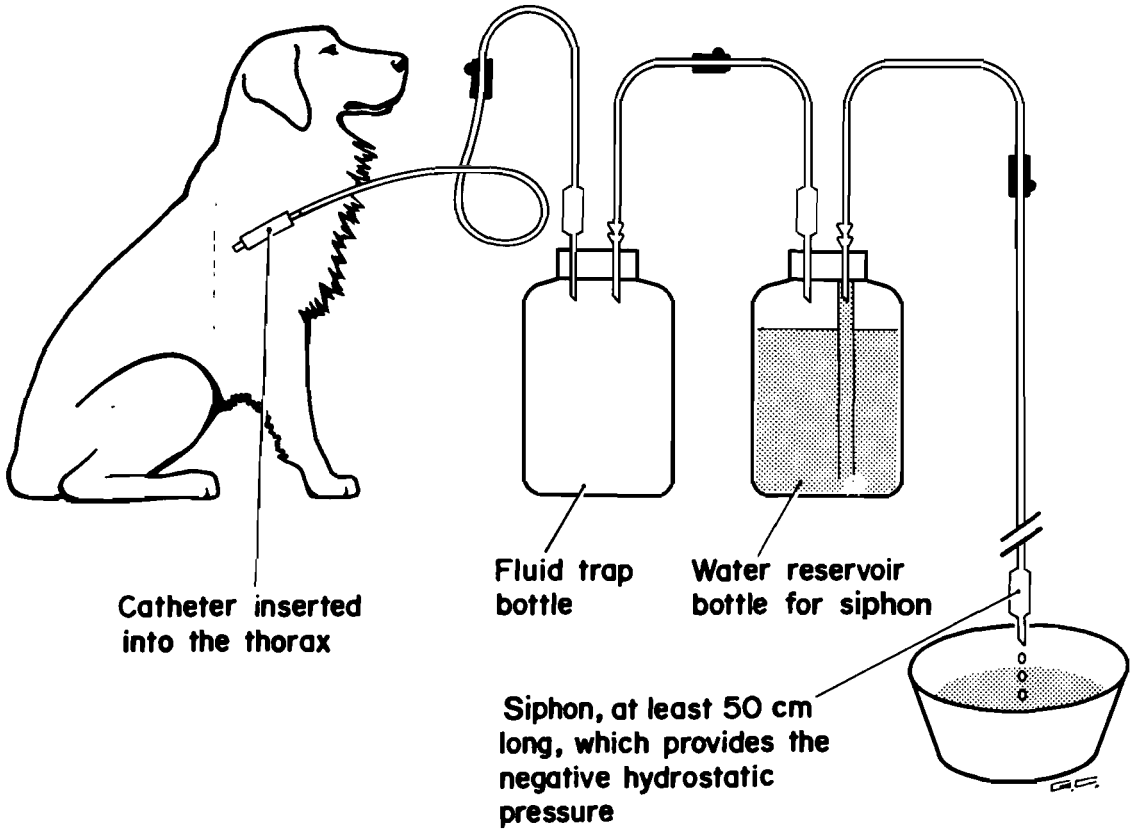

Fig. 3: Hydrostatic pressure aspiration apparatus in operation (not to scale).

clamp to ensure that the tube is securely closed when the animal is not being supervised[3,5].

Check that the needle or catheter is correctly positioned in the pleural cavity by aspirating fluid or air into a syringe. Analyze this sample of fluid and culture it, if relevant. Connect the aspiration tube to the needle or catheter in the chest. With all three valves open, a siphon results. The fluid or air within the pleural cavity (or the peritoneal cavity, as the case may be) moves along the tube attached to the dog and is trapped in the first bottle. This bottle allows you to measure the volume of fluid drained and provides samples for further diagnostic tests, if necessary. It also prevents the fluid from spilling over the animal, veterinarian or technician. Only air passes along the tubes between the bottles into the water reservoir bottle. The siphon is established from the bottom of the water reservoir bottle along the third tube which is filled with water. This tube is hung over the table edge and drains into a collecting bowl. The siphon tube must have a height of at least 50 cm to ensure that the applied negative pressure always remains greater than the negative intrapleural pressure. The pleural pressure may reach -30 mm Hg ($= -40$ cm H_20) in animals in respiratory distress[1]. Partially closing any of the valves on the 3 tubes decreases the fluid flow from the patient. Close the valves completely when the fluid trap bottle must be emptied or the water reservoir bottle refilled.

The hydrostatic pressure aspiration system has several advantages. The pressure gradient established is constant, low to moderate in degree and is easily controlled. It's faster than draining a body cavity using a syringe and 3-way-valve. The aspirated fluid is safely contained in a bottle and infected fluid does not contaminate the area.

CONCLUSION

Urine output monitoring, central venous pressure measurement and hydrostatic pressure aspiration enables the clinician to make more accurate assessments of the status of patients. They also permit you to administer the appropriate therapy rapidly and in the correct quantities. The closed urine output monitoring system using a fluid administration tube and fluid bottle or bag provides a very simple, efficient means of measuring a patient's urine production. It reduces the likelihood of iatrogenic infection and eliminates urine scalding of patients. The central venous pressure monitor using a fluid administration tube provides a cheap, convenient and useful method of objectively evaluating the haemodynamic status of patients. Using this technique, I find it easy to gauge the adequacy of fluid therapy, especially when I am administering large volumes rapidly. The hydrostatic pressure aspiration apparatus utilizing fluid administration tubes and bottles provides an economical, practical method of removing excessive quantities of fluid or air from body cavities. All the equipment described can be put together from materials found in veterinary practices. They can be set up and used by technicians, saving time and money. Mostly importantly, each improves the standard of patient care.

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MYOCARDIAL PATHOLOGY OF DOMESTIC RUMINANTS IN SOUTHERN AFRICA

S.J. NEWSHOLME* and J.A.W. COETZER*

ABSTRACT: Newsholme S.J.; Coetzer J.A.W. **Myocardial pathology of domestic ruminants in Southern Africa.** *Journal of the South African Veterinary Association* (1984) **55** No. 2, 89-96 (En). Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa.

Myocardial pathology of ruminants in southern Africa, including lesions associated with toxic plants, other toxic agents, infectious agents and nutritional deficiency, is discussed with regard to recognition and to aetiological diagnosis. Findings are included which have not been published elsewhere. The importance and difficulties in recognition of myocardial lesions at an early stage are emphasized. Further research into the pathology of cardiac failure caused by toxic plants is clearly needed.

Key words: Myocardial pathology, ruminants, cardiotoxic plants and substances.

INTRODUCTION

Cardiac failure in domestic ruminants can result from a variety of toxic, infectious and nutritional causes in southern Africa. Numerous indigenous plant species are known to be cardiotoxic[47 52], and stock losses caused by some of these species are of considerable economic importance[21 32]. Since cardiac failure in farm livestock is often acute, affected animals are usually found dead. The opportunity for clinical examination, therefore, is limited, and necropsy is frequently the starting-point of diagnostic investigation.

Herein we discuss the myocardial pathology of ruminants in southern Africa, and perpend its diagnostic potential and limitations. Findings from necropsy material examined in recent years by the Section of Pathology, Veterinary Research Institute (VRI), Onderstepoort are included which have not been published elsewhere.

RECOGNITION AND INTERPRETATION OF CARDIAC LESIONS

Macroscopical Examination

To assess cardiac failure at necropsy it is imperative to examine the other organs as well as the heart itself. Passive congestion, oedema of the lungs and/or thoracic, pericardial or peritoneal effusion, if present, may be more important evidence of cardiac failure than are changes in the heart itself. These extracardiac changes are manifestations of cardiac dysfuntion whereas most myocardial lesions, themselves, provide no direct evidence of cardiac dysfuncition.

Some myocardial lesions are readily discernible grossly, particularly when they are sharply demarcated or extensive. Cardiac lesions in many cases of white muscle disease[23] and gousiekte[44], for example, can be detected by gross examination. Since many lesions, however, are not detectable grossly, failure to find them should not preclude microscopical examination.

Delays are often inevitable before necropsies can be done on farm stock, so that post-mortem autolysis is common and may complicate the recognition of lesions. Diffuse myocardial pallor, often more conspicuous in the endocardial zone, may occur as a result of post-mortem autolysis, but the tissue should be examined microscopically if a lesion is suspected. We have also observed irregular, pale areas in the ventricular walls. The only microscopical change that could be detected in these areas was a marked paucity of erythrocytes in the capillaries, and we believe that the pallor may be a manifestation of irregular blood redistribution caused by local differences in the degree of rigor mortis. The size of the cardiac chambers and the thickness of the ventricular wall can vary with the stage and degree of rigor mortis, and such variations may be confused with moderate ventricular hypertrophy, atrophy or dilatation. Accurate assessment of ventricular hypertrophy or atrophy requires weighing of the ventricles, but few figures for ventricular weights of normal ruminants are available. Ventricular weights of Merino sheep in South Africa have been measured and analyzed in relation to body mass, sex and age[37], but the results show considerable variation, and it is unlikely that the weighing of hearts will prove useful in field investigations.

Microscopical Examination

Certain features occur regularly in the myocardium of cattle, sheep and goats with no history to suspect cardiac dysfunction. Awareness of such features is essential to histopathological assessment. Sarcocysts are common, and there is rarely any tissue reaction associated with them. Small foci of lymphocytic infiltration have been found in the myocardium interstitium of many hearts from normal sheep[37]. Vacuolation of cardiac myocytes and reduced packing density of myofibrils in the subendocardial myocardium have been seen commonly in bovine hearts in Britain[10]. We have seen similar vacuolation occasionally in hearts of normal cattle and sheep (Fig. 1).

Artifacts with routine haematoxylin and eosin (HE) staining can also cause confusion. The intensity of eosinophilia of cardiac myocytes in sections differs in line with knife scores, and myocyte fascicles cut transversely stain more weakly than those cut longitudinally. Postmortem autolytic changes include pale staining of myocytes and the appearance of gaps between them. Even staining throughout the tissue, large amounts of formalin artifact pigment and the presence of numerous

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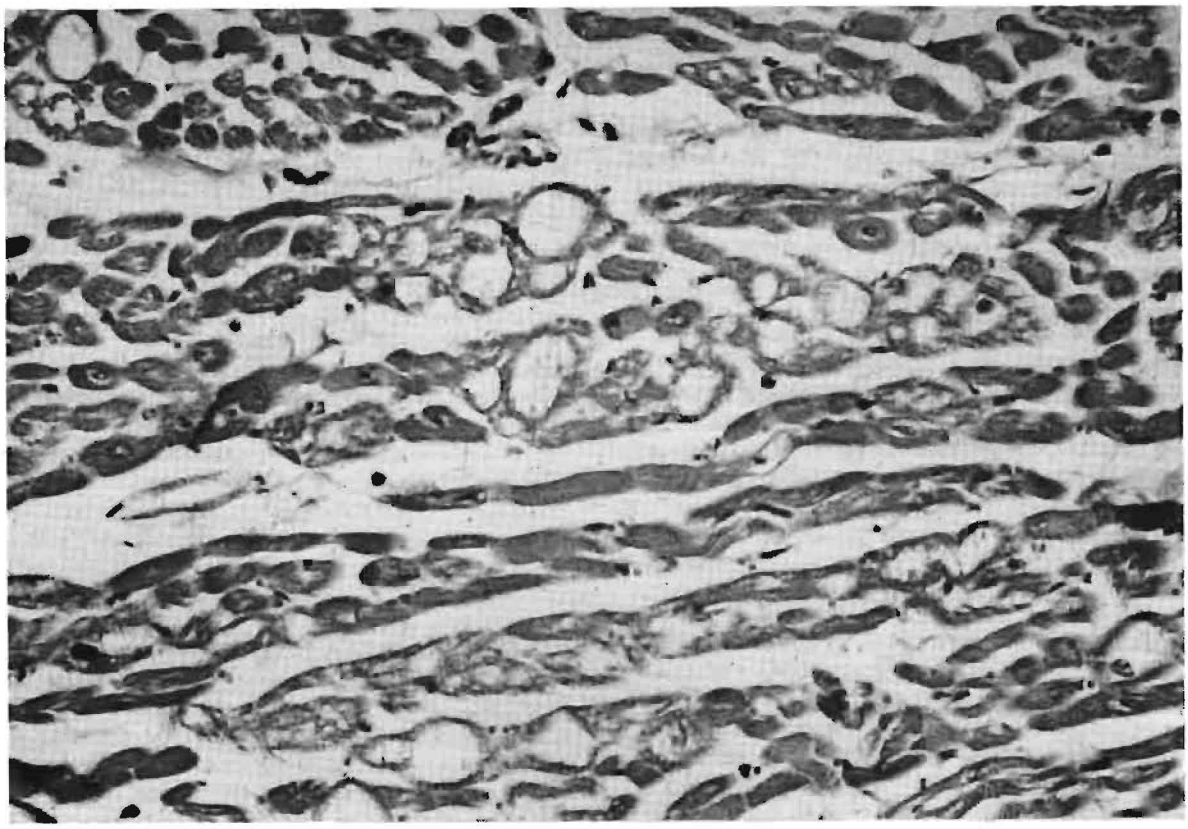

FIG. 1: Vacuolation of cardiac myocytes; heart from normal sheep HE X 600

bacteria are features of myocardial autolysis which serve to distinguish it from necrosis[10].

In many cases of sudden death myocardial lesions are not obvious. It becomes important to determine the earliest light microscopical manifestations of myocardial injury in these cases, and to distinguish them from artifacts and autolytic changes. It is here that we encounter difficulties. Increased eosinophilia of cardiac myocytes occurs as an early event in myocardial ischaemia[28]. It has been reported that variations of myocyte eosinophilia are strongly indicative of myocardial necrosis in cattle, and that such changes do not usually occur in significant numbers of cells in normal myocardium even with substantial delays in fixation[10]. We have seen variations in eosinophilia not only in hearts from cases of acute cardiac failure but also in normal hearts, and we are reluctant to accept them as firm evidence of injury. Empirical staining methods have been developed to differentiate injured myocytes at any early stage. These include modifications[16,25,40] of an acid fuchsin method[43] by which injured myocytes can be identified by their intense fuchinophilia before any changes are detectable in HE sections. A haematoxylin – basic fuchsin-picric acid (HBFP) method has also been developed[26], which has been found effective in detecting ischaemic injury as early as 30 min after vascular ligation. This method has been reported as useful in examining bovine hearts but results are sometimes unaccountably inferior[10]. On occasion we have applied an acid fuchsin technique[40] to myocardium from sheep that had died of acute cardiac failure but our results have been variable. It is clear that the success of these empirical methods depends upon strict attention to staining concentrations and times and on careful comparison with positive and negative control sections.

Irregular contraction of sarcomeres is identifiable by HE staining but appears more clearly in sections stained by Mallory's phosphotungstic acid haematoxylin[27] or Heidenhain's iron haematoxylin[45]. We recommend the use of either of these methods where acute cardiac failue is suspected.

Electron Microscopy

Electron microscopy has proved of value in identifying and clarifying early features of myocardial injury in cattle[10], despite the fact that its routine use for field material is usually limited to formalin-fixed tissues. Interpretation, however, must be made with caution. Mitochondrial matrical electron-dense bodies, for example, increase in number with time after death in bovine myocardium[10]. Studies of delayed fixation of canine myocardium have revealed that several ultrastructural changes occur during autolysis which resemble those occurring in ischaemic injury[4,20].

CARDIOTOXIC PLANTS

Plants Containing Cardiac Glycosides

Cardiac glycoside poisoning of cattle and sheep is of considerable economic importance in South Africa compared to other countries. This is probably due to the large variety and wide distribution of plants here which contain cardiac glycosides and the fact that most stock is kept under range conditions[32]. The most important of these plants are species of *Homeria* and *Moraea,* which

are commonly known as 'tulp', species of *Urginea, Ornithoglossum* and *Pseudogaltonia,* known as 'slangkop' and species of *Cotyledon, Kalanchoe* and *Tylecodon.* Less important plants include *Melianthus comosus, Bowiea volubilis, Nerium oleander, Thevetia peruviana, Digitalis* spp, *Asclepias fruticosa, Acokanthera* spp, *Adenium* spp and *Strophanthus* spp.

Severe cardiac arrhythmias, leading to ventricular fibrillation have been described in poisoning of ruminants by some of the cardiac glycoside-containing plants[13,14], but associated myocardial pathology has not been studied in detail. An assay method for cardiac glycosides[9] has been adapted for use on ruminant tissues and rumen contents (R A Schultz and T W Naudé 1975, VRI, Onderstepoort, unpublished work), but the method is time-consuming and is not in routine use. In numerous necropsies of cattle and sheep with a history of suspected tulp or slangkop poisoning we have sometimes found extracardiac evidence of congestive cardiac failure, but gross and microscopical findings have been limited to epicardial and endocardial haemorrhages. In occasional cases, however, and particularly where the history suggested that the animal had lived for several days after ingestion of the toxic plant, we have found scattered foci of myocardial necrosis, sometimes with mononuclear inflammatory cell infiltrates and evidence of early fibroplasia. These findings suggest that morphological evidence of myocardial injury in cardiac glycoside poisoning in ruminants may be more consistent than has been recognized, and that efforts should be made to identify it at an earlier stage.

Poisoning by *Cotyledon* and *Kalanchoe* spp is normally manifest as a chronic disease known as "krimpsiekte"[19], which is characterized by clinical signs referable to nervous system dysfunction rather than to cardiac failure. It has been shown experimentally that cardiac glycosides extracted from *Cotyledon, Kalanchoe lanceolata* and *Tylecodon glandiflorus* caused krimpsiekte when small doses were given repeatedly to sheep. When large doses were given, however, acute disease with evidence of cardiac failure resulted[2,3]. In some of these cases multifocal myocardial degeneration and necrosis, often with lymphocytic, lymphoblastic and macrophage infiltrations, were observed. Microscopical examination of myocardium should not be neglected if poisoning by these plants is suspected.

'Gousiekte'

'Gousiekte' is the name given to a disease of ruminants which is characterized by sudden death from cardiac failure and which is caused by ingestion of certain plants of the family Rubiaceae. Plants shown to cause gousiekte are *Pachystigma pygmaeum*[48], *Pachystigma thamnus*[1], *Pavetta schumanniana, Pavetta harbori*[51] and *Fadogia monticola*[21]. 'Gousiekte' is a plant toxicosis of economic importance in South Africa[21]. There is a latent period of 4-8 weeks between ingestion of the plant and the time that deaths occur. Many, but not all, cases show macroscopical evidence of congestive cardiac failure, including generalized congestion, ascites, hydropericardium, hydrothorax and pulmonary oedema. Such cases resemble heartwater macroscopically, and brain smears should be examined. Features which may help to distinguish heartwater are the presence of abomasal oedema, nephrosis and wetness of the cut surface of the brain. An early report[48] described marked ventricular dilatation as a consistent feature, but subsequent observations[44] including our own indicate that ventricular dilatation occurs in only a small proportion of cases. In many cases the ventricular walls are thinner than normal and have a tough consistency. Irregular areas of pallor, especially in the endocardium, may be observed. in some cases, however, macroscopical changes in the heart are not recognizeable. Microscopical examination reveals foci or areas of loss of myofibres with replacement by fibrous tissue. Atrophy of myofibres may be evident, and lymphocytic infiltrates of varying intensity are often present[21,44,48]. These lesions are generally more pronounced in the endocardial zone. They are found most consistently in the cardiac apex, which has been recommended as the area of choice for routine examination. Connective tissue stains can be useful to appreciate the extent of fibroplasia. The identity of the toxic principle in gousiekte is not known and the pathogenesis is not clear. Selective loss of myosin filaments from cardiac myocytes has been reported as an early change in a preliminary ultrastuctural study[33], but there is clearly a need for more research into the pathogenesis.

Dichapetalum cymosum

Dichapetalum cymosum ('gifblaar') is an important cause of stock losses in certain areas. The toxic principle of the plant is monofluoracetic acid[29], which is converted within the body to fluorocitrate[39]. There is no chemical test for the routine diagnosis of gifblaar poisoning. Citrate levels in heart muscle and diaphragm have been found to be elevated in experimental monofluoracetate poisoning in sheep, but the stability of citrate in the tissues requires further investigation[41]. In most field cases of 'gifblaar' poisoning death occurs within a few hours after ingestion of the plant. In ruminants peritoneal, thoracic and pericardial effusion have been documented[46]. In most cases, however, no pathological features of diagnostic value have been found, and diagnosis must be based on circumstantial evidence and the finding of *D. cymosum* leaves in the rumen contents. This lack of pathological findings is to be expected, since no consistent pathological changes were observed in an experimental study of acute fluoracetate toxicosis in sheep[22]. Here, again, application of methods to detect early myocardial injury might be useful.

Myocardial lesions have been seen occasionally in ruminants that have died after graze on veld containing *D. cymosum*[41], and some of these animals were known to have ingested *D. cymosum* several days before they died. These lesions consist of multiple small foci of myocardial necrosis or loss of myofibres (Fig. 2), often with lymphocytic infiltrates and early fibroplasia. Similar lesions have been produced experimentally in sheep by the administration of low doses of monofluoroacetate over a prolonged period[41]. These lesions also resemble those caused by *Acacia georginae,* an Australian plant that contains lower levels of monofluoracetate than does gifblaar[53]. This evidence suggests that the myocardial lesions seen in ruminants after grazing on veld containing *D. cymosum* are a manifestation of chronic *D. cymosum* poisoning. It is important to distinguish these lesions from those of gousiekte, since *D. cymosum* and plants causing gousiekte can occur in the same veld. In contrast to gousiekte, the lesions in monofluoracetate poisoning tend to be multifocal and

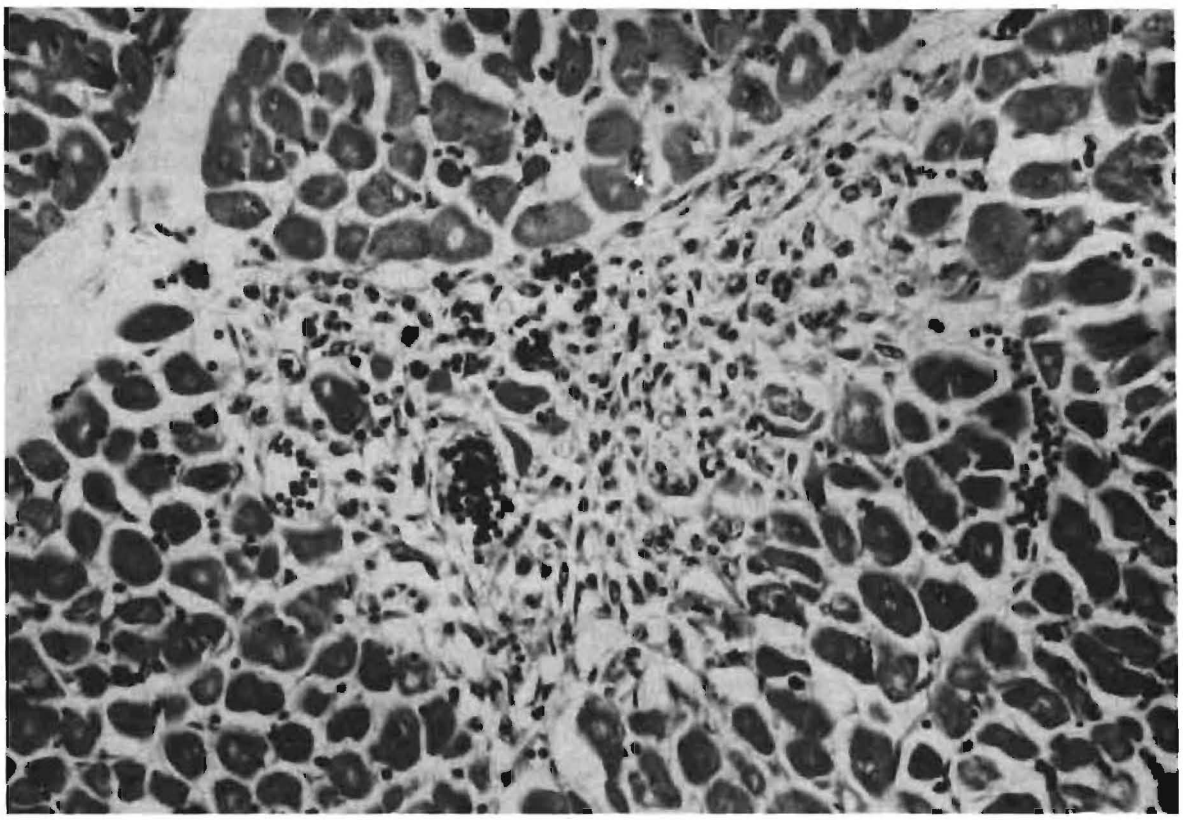

FIG. 2: Focus of loss of myocytes with fibroplasia in bovine myocardium; *D. cymosum* poisoning. HE X 600

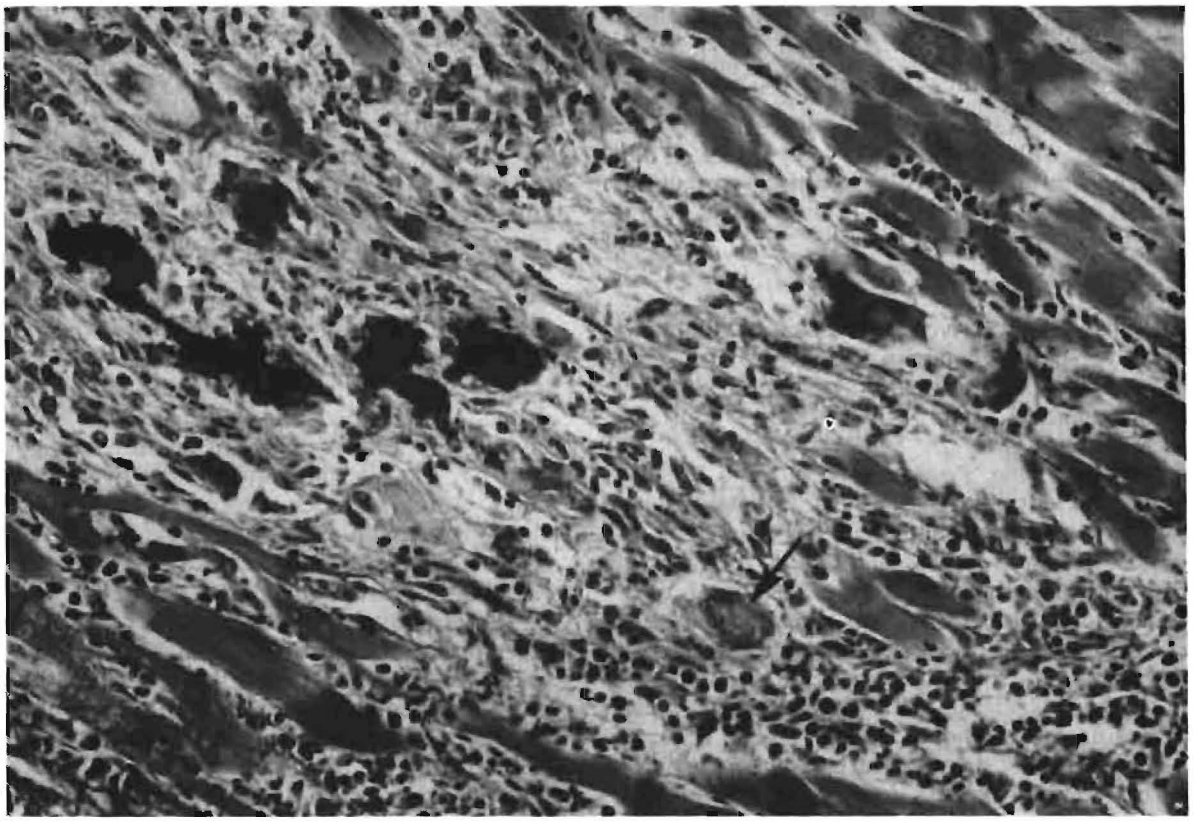

FIG. 3: Necrosis and mineralization of myocytes, fibroplasia and mixed inflammatory cell infiltrates including multinucleated giant cell (arrow) in bovine myocardium; hybrid vetch poisoning. HE X 600

distributed throughout the myocardium. Fibroplasia is less marked[41].

Other Plants

Distinctive lesions occur in *Vicia villosa* (hairy vetch) poisoning[38], and these lesions involve the myocardium in some cases. Similar myocardial lesions have been described in an outbreak of suspected poisoning by a hybrid vetch in South Africa[12]. The lesions were often detectable macroscopically as irregular, yellowish grey foci or streaks, and microscopically they consisted of fibrous tissue containing necrotic and mineralized myofibres and infiltrates of mononuclear cells, eosinophils, plasma cells and multinucleated giant cells (Fig. 3).

Galenia africana, a plant that grows in certain areas of the Karoo, has been implicated as the cause of 'waterpens', a condition characterized by severe ascites, in sheep and goats. The plant was believed to be hepatotoxic because marked liver lesions were found in cases of 'waterpens'[17]. In a retrospective study of field cases of 'waterpens', however, we have found microscopical changes in the liver (Fig. 4) compatible with chronic congestive right ventricular failure, and multifocal myocardial lesions, including vacuolar degeneration, hyaline degeneration and necrosis, mononuclear cell infiltration and fibrosis (Fig. 5).

An outbreak of a disease in sheep in South Africa has been described in which there was widespread arterial calcification[50]. Evidence of congestive cardiac failure, including thoracic, pericardial and peritoneal effusion and pulmonary oedema, was found in many of these sheep. The hearts of many of them were dilated, and focal calcification of cardiac myofibres and myocardial infarction were observed in several. It was assumed that these cardiac changes were secondary to the arterial pathology. The cause of the outbreak was not identified, but the possibility of a plant toxicosis was not dismissed.

Another plant that may be cardiotoxic is *Thesium namaquense*. The pathology of this plant remains to be investigated.

OTHER TOXICOSES

Outbreaks of sudden deaths in sheep in South Africa attributed to monensin toxicosis have been reported[36]. Affected sheep showed macroscopical evidence of congestive cardiac failure. Myocardial lesions varied from small foci to extensive areas of necrosis with mild, mixed inflammatory cell infiltrates and early fibroplasia. The distribution of the more severe myocardial lesions was predominantly in the epicardial zone, and it was suggested that this may be a useful diagnostic feature. This suggestion, however, may be misleading. The number of cases examined was small, and in a study of experimental monensin toxicosis in sheep[15] predominance of the lesions within the epicardial zone was not reported.

Sudden deaths in sheep also occurred after accidental overdosage with salinomycin, an ionophore antibiotic similar to monensin. Myocardial lesions in these cases resembled those in monensin toxicosis (S.S. Bastianello 1983, Faculty of Veterinary Science, University of Pretoria, personal communication).

INFECTIOUS AGENTS

Bacteria

Myocardial abscesses and multifocal or diffuse fibrinopurulent myocarditis occur sporadically in ruminants in South Africa. Bacteria which have been isolated include *Pasteurella* spp and *Corynbacterium pyogenes*. Lesions of tuberculosis may also involve the myocardium. Recently several bovine cases of blackleg ('spons-siekte'), caused by *Clostridium chauvoei,* have been encountered in which fibrinopurulent epicarditis and gangrenous myocarditis were present, in addition to the skeletal muscles typical of the disease.

Viruses

Necrotizing myocarditis can occur in young ruminants in foot and mouth disease[23]. In malignant catarrhal fever ('snotsiekte') of cattle the perivascular lesion frequently involves the myocardium. Myocardial degeneration and necrosis of the papillary muscle of the left ventricle have been described as a consistent lesion in bluetongue disease of sheep[31], and our own observations agree with this.

Protozoa

Myocardial interstitial lymphocytic proliferation occurs in East Coast fever[18], caused by *Theileria parva parva.* Myocarditis has been reported as a fairly constant lesion in Corridor disease of cattle[34], caused by *Theileria parva lawrencei*, and we have seen marked myocardial perivascular lymphoblastic proliferation consistently in this disease. Sarcocysts have been mentioned above.

Metazoa

Occasional myocardial granulomas have been associated with cysticerci in ruminants. Myocardial infarction caused by larvae of *Gedoelstia* spp. has been described in sheep in South West Africa[56]. Cardiac lesions associated with the nematode, *Cordophilus sagittus*, have been described in kudu[30], and recently this parasite has been found in cattle (J. Boomker and Anna Verster 1983, VRI Onderstepoort, unpublished observations).

NUTRITIONAL CAUSES

White Muscle Disease

Cardiac lesions of white muscle disease in lambs have been reported in South Africa[11,49], and we have seen such lesions in young cattle, sheep and goats. The lesions do not differ from those described elsewhere[23].

Outbreaks of sudden death in young dairy calves in Britain have been investigated in which patchy myocardial pallor was seen macroscopically[14]. HBFP staining revealed areas in which myocardial fibres stained intensely with fuchsin, suggesting peracute myocardial injury. Lesions typical of white muscle disease were absent, but there was biochemical evidence of selenium deficiency in in-contact calves. Further deaths ceased after selenium supplementation. These findings indicate that acute cardiac failure associated with selenium deficiency may occur without the typical lesions. No such cases have yet been reported in southern Africa, but the importance of detecting early myocardial lesions is again emphasized.

Copper Deficiency

A condition associated with copper deficiency in cattle

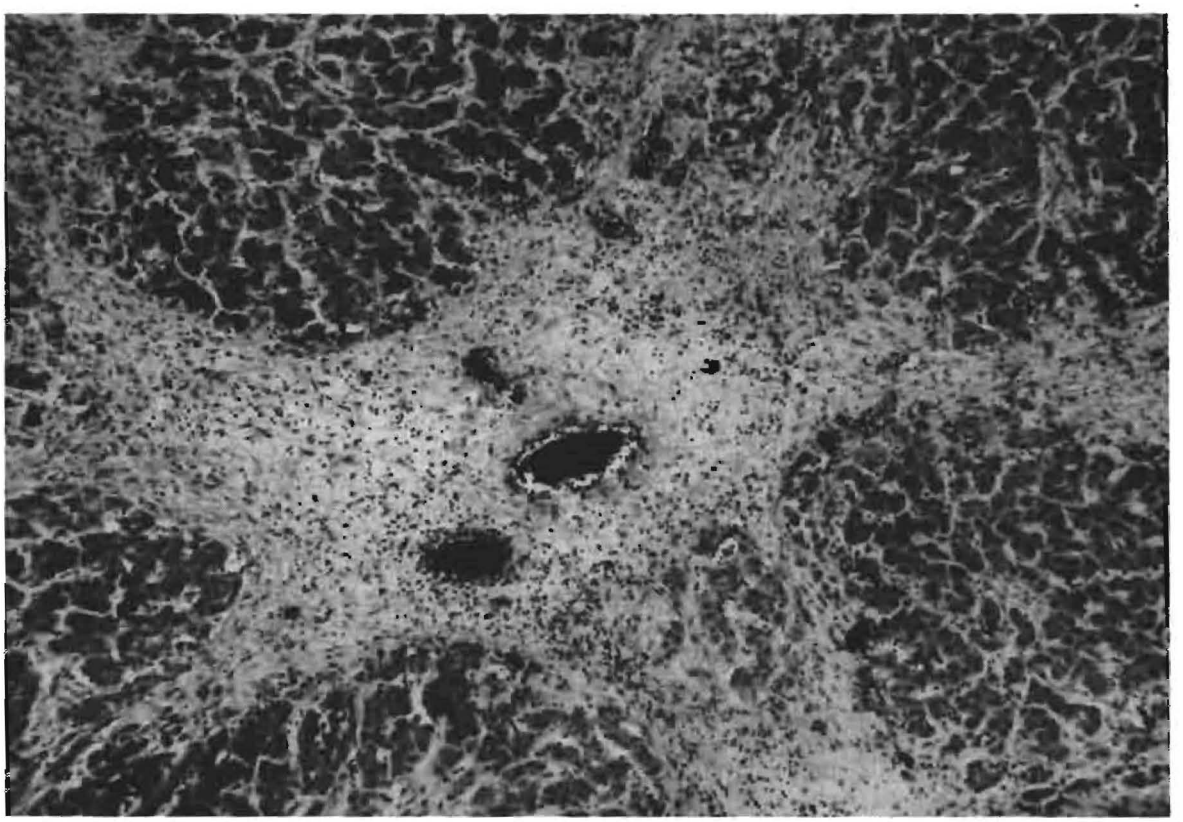

FIG. 4: Centrizonal fibrosis with bridging between adjacent lobules and duplication of the central vein in ovine liver; *Galenia africana* poisoning. HE X 200

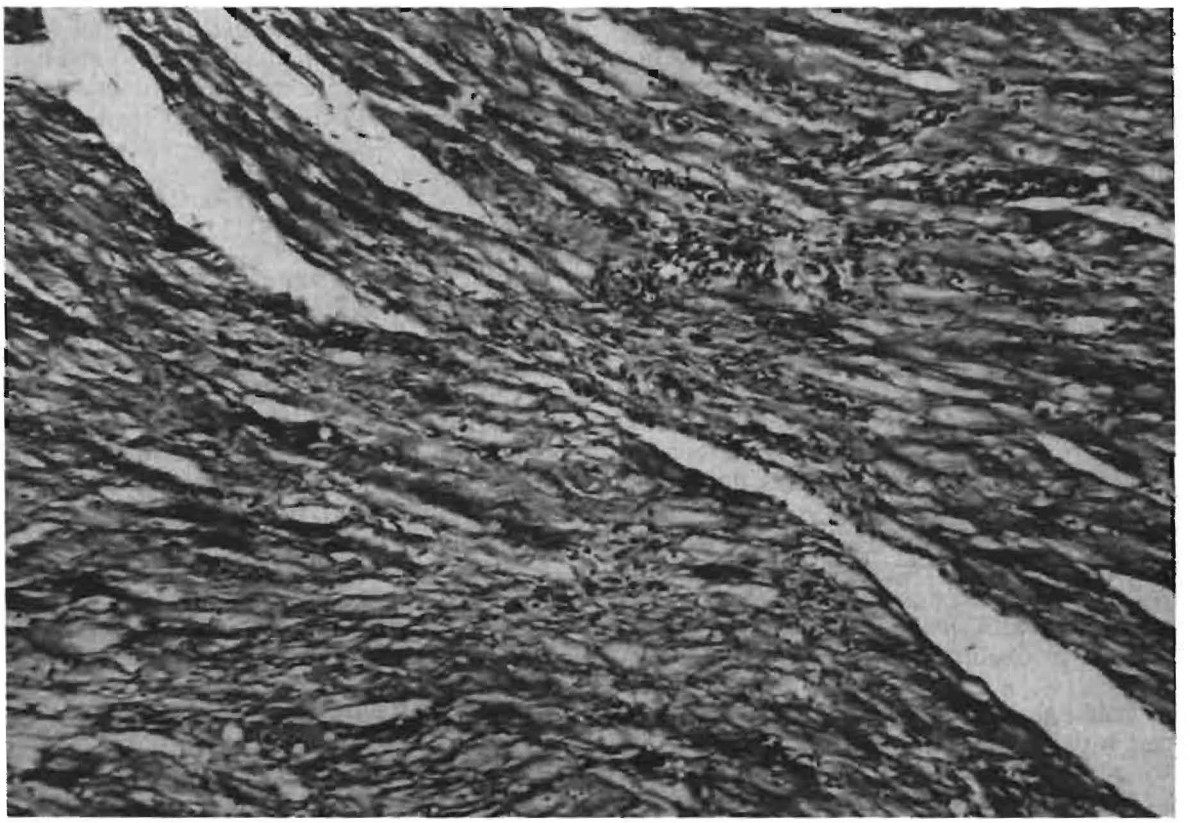

FIG. 5: Focus of vacuolation and loss of myocytes with fibroplasia in ovine myocardium; *Galenia africana* poisoning. HE X 200

known as "falling disease" is recognized in Australia. Affected animals die suddenly, and there is marked myocardial fibrosis[8]. Although certain soils in southern Africa are copper-deficient, this condition has not yet been reported here.

CENTRAL NERVOUS SYSTEM INJURY

The occurrence of multifocal myocardial necrosis following injury to the central nervous system has been documented in domestic animals, including a few cases in cattle, sheep and goats[24]. The diagnoses in these cases included listeriosis, polioencephalomalacia, meningoencephalitis and cerebral trauma. Systematic examination of the heart should not be neglected in cases of cerebral disease.

NEOPLASIA

We have seen lymphosarcomas with myocardial localization occasionally in cattle, but other neoplasms in the hearts of ruminants in southern Africa appear to be rare. A rhabdomyosarcoma in the heart of a sheep has been documented[7].

CONCLUSIONS

Morphological examination of ruminant myocardium is useful in the aetiological diagnosis of certain conditions such as white muscle disease, gousiekte and hairy vetch poisoning, but its value in the diagnosis of many conditions will remain limited unless the early manifestations of injury can be characterized and recognized.

The pathology caused by many cardiotoxic plants in southern Africa, some of which cause stock losses of economic importance, has received little attention and needs futher study.

When myocardial lesions are recognized, their nature often does not betray their specific cause. Myocardial reactions in response to injury by a variety of agents tend to follow a similar pattern of degeneration, necrosis, inflammatory cell infiltration and resolution, some aspects of which have been discussed previously[35]. Differences observed in lesions, therefore, often reflect only their stage of chronicity, rather than the nature of the injurious agent. Myocardial lymphocytic infiltration occurs in plant toxicoses and other toxicoses. It is clear such infiltrates do not necessarily indicate an infectious cause.

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BOOK REVIEW

BOEKRESENSIE

POULTRY DISEASES

R.F. GORDON and F.T.W. JORDAN

2nd edn. Bailliére Tindal, London 1983, pp 401 Price R42,25 (ISBN 0-7020-0907-5)

The second edition of Poultry Diseases has been altered to incorporate the important recent advances in poultry diseases with the inclusion of some diseases not mentioned in the first edition, viz. infectious coryza (*Haemophilus paragallinarum*), adenovirus, reovirus and neoplastic conditions. A new chapter on the avian immune system is a welcome addition. Unfortunately erosion diseases with a multifactorial aetiology related to ventilation and management deficiencies are not sufficiently emphasized while the chapter on how to conduct a field investigation is of considerable merit. The omission of certain chapters in the first edition does not in any way detract from the book's value.

Students and practitioners will find this volume very informative as the book provides a good overview of poultry diseases, their aetiology, symptoms and methods of control and should serve as as a useful companion book to other books on this subject.

L. Abrams

THE VETERINARY AND PARA-VETERINARY PROFESSIONS ACT ACT NO. 19 OF 1982

INTRODUCTION

The above Act replaces the Veterinary Act no 16 of 1933 and The Veterinary Amendment Acts of 1963, 1972, and 1974.

The Department of Agriculture was no longer prepared to administer and fund the workings of the juristic body responsible for controlling veterinarians in South Africa.

The Act had to provide for a period of transition and this was done by means of the Registrar and the Veterinary Board on a caretaker basis for a period of six months from the 1st October, 1982. Their main function was –

(a) to re-register all veterinarians in South Africa; and
(b) to organise the election, as prescribed, of a new Veterinary Council.

In a letter dated 1982/11/30, the Registrar notified all veterinarians of the coming into operation of the new Act and highlighted some important aspects, namely The S.A. Veterinary Council; The Register of Veterinarians; Continued Registration; The Maintenance of Registration; The Election of Members to the Veterinary Council; The Application of the Act to Veterinary Nurses and Change of Address.

The Registrar again communicated with all Veterinarians in a letter dated 1983/02/07 in which he informed them that the election for the S.A. Veterinary Council would take place on the 4th of March, 1983.

In both letters veterinarians were informed that any enquiries should be directed to Mr. Saayman. From the enquiries received it would appear that the whole question of registration was misunderstood, in many cases simply due to failure on the part of the veterinarian to read the documents posted to him.

The responsibility of the old administration, acting in a caretaker capacity, was by very deliberate intention engineered to expire on the 31st of March, 1983. The reason was very simply that this date was the last day of the financial year of the Department of Agriculture. It was no accident therefore that the new Act came into operation on October 1st, 1982. There was absolutely nothing that you as members or your Council (SAVA) could do about this Ministerial decision.

Details of registration follow:

Section 25(7)(b)(ii) Continued registration shall be subject to payment to the Council of an amount of R50,00 within 90 days of the commencement of this section (i.e. 82/10/01)

This payment of R50,00 was to provide for working capital for the new Council.

Section 26(i) of the Act determines that person registered in terms of section 25(i)(7)(a) may *maintain* such registration by paying annually the prescribed amount on or before the 1st April.

The current fee for maintenance of registration is –

R50,00 per annum for practising a veterinary profession
R25,00 per annum for practising a para-veterinary profession
R5,00 per annum for a student

The 90 day concession period for continued registration expired on 31st December, 1982, after which all persons who had not continued their registration, as prescribed, had to *re-register* at the following registration fees –

R75,00 for practising a veterinary profession
R50,00 for practising a para-veterinary profession
R10,00 as a student

Section 26(2) of the Act provides for exemption from payment of the whole or a portion of the prescribed maintenance fee by the Council as it may deem fit and subject to such conditions as it may in such case determine.

The S.A.V.A. has been informed that maintenance of registration over the age of 65 will be subject to an administration fee of R15,00 per annum. No other exemptions have been applied for and consequently no decisions have been taken. Your Association is, however, aware that many other categories possibly exist which deserve some form of exemption from the maintenance of registration fee – overseas membership, overseas study, pregnancy and child care leave, disabled members to name but a few. At the moment it is YOUR responsibility to apply for exemptions, but your comments in this regard will be welcomed by your Association. We do not administer the Act, but we do have our nominated representative on the Council. Send us your comments and complaints and we will brief our representative.

A paraphrase of the Act follows:

THE VETERINARY AND PARA-VETERINARY PROFESSION ACT, 1982 ACT NO. 19 OF 1982

A. Aim:

A.1 To establish and give powers and functions to a South African Veterinary Council;
A.2 To provide for the registration of persons practising veterinary professions and para-veterinary professions; and
A.3 To control such persons and unregistered persons.

B. The Philosophy:

The Act establishes the South African Veterinary Council as a body and juristic person to deliberate on its prescribed objects as follows:

B. 1 The registration of persons practising the veterinary professions;
B. 2 The regulation of the practising of such professions;
B. 3 The determination of a minimum standard of tuition and training to satisfy such registration;
B. 4 The exercise of effective control of the professional conduct of registered persons;
B. 5 The determinaton of the standards of professional conduct;
B. 6 The promotion of efficiency in and responsibility with regard to the practice of the professions;
B. 7 The protection of the interests of the professions;
B. 8 The maintenance and enhancement of the prestige, status and dignity of the professions;

B. 9 The maintenance and enhancement of the integrity of persons practicing the professions; and
B.10 Advising the Minister in relation to any matter affecting a veterinary profession or para-veterinary profession.

C. How the Act works:

The powers and functions of the Council enable it to achieve its objects as follows:

C.1 The aquisition or hiring of property;
C.2 The management (in broad terms) of such property;
C.3 The management (in broad terms) of negotiable instruments;
C.4 The spending and investment of funds;
C.5 The entering into of contracts;
C.6 Exercising or performing any power or function conferred or imposed upon it by or under this Act; and
C.7 Generally take such other steps as may be necessary to achieve the objects of the Council.

The Source of Funds is Prescribed

Proper finanical records must be kept and an audited balance sheet prepared for each financial year – This balance sheet being open to inspection at the Council's office by persons registered under this Act.

This Council must report to the Minister on its activities during the year at the end of each financial year. The viewing of such a report is prescribed.

Registers must be kept in respect of all persons whose applications for registration in terms of this Act have been approved and the qualifications for registration are prescribed.

The requirements for registration, details of registration, maintenance or alteration of registration and the termination of registration are covered in detail in the Act.

An unregistered person shall not practise veterinary or para-veterinary professions.

Any profession which has as its object the rendering of services supplementing the service deemed to pertain specially to a veterinary profession will be subject to the provision of the Act if so declared by the Minister – Para-veterinary professions.

Provision is Made for Student Registration

The Council may make rules to achieve or promote its objects or to exercise its powers or perform its functions.

The Minister may, on the recommendation of the Council, make regulations under the Act in order to attain or promote the objects of the Act.

A person registered to practise a veterinary profession may compound or dispense any medicine – provided he does not keep an open shop or pharmacy.

Arbitration in respect of fees charged for the rendering of a service is provided for and an unregistered person is specifically excluded from recovering renumeration for services rendered.

Employers may not demand that a registered person performs any work which he may not perform in terms of the rules.

In this Act a person accused of being unregistered or of having performed the act in respect of which the prosecution is instituted, for gain, is guilty until proved otherwise.

Provisions is made for Offences and Penalties

D. Administration of the Act is administered by the South African Veterinary Council a juristic body established under the Act and elected or nominated as follows:
D.1 Two officers designated by the Minister
D.1.1 a veterinarian of the Department of Agriculture.
D.1.2 an officer designated on account of his knowledge of law;
D.2 A representative of each university in the Republic which has a faculty of veterinary science – currently two;
D.3 A representative designated by the South African Veterinary Association; and
D.4 Six persons elected in the prescribed manner.

Provision is made for an association of persons representing the persons practising a para-veterinary profession to delegate a person who shall be co-opted as a member of Council whenever a matter affecting those persons is dealt with by the Council.

A member of Council holds office for a THREE year period, but may be redesignated or re-elected.

The persons who were members of the Veterinary Board (Section 1 of the Veterinary Act, no. 16 of 1933) constituted the Council for a period of six months after the commencement of the Veterinary and Para-veterinary Professions Act. no. 19 of 1982, on the 1st of October, 1982.

The qualifications of members of Council, the vacation of office and the filling of vacancies is prescribed.

A President and Vice-President are elected from their number by the newly constituted Council at its first meeting.

The President and Vice-President may not hold office for longer than two consecutive terms of office, but may vacate such office without terminating his membership of the Council.

The Council MUST meet THREE times at least each year.

Three Council members may call for a special meeting in writing and such meeting must be held within 30 days of the request.

The majority of members of the Council shall constitute a quorum for a meeting.

A decision of the Council is a decision by the majority of members present at the meeting.

The member presiding at a meeting has a casting and a deliberative vote in the event of an equality of votes.

A member may not miss two consecutive meetings of the Council without its permission.

The executive committee of Council shall be the President and two other members of Council designated by the Council and this executive committee shall exercise all the powers and perform all the functions of the Council between meetings. The executive committee may not change any decision of the Council and although its decisions are binding on the Council, they may be set aside by the Council.

The Council may establish other committees.

The Council shall appoint a Registrar for the purposes of the Act.

The Council may institute an inquiry into the conduct of a person who is registered or deemed to be registered

under the Act, or into an alleged act or omission by such a person in the practising of his profession or into an alleged contravention of the Act or the rules by such a person and the procedure at or for such an inquiry is laid down.

The Act confers disciplinary powers on the Council.

E. Delegated Powers:

The Act confers power on, assigns functions to and imposes duties on the Registrar appointed by the Council. These powers, functions and duties may be delegated to staff members acting under the control and direction of the Registrar.

Paraphrased by Dr. C.M. Veary

BOOK REVIEW **BOEKRESENSIE**

RADIOGRAPHIC TECHNIQUE IN VETERINARY PRACTICE

JAMES W. TIGER, D.V.M., Ph. D.

2nd Edn. W.B. Saunders Company, Philidelphia 1984 pp XII + 511Figs. 382. ISBN 0-Y216-8861-6
Price R109,50

The author of this book is a former Professor of Radiology of the University of Florida who left the "rewards and frustrations of teaching to establish a consultative and referral practice in veterinary radiography and radiology". He therefore understands the needs of those to whom his book is addressed, veterinary students, practitioners, and their technical assistants. The declared purpose of the book is to provide these people with a source of information on radiographic technique in veterinary practice.

The book is presented in 3 sections, the first entitled "Physical Principles", the second "Radiographic Positioning and Technique in Small Animals" and the third "An Atlas of Radiographic Positioning and Technique in Large Animals".

The first few chapters of Section I deal clearly with the theory of X-ray production, image formation and image recording. Here is included information about X-ray tubes, collimators, cassettes, intensifying screens, grids, film types and fluoroscopic screens. The author does not wish to burden his readers with unnecessary detail but I think that a little more information on the correct use of grids would have been helpful. Incorrect use of focussed grids especially can present many hazards for the inexperienced.

The chapter on dark-room theory and techniques is excellent, including as it does a section on the causes of unsatisfactory radiographs produced by both manual and automatic processing of films.

A comprehensive chapter on the selection of exposure factors and formulation of technique charts should take the guesswork out of radiography, even for adherents of the "point and shoot" method. Useful exposure charts for both small and large animals, on both small and larger machines, are included here, and can provide a useful starting point.

Planning and equipping a radiology department is dealt with, as is radiation protection. This latter chapter is necessary, factual, and sensible. There is a fascinating chapter on the costing and setting of fees for radiographs and Section I ends with information on making copies of radiographs, and on making slides from radiographs for projection purpose.

Section II and III constitute the atlases of positioning in small and large animals respectively. The importance of these Sections is underlined by the author's note that faulty preparation and positioning of patients are the major causes of radiographs which are not of diagnostic quality.

The format is similar throughout both these sections, examinations being arranged according to regional anatomy. The positioning of the patient is fully described verbally and shown clearly in a photograph. The photograph of the resulting radiograph is accompanied by an overlay line drawing illustrating the normal radiography anatomy. Sections II and III thus combine an atlas of positioning with an atlas of radiographic anatomy. Praise for this useful and instructive method of presentation cannot be too high.

Contrast media techiques are fully described in Section II. Here the author acknowledges his debt to the various specialists who have each contributed a chapter in their own field. These contributions greatly enhance the book. Materials and methods are discussed, the normal appearance of these studies described and helpful advice is offerred in dealing with complications which may occur.

Patients in Section I are shown anaesthetized where necessary or restrained without manual aid, in compliance with regulations pertaining in U.S.A.

A chapter on techniques in avian radiography closes Section II.

In Section III, the model patient is the horse, and the examinations described are those which can be performed with modest equipment on the standing, conscious animal. Modern anatomical terminology is used throughout the book, but the author is kind to the older veterinarian, and in this Section includes the previous terminology where this is helpful.

Throughout the book excellent bibliographies are provided at the end of each chapter. For example, over 70 references are listed at the close of the chapter on contrast urography.

This beautifully produced book extends to 511 pages, and its local prices of approximately R110 reflects its quality.

The purchase of this book can be confidently recommended, for its usefulness and excellence will not easily or speedily be surpassed.

I.E. Gordon

AAN DIE REDAKSIE — TO THE EDITOR

PROGRESSIEWE RETINALE ATROFIE (PRA) IN HONDE

Daar word PRA vry sertifikate uitgereik aan telers van honde wat 'n oftalmoskopiese ondersoek gehad het.

Aangesien PRA in honde eers heelwat later oftalmoskopies gediagnoseer kan word as met elektroretinografie moet daar dus groot versigtigheid aan die dag gelê word met die uitreiking van so 'n sertifikaat. 'n Dier kan reeds PRA onder lede hê terwyl dit klinies en oftalmoskopies nie gediagnoseer kan word nie.

Enkele voorbeelde is die Ierse Setter wat oftalmoskopies op 3-6 maande[1,2,3] gediagnoseer kan word en met retinografie op 4-9 weke[1,3], en die miniatuur Poodle oftalmoskopies op 2-3 jaar[1,2,3] en met retinografie op 10 weke[1].

'n PRA vry sertifikaat sal waarskynlik geldig wees vir 'n hond ouer as 5 jaar en negatief met oftalmoskopiese ondersoek (ook elektroretinografie indien daar twyfel bestaan) of honde ouer as 1 jaar en negatief met elektroretinografie[3].

'n Gesonde hond vrywaar egter nie sy nageslag nie aangesien PRA outosomaal resessief[1,2,3] is en daar dus gesonde draers voorkom.

BRONNE

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2. Rubin Lionel F 1974 Atlas of Veterinary Ophthalmoscopy Lea & Febiger, Philadelphia
3. Severin Glenn A 1976 Veterinary Ophthalmology Notes 2nd edn Colorado State University Fort Collins, Colorado

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BOOK REVIEW — BOEKRESENSIE

CONTROLLED RELEASE DELIVERY SYSTEMS

Edited by T.J. ROSEMAN and S.Z. MANSDORF

Marcel Dekker Inc., New York. 1983. pp. XV and 402, numerous tables and figures, Price S Fr 153
(ISBN-0-8247-1728-7)

This new text had its origins in the 8th International Symposium on Controlled Release of Bioactive Materials held in July 1981, in Fort Lauderdale, Florida. The 25 chapters represent contributions by a total of 77 authors.

The book provides a solid background of information on the present technology of controlled drug release system, for example drugs in polymer matrices, coated ion exchange membranes, matrices with magnetic particles, bioerodible polymers, miniaturized osmotic pumps, controlled release microcapsules etc. The text provides a fascinating glimpse at the future where "closed loop systems" may be used to administer drugs as they are needed. For instance a working system has been built for controlling the rate of insulin and dextrose infusions in diabetic patients. The computer-controlled apparatus is linked to a sensor which continuously monitors blood glucose. When blood glucose rises, insulin is infused and if blood glucose drops excessively, dextrose is infused.

Another interesting system described is the use of liposomes, small phospholipid molecules, as drug carriers. Water soluble drugs are enveloped by the liposome while lipid soluble drugs are dissolved in it. The liposome is itself lipid soluble and can act as a vehicle for the intracellular entry of the molecules it carries. This system has the potential for carrying enzymes, genetic material and drugs into cells. Polar, water soluble and macromolecular drugs do not readily cross the lipoidal cellular membrane so that the use of liposomes represents a potential advance of major proportions.

Pro-drugs are derivatives of active parent drugs which are synthesized to overcome shortcomings of the parent molecule. Shortcomings such as poor solubility characteristics, and susceptibility to enzymic degradation can be overcome by using suitable derivatives. Obviously the derivative must itself be biologically active or it must be biotransformed at the relevant site to the active moiety.

The development of implantable polymer matrices containing drug and magnetic steel beads is also described. The matrices release a constant low level of drug molecules until they are energized by an external oscillating bar magnet when they release a greatly increased (up to 30 times) number of molecules. The above system has great potential for the treatment of conditions requiring intermittent pulses of drug, for example, diabetes mellitus, and attacks of migraine.

In addition to purely medical applications, the book discusses controlled release in relation to insecticides, herbicides, molluscicides, marine antifouling chemicals and wood preservatives.

The book will be of general interest to research pharmacologists and to those involved in the pharmaceutical industry and of particular interest to scientists in the field of controlled release systems.

C. Button

AAN DIE REDAKSIE — TO THE EDITOR

TORAKSCHIRURGIE – WAT IS MOONTLIK

Dit word dikwels onder ons aandag gebring dat kollegas in praktyk, as gevolg van onkunde, eienaars van diere afskrik met onverantwoordelike stellings oor borskaschirurgie.

Meegaande stuk is dus om kollegas in te lig oor dié siektetoestande van die borskas wat wel chirurgies suksesvol behandel kan word.

Toestande soos byvoorbeeld *Spirocerca* granulomata, akalasie van die kardia, 'n blywende ductus arteriosus, regter aortaboog en stenose van die arterie pulmonalis word egter nie te dikwels in praktyk gediagnoseer nie terwyl dit geredelik voorkom.

Die gebrek aan gesofistikeerde toerusting verhoed dat gevorderde borskaschirurgie uitgevoer kan word en prostesis vir byvoorbeeld die slukderm en tragea is ook nie vrylik beskikbaar nie.

Opsommend word die struktuur, die tegniek en die toestande weergegee van die chirurgie wat wel uitgevoer kan word.

Slukderm	
Esofagotomie	Vreemde voorwerp
	Spirocerca lupi granulomata
	Ring-stenose
Esofagektomie	Gewas
	Spirocerca lupi granulomata
	Segment-stenose
Esofagogastrostomie	Reseksie van 'n te groot gedeelte van die slukderm
Wandreseksie of instulping	Divertikulum
Kardioplastiek	Akalasie kardia
Fistel ligatuur en biseksie	Trageo-esofageale fistel
Prostese	Eksperimentele chirurgie
Hegting	Skeure
	Perforasie
Tragea	
Trageotomie	Vreemde voorwerp
	Gewas
Ring(e)reseksie	Gewas
	Stenose
	Kollaps (nie te lank)
	Trouma
Prostese	Eksperimentele chirurgie
Hegting	Trouma
Longe	
Gedeeltelike of volledige lobektomie	Gewas
	Abses
	Sist
	Trouma
Hegting	Trouma
Bloedvate	
Ligatuur en biseksie	Blywende *ductus arteriosus*
	Ligamentum arteriosum
	Dubbel aorta-boog
	Ander vaskulêre ringstenoses
Bind van *A. pulmonalis*	Ventrikulêre septale defek
Arteriotomie	Stenose van *A. pulmonalis*
Tydelike sluiting van veneuse toevoer na die hart	Arteriotomie
	Ventrikulektomie
Limfvate	
Ligatuur	Chilotoraks
Hart	
Ventrikulektomie	Stenose van *A. pulmonalis*
	Hartwurm
	Vreemde voorwerp
Pleurale holte	
Parasentese	Pneumotoraks
	Hidrotoraks
	Hemotoraks
	Piotoraks
	Chilotoraks
Torakotomie	Soos vir Parasentese
	Gewas
	Abses
	Vreemde voorwerp
	Trouma
	Eksploratief
Diafragma	
Torakotomie (meer dikwels 'n laparotomie)	Troumatiese skeure
	Agenese

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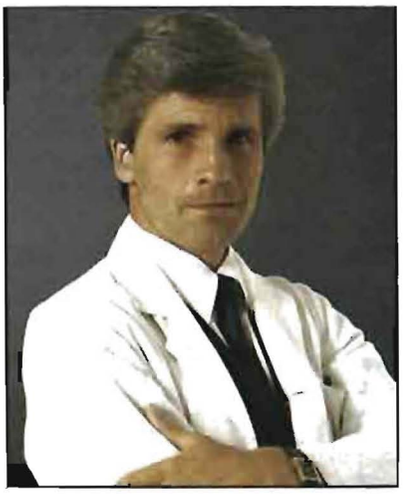

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Ogilvy & Mather Direct 826

BOOK REVIEW **BOEKRESENSIE**

STANDARD METHODS FOR COUNTING SOMATIC CELLS IN BOVINE MILK IN THE REPUBLIC OF SOUTH AFRICA

L.W. VAN DEN HEEVER, K.W. KATZ, J.D. PRINSLOO, W.H. GIESECKE, G. RAWLINS and A. JONES

Technical Communication No. 190, Department of Agriculture, Republic of South Africa.

1983 pp V and 8, ISBN 0 621 08254 6, Obtainable from Director, Division of Agricultural Information, Private Bag X144, 0001 Pretoria.

This Technical Communication sets out standard methods for the counting of somatic cells in bovine milk. The methods are based on recommended international standard methods which have been adapted for use under South African conditions. They comprise the Microscopic Somatic Cell Count and Somatic Cell Counting by means of the Coulter Counter and of the Tossomatic.

BOOK REVIEW **BOEKRESENSIE**

VETERINARY MEDICINE

D.C. BLOOD, O.M. RADOSTITS and J.A. HENDERSON with contributions by J.H. ARUNDEL and C.C. GAY

6th Edn. Bailliére Tindall, London, 1983 pp. 1310, Figs 22, Tabs 92, ISBNO-7020-0987-3, South African price R59,95.

This textbook has long been the standard reference work for large animal medicine in this country in the absence of a book of similar standard which is more directed towards diseases and conditions on the African continent. With this situation unchanged a new edition of this book is very welcome.

Despite the explosion of knowledge and development in large animal medicine the authors have greatly succeeded by limiting the contents but still maintaining a high standard. This was partially obtained by updating the lists of recommended review literature. However in my opinion some sections of the book contain some outdated practises. With the great emphasis on herd health in veterinary medicine today, the absence of a section on herd health programs can definitely be regarded as a shortcoming.

I still regard this as the best reference work in large animal medicine for both the veterinary student and the practising veterinarian. The use of very comprehensive tables in all the sections makes it a very good everyday reference book.

S. van Amstel

BOOK REVIEW **BOEKRESENSIE**

FOOD QUALITY CONTROL

A SYLLABUS FOR VETERINARY STUDENTS

HARRY V. HAGSTAD and WILLIAM T. HUBBERT

The Iowa State University Press, Ames, 1982 pp IX + 148 Figs. 23 Tables II ISBN 0-8138-0701-8. Price not mentioned.

The book is designed to serve as a teaching text in food hygiene for students in professional curricula of United States of America veterinary schools. The text is divided into 3 chapters, namely causes of food-borne disease, food production technology and consumer protection. At the beginning of each chapter the learning objectives for the subject material to be covered are listed. At the end of each section within a chapter a comprehensive list of references of further reading is provided.

Much of the information is more applicable to the United States. The book should be of use as a guide to those involved in teaching food hygiene to under-graduate veterinary and other health professional students. The syllabus gives an indication as to what depth food hygiene is dealt with at an under-graduate level in a food quality conscious country such as the United States.

G. Turner