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TYDSKRIF VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING

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JAARGANG 56 Nr. 2

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MYCOTOXINS

Mycotoxins are toxic metabolites of fungi which have undoubtedly presented a hazard to human and animal health since the earliest times. Thus it is surprising that the mycotoxicoses caused by these fungal toxins received little attention in the West prior to 1960. In that year the situation changed dramatically with the discovery that Turkey X disease in England was caused by aflatoxin in Brazilian groundnuts, and that this metabolite of *Aspergillus flavus* was not only hepatotoxic but also hepatocarcinogenic. In this context, it is remarkable that some of the pioneering work in mycotoxin research in the Western world was done in South Africa during the first half of the present century. The neurotoxic syndrome diplodiosis was reproduced experimentally in cattle with pure cultures of *Diplodia maydis* by D.T. Mitchell in 1918. This was probably the first mycotoxicosis that was reproduced experimentally in the target animal with a pure culture of a fungus. Sir Arnold Theiler also published on this mycotoxicosis in 1927. Shortly afterwards Prof Douw G. Steyn became very active in mycotoxin research at Onderstepoort and published a major review of fungi in relation to human and animal health in 1933. Since these early beginnings, active research programmes on mycotoxins have been and still are in progress at several research institutes and universities in South Africa including the Veterinary Research Institute, Onderstepoort; Plant Protection Research Institute, Pretoria; National Chemical Research Laboratory, CSIR, Pretoria; National Research Institute for Nutritional Diseases, South African Medical Research Council, Tygerberg; and the Universities of Pretoria, Natal, RAU and Medunsa. Two important symposia on mycotoxins have been held in South Africa, i.e. the Symposium on Mycotoxins in Foodstuffs, Pretoria, 1965 and the Symposium on Mycotoxins in Human Health, Pretoria, 1970. In view of the prominent role played in mycotoxin research by South African Scientists, it is commendable that the Sixth International IUPAC Symposium on Mycotoxins and Phycotoxins will be held at the CSIR in Pretoria from 22 to 25 July, 1985.

Mycotoxicological research in South Africa has resulted in the positive diagnosis of several economically important veterinary problems as mycotoxicoses. These include aflatoxicosis, *Aspergillus clavatus* tremorgenic syndrome, equine leukoencephalomalacia, facial eczema, lupinosis, *Paspalum* staggers, stachybotryotoxicosis and vulvo-vaginitis. In addition, compelling evidence has been presented that the mycotoxin sporidesmin is involved in the aetiology of geeldikkop, a photosensitivity disease of major economic importance in South Africa. These acute, fulminating outbreaks of mycotoxicoses are probably the tip of the iceberg and the long-term consumption of low levels of mycotoxins such as aflatoxin probably constitutes a much more serious veterinary problem. Thus the major economic portion of the mycotoxin problem in veterinary medicine lies under the surface and is mostly obscure and manifested mainly as reduced feed efficiency, retarded growth rate and predisposition to secondary infectious diseases due to the immunosuppressive effects of several mycotoxins.

It is well known amongst researchers and importers in countries such as Japan, the Republic of China, and the United Kingdom that the quality of maize and groundnuts imported from South Africa is amongst the best in the world. Groundnuts exported from South Africa are cleaned electronically and by hand sorting to reduce the level of aflatoxin contamination. While we export the best quality maize and groundnuts in the world, we should also be at pains to ensure that what remains behind in our country should be fit for human and animal consumption. When we are forced to import these commodities as was the case during 1983 and 1984, we should ensure that our country does not become a dumping ground for sub-standard products from other countries. The aflatoxin contamination of maize imported during this period reported in the news media was an insult to the high standards of mycotoxin research in this country and presented a real threat to human and animal health. The monitoring of these commodities should be done by independent organisations which do not have a financial stake in the products concerned and which make the results of their analyses known. The tolerance levels of aflatoxin and other mycotoxins in animal feeds and human foods in South Africa are regulated by several Acts. However, in order to guarantee the safety of feed and foods from mycotoxins and to prevent a recurrence of the imported maize fiasco of 1983/84, we need more than the existence of the Law. We need enforcement of the Law. Otherwise the Law is not only an ass, but also a smokescreen.

PRELIMINARY RESULTS OF NON-SURGICAL INTRA-UTERINE INSEMINATION OF SHEEP WITH THAWED FROZEN SEMEN

R. LAWRENZ*

ABSTRACT: Lawrenz R..**Preliminary results of non-surgical intra-uterine insemination of sheep with thawed frozen semen.** *Journal of the South African Veterinary Association* (1985) **56** No. 2, 61-63 (En). P.O. Box 1539, 9500 Kroonstad, Republic of South Africa.

Non-surgical intra-uterine insemination was achieved in 70 of 97 ewes (72%). Of the 70 ewes in which intra-uterine thawed semen was deposited, a total of 50 ewes (71%) conceived. Routine use of this technique is not at present foreseen due to the complicated and time consuming nature of the procedure.

Key words: Frozen ram semen, non-surgical intra-uterine insemination.

INTRODUCTION

The freezing of ram spermatozoa and the artificial insemination of ewes with thawed semen has been described as early as 1950[6 20]. During the past three decades artificial insemination with thawed frozen ram semen has been carried out on an experimental basis in many countries[15 16]. In order to permit the use of frozen semen in a rational breeding programme for sheep, many attempts have been made to solve special problems connected with the freezing and insemination technique[4].

The freezing of ram spermatozoa, according to the method described for freezing bull semen[19], was performed in 1950, but the percentage of surviving spermatozoa, after thawing, was very low[7]. Despite occasional satisfactory results obtained in this field, the use of thawed ram semen for artificial insemination cannot yet be regarded as ready for use in practice[1].

It is generally accepted that a serious loss in the fertilizing capacity of ram spermatozoa occurs during freezing[17]. Conception rates obtained after artifical insemination with thawed ram semen vary quite considerably. Salomon and Lightfoot[18] achieved a better conception rate with two inseminations (38,8%) compared to a single insemination (22,6%) at oestrus. Colas[3] reported that a single insemination with a double insemination dose resulted in a higher lambing percentage than a double insemination with a single dose (48,7% compared to 44,3%). With intracervical insemination Neves[16] obtained a 16,4% lambing rate after double insemination, 24 hours apart. These low conception rates may be due to impaired sperm transport through the cervix, since intra-uterine insemination has resulted in remarkably improved fertilisation rates[1 8 11-15].

Andersen et al.[1] used deep frozen ram semen in three experimental groups, depositing the thawed semen in various parts of the genital tract of the ewes. In the first group, consisting of 83 Norwegian Landrace ewes, a single intra-uterine insemination was carried out, whilst the second group of 60 ewes were inseminated once deep cervically. The third group of 21 ewes were inseminated twice, 12 hours apart, in the caudocervical region. The rates of conception in the groups were, 89%, 45% and 57% respectively. Following intra-uterine insemination, Linge[12] obtained a 75% lambing rate compared to 17% after single deep cervical insemination, and a 30% lambing rate after double caudocervical insemination. Notwithstanding the relatively good results achieved with intra-uterine insemination, the practical application thereof remains a problem, as it takes an experienced person to apply this technique[1 12]. In an experiment carried out by Edqvist et al.[5] it was shown that the supplementation of Prostaglandin F2α to semen shortly before freezing in liquid nitrogen resulted in a greatly increased number of spermatozoa in the uterus and oviducts following artificial insemination in the test group in comparison to a control group. Gustafsson et al.[10] reported that 7 out of 10 ewes lambed when inseminated with thawed semen to which Prostaglandin F2α has been added.

This paper describes preliminary results obtained with artificial insemination of ewes with thawed semen under field conditions in South Africa. The application of such a technique could possibly be beneficially used in South African sheep breeding programmes provided satisfactory conception rates are achieved.

MATERIALS AND METHODS

A total of 97 ewes kept on three different farms, were used for this study. Collection of semen from a South African Mutton Merino ram was done in July 1983 and the first group, consisting of 14 ewes, were inseminated in March 1984 (Group A). In Group B, where a total of 26 ewes was available for this trial, semen from a Merino ram was collected during March 1984 and used for insemination during July 1984. Semen from a Dorper ram was collected in June 1984 and used on Group C, consisting of 57 ewes, during August 1984.

Semen collection from each ram was done with an artificial vagina and the further processing done according to the method described by Günzel et al.[9]. Shortly before freezing an aliquot of 0,2 ml of diluted semen with a total of 200 $\times$ 20^6 spermatozoa was put into "Minitüb"® straws (Minitub GmbH, Abfüll and Labortechnik, D-8311 Tiefenbach, West-Germany). The semen was thawed in a waterbath at 70°C for 10 seconds, immediately prior to insemination.

The ewes in the different groups were synchronised using intra-vaginal sponges, each impregnated with 60 mg medroxy-progesterone acetate (Repromap, Upjohn). On Day 12, when the sponges were withdrawn, each ewe received an intramuscular injection containing 300 international units of pregnant mare serum (Fostim, Upjohn). Insemination of the ewes was only done on the second oestrus after sponge removal. Oestrus detection was done twice daily with the use of vasectomised rams. For the purpose of this experiment only one insemination per ewe, 12 hours after the onset of oestrus, was carried out.

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Table 1: THE RATE OF SUCCESSFUL INTRA-UTERINE INSEMINATIONS AND CONCEPTIONS IN THREE GROUPS OF EWES

	Total No. of ewes	Intra-uterine inseminations performed	Rate of success (%)	No. of ewes conceiving	Conception rate (%)
Group A	14	12	85,7	10	83,3
Group B	26	10	38,5	7	70,0
Group C	57	48	84,2	33	68,8

The procedure of intra-uterine semen deposition was based on the insemination technique as described by Andersen et al.[1]. The deposition of semen was done only in those ewes where intra-uterine insemination was possible. Ewes returning to oestrus after insemination were taken to a ram for handmating. Pregnancy examinations with the use of an ultrasonic intra-rectal probe (Medata Ultrasound Pregnancy Detector; Squab International, Veterinary Wholesalers, Bloemfontein) to determine foetal and or uterine arterial blood flow, were done on the 40th and again on the 70th day after insemination.

RESULTS

The rate of success of non-surgical, intra-uterine insemination, in the three different groups, is depicted in Table 1. The conception rates achieved following intra-uterine deposition of the thawed semen are also shown.

DISCUSSION AND CONCLUSION

The fairly good rate of conception following intra-uterine deposition of the thawed semen, as compared to results based on semen deposition among the folds of the external *os cervix*, was probably the result of a increased number of spermatozoa reaching the inner genitalia of the ewe and thus the site of fertilisation[1,12].

The marked difference in conception rates between Group A on the one hand, and Group B and C on the other, could be explained by the fact that the inseminations performed in the last two groups were at the very end of the natural breeding season, that is, during August and September, when oestrus activity reaches a low level. Another negative contributing factor is probably the drought experienced during that time, and the subsequent poor condition of the veld grazing.

With non-surgical intra-uterine insemination, only one insemination per oestrus seems to be necessary to establish a satisfactory conception rate[1,8]. There are, however, examples in which frozen ram semen used under ideal conditions, has resulted in quite normal conception rates when double inseminations in the *os cervix* are done[2].

With good flock management, it seems possible that intra-uterine artificial insemination with frozen thawed semen can be utilised in a breeding programme in stud animals. Ewes returning to oestrus after the first insemination should be identified and could then be brought to a ram for handmating. This technique could successfully be used in the different Group Breeding Schemes where the selection and mating is performed in the Nucleus Herds. Semen could be stored until the performance or even the progeny tests are completed. The best rams are then identified and the frozen semen used for the best ewes. With the increasing awareness of performance testing of sheep in South Africa, means and ways should be found of spreading the genetic material of superior rams over a much larger population of the national sheep flock. On the other hand, it is doubtful whether frozen ram semen can be used on an economical basis in commercial herds due to the difficulty of the insemination technique and the time required to carry out the procedure.

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BOOK REVIEW

BOEKRESENSIE

THE VETERINARY ANNUAL

C.S.G. GRUNSELL, F.W.G. HILL and MARY-ELIZABETH RAW (EDS)

25th Issue. Scientechnica, Bristol. Published by John Wright & Sons, 823-825 Bath Road, Bristol BS4 5NU, England. 1985 pp XIV and 400, illustrations & figures 106, tables 42. Price not given (ISBN 0-85608-938-X)

Previous issues of this renowned publication have received consistently high praise in Book Reviews of this journal. I find the 25th issue well up to the standard of its predecessors and equally praiseworthy.

The Veterinary Annual consists of concise review articles covering a wide range of important topics in veterinary science. In this issue, there are special articles on animal husbandry, reproduction and infertility, the new anaesthetic, isoflurane and protection of young animals from infectious disease. The cattle section includes articles on cerebrocortical necrosis, mastitis, leukosis, veal production and warble fly eradication. Articles on sheep deal with copper deficiency, coccidiosis, toxoplasmosis, early tooth loss, diagnosis of diarrhoea and disease surveillance. The pig section contains articles on routine procedures in piglets, induction of farrowing, *Streptococcus suis* type 2 infection, preventive medicine, atrophic rhinitis and porcine stress syndromes. Equine topics consists of rectal examination, electrolyte profiles and two orthopaedic conditions. The goat section deals with osteodystrophia fibrosa and paratuberculosis. The small animal section contains 19 articles which, as mentioned in the preface, are too widely diverse to group systematically. Nonetheless, all the topics included are of some importance.

A glance at the reference lists leaves no doubt that the contributions are as up-to-date as a review can be. In spite of this, special knowledge of the reader has not been assumed.

Some of the topics have been reviewed in previous issues, which I find attractive. For example, comparison of the article on cerebrocortical necrosis with that in the 20th issue provides an insight to the progress of research in this field.

The print and figures are of good quality, but I should have preferred more illustrations. It is mildly disappointing that the titles of papers are absent from most of the reference lists.

Much of the content has relevance to South Africa, and I am sure that there will be at least something of interest to all involved in any aspect of veterinary science in this country.

S.J. Newsholme

BOOK REVIEW **BOEKRESENSIE**

ANIMAL HEALTH IN AUSTRALIA VOLUME 5 PROTOZOAL AND RICKETTSIAL DISEASES

L.L. CALLOW

Australian Government Publishing Services, G.P.O. Box 84, Canberra, A.C.T.2601. 1984 pp ix and 264, illustrations 9, black and white photographs 13 and tables 15, Price not given (ISBN 0644022884)

This soft covered volume, which is one in a series, describes the protozoal and rickettsial diseases as they occur in Australia; however, extensive use is made of literature from other parts of the world, so that this book will also be useful to a wider reader audience.

The book is divided into 4 parts. Each part starts with a general consideration of the biology and taxonomy of the parasite followed by comparative aspects of the disease as it occurs in the various host species. Emphasis is placed on the biology and epidemiology of the parasite, followed by a fairly detailed description of the disease and its specific treatment.

Part one deals with the coccidia with chapters on coccidiosis, toxoplasmosis and sarcocystosis. Part two deals with the piroplasms with a detailed discussion of babesiosis and theileriosis as it occurs in Australia. The latter is primarily a discussion on *Theileria buffeli*. Part three deals with the arthropod-borne Rickettsias of the blood and include *Anaplasma*, *Eperythrozoon* and *Haemobartonella*. The final section deals with the Flagellates with sections on *Trichomonas*, *Tritrichomonas*, *Histomonas*, *Giardia*, *Spironucleus*, *Trypanosoma* and *Balantidium*. Reference lists are extensive to provide for those who desire greater insight into the subject.

This work is of a very high standard and a good source of reference for the serious students of protozoology. With a liberal use of personal communications and personal observations of the author, who is well recognised for his work in tick-borne diseases, this book offers a very informative analysis of the subject. It is highly recommended for anyone requiring more detailed information on protozoal and rickettsial diseases. Its main limitation is that Australia is free of many of the more serious protozoal and rickettsial diseases affecting animals.

C.G. Stewart

BOOK REVIEW **BOEKRESENSIE**

CLINICAL TEXTBOOK FOR VETERINARY TECHNICIANS

DENNIS M. McCURNIN

1st Edn. W.B. Saunders Company, Philadelphia. 1985 pp ix and 511, illustrations 455, tables 70 and one colour plate, Price 79,95 (ISBN 0-7216-1174-5)

Compared to one of the first books published for "Animal Health Technologists" (as veterinary nurses were first called in America), this book reflects the advancement in the standards of veterinary nursing training since the early 1970's.

This is the most comprehensive book yet published for veterinary nurses. The subject matter covers the whole range of activities including (surprisingly) necropsies, taking of necropsy specimen, bacterial isolation and identification procedures, antimicrobial susceptibility testing, collection procedures for milk cultures and somatic cell counts.

Thirty contributors, mainly from the Colorado State University wrote the twenty-four chapters covering restraint and handling of animals; history and physical examination; medical records; clinical pathology; parasitology and public health; microbiology; radiology; diagnostic sampling; emergency care; small animal, equine and food animal medical and surgical nursing; anaesthesia; surgical assistance and instrumentation; wound management and bandaging; exotic pet nursing; oncology; pharmacology; nutrition; basic necropsy procedures; hospital management; euthanasia and an appendix on abbreviations, not all of which are used in our country.

The subject matter covers most domestic animal species and includes turtles, iguanas, snakes and ferrets.

This is a very practically orientated book. One cannot, however, expect to find too much detailed knowledge on each specific subject in a book that covers such a very wide field.

The book can be recommended to veterinary nursing students, to qualified veterinary nurses as a reference guide and to veterinarians who wish to re-organize their practices to incorporate a veterinary nurse. They will be surprised at the wide field of procedures covered in the training of veterinary nurses today!

On the whole, a recommendable book.

I. Wolleschak

VALSIEKTE (*FALLING DISEASE*): A NERVOUS DISORDER IN LAMBS SUSPECTED OF BEING CAUSED BY THE PLANT *CHRYSOCOMA TENUIFOLIA*

F.H. VAN DER VYVER*, T.S. KELLERMAN**, STELLA S. BASTIANELLO***, J.A.L. DE WET****, J.P.J. JOUBERT***** and ADELE FAUL******

ABSTRACT: Van der Vyver F.H.; Kellerman T.S.; Bastianello Stella S.; De Wet J.A.L.; Joubert, J.P.J.; Faul, Adele. **Valsiekte (*Falling disease*): a nervous disorder in lambs suspected of being caused by the plant *Chrysocoma tenuifolia*.** *Journal of the South African Veterinary Association* (1985) **56** No. 2, 65-68 (En). Section of Pathology, Veterinary Research Institute, P.O. Box 12502, 0110 Onderstepoort, Republic of South Africa.

A description is given of the clinical signs and pathological changes in 23 field cases of *valsiekte* from the Bethulie region of the Orange Free State, Republic of South Africa. The disease, which occurred almost exclusively in 2-4 month-old Dorper or Dorper cross-bred lambs, was characterized by protracted ataxia, paresis and high mortality. Microscopical changes were consistently found in the neurons and white matter along the entire length of the spinal cord, and rarely in the *medulla oblongata*. These changes included vacuolation and degeneration of neurons, mainly of the lateral and ventral horns in the spinal cord, and a *status spongiosus* which was most noticeable in the lateral and ventral tracts of the spinal cord. All affected lambs had access to the plant, *Chrysocoma tenuifolia* (*bitterbos*), but trials to reproduce the condition by dosing the plant, were not successful.

Key words: Nervous disorder, neuronal vacuolation, neuronal degeneration, *status spongiosus*, lambs, poisonous plant, *Chrysocoma tenuifolia*.

INTRODUCTION

Ataxia or paresis in lambs in South Africa can occur in vitamin E-selenium deficiency[1,2], *krimpsiekte*[2] a chronic cardiac glycoside intoxication, copper deficiency[4] and botulism. As early as the 1930's Schulz[4] noticed paresis in lambs, with normal liver copper levels, during outbreaks of *kaalsiekte*[8] an alopecia due to *Chrysocoma tenuifolia*, in the Willowmore district of the Cape Province. In the late winter of 1979, a similar disease, named *valsiekte* (literally 'falling disease'), occurred in lambs in the Bethulie district and since then also in the Jagersfontein district, both in the Orange Free State, as well as in the Middelburg district of the Cape Province.

This paper documents the symptomatology and pathology of field cases of valsiekte as well as attempts to reproduce the condition experimentally.

HISTORY AND CLINICAL SIGNS

Outbreaks of *valsiekte* occurred periodically, especially in the winter months from June onwards or during droughts, when the veld was overgrazed and invaded by shrubs such as *Chrysocoma tenuifolia* (*bitterbos*) and *Rosenia oppositifolia* (*bekkerbos*). In answer to questionaires distributed to farmers in the 1980-81 season, they reported that grazing in a typical outbreak of *valsiekte* consisted mainly of 10-30% *C. tenuifolia*, and 10-80% *R. oppositifolia*. *R. oppositifolia* was not always present during outbreaks of *valsiekte*, and some farmers claimed that it was only eaten in the flowering stage. In 13 of 15 outbreaks, *valsiekte* was associated with *kaalsiekte*, a disease known to be caused by *C. tenuifolia*, but the reverse was not always true.

The districts where *valsiekte* have been reported are known to be high copper containing areas, where enzootic icterus is commonly encountered.

The disease had been found almost exclusively in Dorper or Dorper cross-bred lambs of 2-4 months-old. Affected lambs dropped out of the flock when driven hard; initially they only stumbled and knuckled over at the fetlock joints, but the hindquarters soon gave way, causing them to fall. In this position they struggled foreward, often on bent knees (Fig. 1), with their hindlegs dragging behind them (Fig. 2), frequently falling in sternal or lateral recumbency. After a short rest, less severely affected ones could get up. The lambs were not hypersensitive.

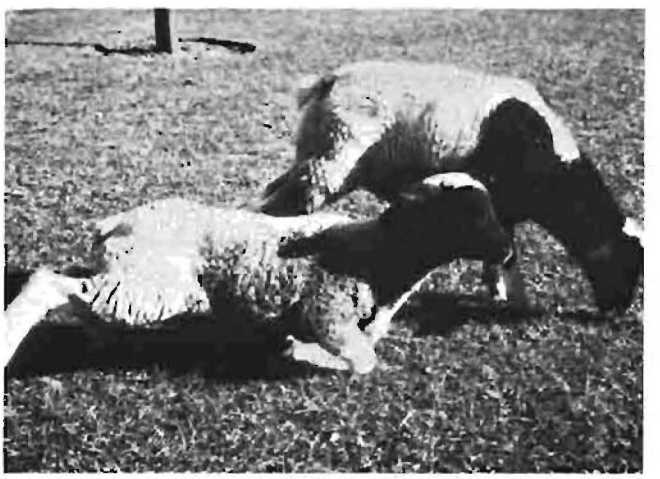

Fig. 1: A typical clinical presentation of 'Valsiekte', observed after lambs have been chased.

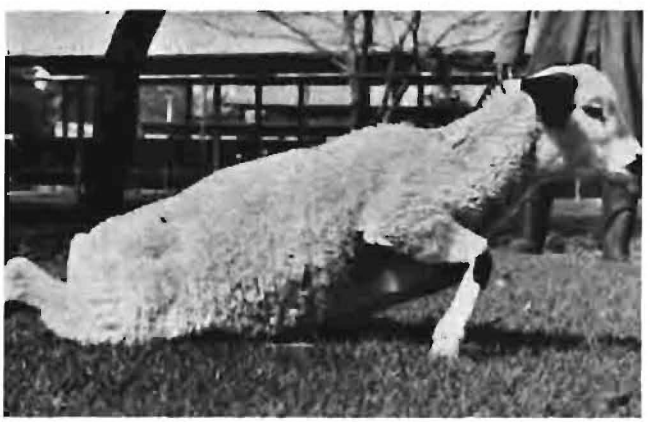

Fig. 2: Lamb dragging hindlegs.

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The paresis appeared to be irreversible and affected lambs sometimes died suddenly after a few weeks. With severely affected ones in lateral recumbency, euthenasia had to be performed, as they were unable to graze on their own. Apart from hindquarter ataxia and paresis the lambs maintained a normal habitus and appetite throughout. Many affected sheep therefore, maintained a fairly good condition. According to farmers, morbidity in a typical outbreak of *valsiekte* varies from 1-10%, with a mortality of 50-100%.

MATERIALS AND METHODS

Field cases:

The 23 cases studied, all from the Bethulie district, were constituted by 8 lambs from the 1979 season and 15 from the 1983 season. Necropsies were performed on 11 lambs, of which 9 were presented live with typical clinical signs, and 2 were dead on arrival. Only histopathological sections were examined of the other 12 cases. In all cases the tissues were fixed in 10% buffered formalin, routinely processed, embedded in wax and cut to a thickness of 4-6 μm. Sections were stained with haematoxylin and eosin and in 10 cases tissue blocks impregnated with Marchi's stain[6,10] were prepared for sections of the brain and spinal cord. In 17 lambs, specimens of the liver and sometimes also kidney were collected for mineral analysis by atomic absorption spectrophotometry. Copper levels of the liver and sometimes the kidneys were determined in 14 lambs (Table 1), whereas in 3 others more extensive determinations were done on their livers (Table 2).

Table 1: THE LIVER AND KIDNEY COPPER LEVELS (p.p.m.) ON A WET BASIS, OF AFFECTED FIELD CASE LAMBS

Sheep	Liver	Kidney
1	38,0	3,0
2	63,0	3,5
3	48,0	3,0
4	10,2	5,0
5	67,5	3,0
6	92,5	2,25
7	68,0	4,0
8	98,0	6,0
9	55,0	10,0
10	44,3	
11	72,2	
12	77,0	
13	45,0	
14	80,0	

Table 2: THE LIVER TRACE ELEMENT LEVELS (p.p.m.) ON A WET BASIS, OF AFFECTED FIELD CASE LAMBS

Sheep	Mg	Mn	Fe	Zn	Ca
1	179	3,4	82	20	57
2	228	4,2	102	22	42
3	220	4,0	141	27	42

The 2 predominant plants on the veld where outbreaks occurred were identified by the Botanical Research Institute, Pretoria, as *C. tenuifolia* Berg and *R. oppositifolia* DC Bremer.

Attempts to reproduce *valsiekte:*

In a limited trial, dry, milled *C. tenuifolia* and *R. oppositifolia* from a farm in the Bethulie district, were dosed seperately or together (1:1 mixture) to 3 pregnant Dorper ewes. The animals were given 19,4 – 25,8 kg of the material in doses of 5 g/kg over 151 days and received a total dose of 474 – 563 g/kg. During this period their lambs were born. In the last 25 days of the dosing programme, the ewes were additionally fed a concentrate meal containing 300 g of the respective plant materials. The lambs also had access to this ration, which was fed for a further 34 days after dosing of the ewes had ceased (Table 3). This additional daily ration was consumed in full by the ewes and lambs.

Table 3: DOSING AND FEEDING TRIAL OF *C. TENUIFOLIA* AND *R. OPPOSITIFOLIA* TO 3 PREGNANT DORPER EWES AND THEIR LAMBS

Ewe				Dosing regimen of ewes from day 0-151*			
No.	Initial mass (kg)	Age	Day lambed	Plant	Dose (g/kg x n)	Total dose (g/kg)	Total dose (kg)
1	46	2 tooth	119	*C. tenuifolia*	5 x 104	562	25,87
2	44	6 tooth	99	*R. oppositifolia*	5 x 105	525	23,10
3	41	6 tooth	90	*C. tenuifolia* + *R. oppositifolia*	5 x 100	474	19,46

*In addition each of the ewes (and their lambs after birth) were fed 300 g of the same plant material daily from day 126-185 (i.e. a total of 17,7 kg for each ewe and lamb over 59 days)

Different chemical determinations on blood samples of ewes, including sedimentation rate, red blood cell volume, haemoglobin, serum amino-aspartic transferase, serum urea nitrogen, glucose, total plasma protein, calcium, sodium, potassium, magnesium and phosphate were done at various intervals.

In a second trial 5 milk-tooth Dorper lambs of live mass 14 – 36 kg were dosed approximately 4 – 10 kg dry, milled *C. tenuifolia*, from toxic camps, at doses of 5 – 15 g/kg over a period of 10 – 86 days. The experiment continued for 16 – 89 days and the lambs received a total dose of 112 – 562 g/kg of live weight.

In a third trial 2 more milk-tooth Dorper lambs of live mass 17 kg were also dosed *c.* 10 kg dry, milled *R. oppositifolia* from the same toxic camps. They received doses of 5 – 10 g/kg over a period of 88 and 89 days, i.e. total doses of 542 and 562 g/kg, respectively.

RESULTS

Field cases:

In most animals traumatic hairless skin lesions were present on the carpal joints, dorsal aspects of the fetlocks, abdomen and other areas where they injured themselves during creeping episodes. Mild fatty changes in the liver were observed in some lambs.

The most consistent and significant microscopical lesions involved the spinal cord. Lesions were present along the entire length of the spinal cord and varied in degree from animal to animal and even in the same

animal. The changes were of 2 types, namely, a *status spongiosus* and neuronal degeneration and necrosis. A mild to severe *status spongiosus* involved the lateral and ventral tracts, and was most apparent in the lateral columns just below the points of entry of the dorsal nerve roots and at the periphery of the lateral and ventral columns. Marchi's staining method indicated varying degrees of demyelination (Fig. 3).

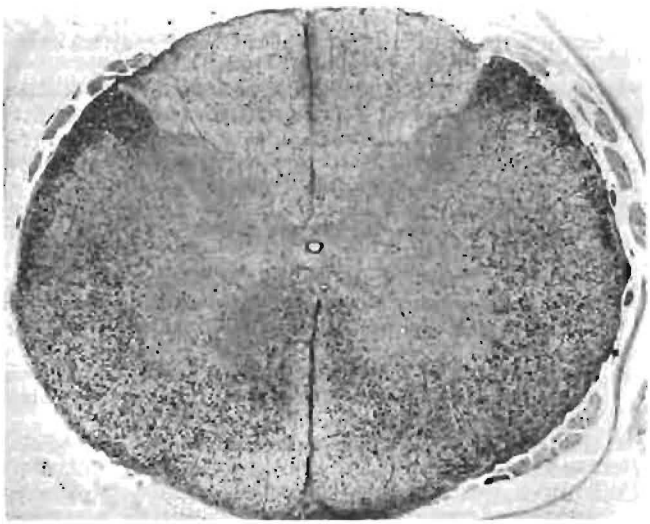

Fig. 3: Typical distribution of Marchi-positive material; thoracic spinal cord. Marchi and HE X 12

The neuronal changes varied in degree from small to large cytoplasmic vacuoles (Figs. 4 & 5), to enlarged neurons with pale, swollen eosinophilic cytoplasm and absence of Nissl's substance (Fig. 6). Their nuclei were often peripherally situated or absent.

Although the larger motor neurons of the ventral horns were more regularly affected, the neurons of the lateral and dorsal horns were also involved. In many spinal cord sections the degree of neuronal damage and demyelination did not always seem to be correlated. The same section could have severe neuronal lesions but only a mild *status spongiosus* and *vice versa*. Both types of lesions were, however, invariably present in all the affected lambs. Neuronal changes were also evident in the *medulla oblongata* in 2 animals.

Where mineral analyses were done, levels were found to be within normal limits.

Attempts to reproduce *valsiekte*:

Apart from a transient diarrhoea in some animals which received high doses of plant material, none of the ewes or lambs showed clinical signs of *valsiekte* and were not sacrificed for necropsy. No significant chemical pathological changes were recorded.

DISCUSSION

The clinical signs and histopathological lesions in the spinal cord of lambs with *valsiekte* bear a close resemblance to those induced by copper deficiency[3]. Contrary to the spinal cord, which was always affected, the *medulla oblongata* was involved in only 2 cases. Other signs of copper deficiency such as congenital swayback[5,7] and fleece abnormalities[11], were not reported from the regions involved in *valsiekte* outbreaks, and the liver copper levels of affected lambs were always within normal limits. The districts where *valsiekte* occurs are known to be associated with chronic copper poisoning[1] in sheep.

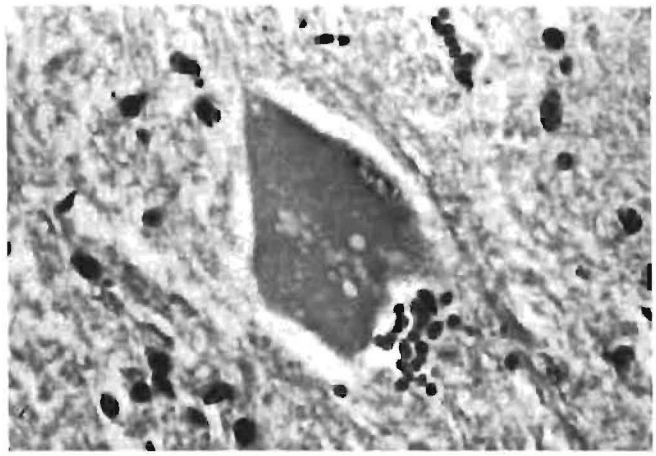

Fig. 4: Neuronal cytoplasmic vacuolation and peripheral nucleus; ventral horn of the spinal cord. HE X 800

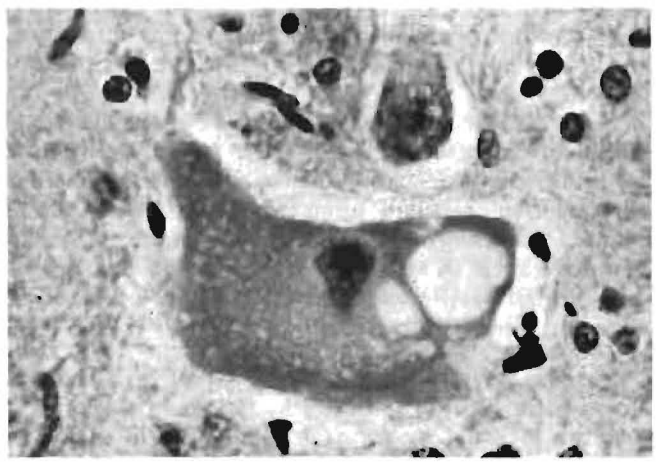

Fig. 5: Two large neuronal cytoplasmic vacuoles; ventral horn of the spinal cord. HE X 800

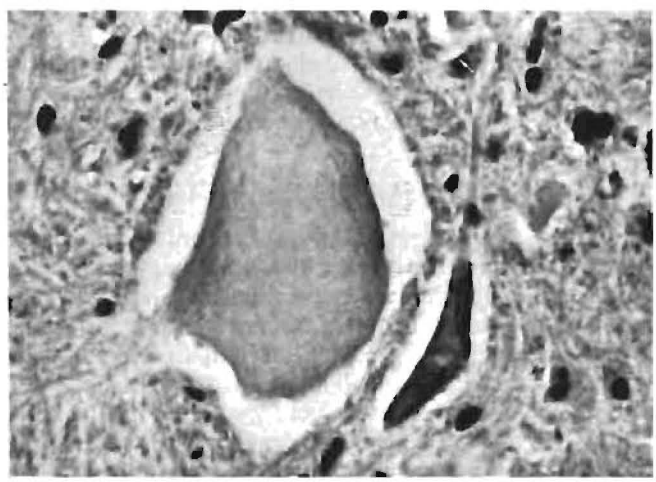

Fig. 6: Enlarged, pale neuron with loss of nucleus and Nissl's substance; ventral horn of the spinal cord. HE X 800

There is some circumstantial evidence to suggest that *C. tenuifolia* might be either directly or indirectly involved in the aetiology of *valsiekte*. The plant has always been present in outbreaks of the disease and in 13 of 15 outbreaks of *valsiekte* has occurred concurrently with *kaalsiekte*, which is known to be caused by *C. tenuifolia*[9]. The plant is known to cause 2 syndromes in sheep, namely alopecia (*kaalsiekte*) in new-born lambs whose dams browsed large quantities of *C. tenuifolia* during late pregnancy, and diarrhoea in mature animals ingesting large amounts of the plant. Since 1979 droughts and overgrazing have led to deterioration of the veld and invasion and spread particularly of *C. tenuifolia*. The disease occurs exclusively in the winter when the pasture is poor and animals are forced to eat the untasty *C. tenuifolia*. Dorpers being mutton sheep, tend to eat more voraciously than wool-producing breeds, and this may possibly explain why Dorper or Dorper crosses have almost exclusively been affected with *valsiekte*. Merinos tend to be more selective grazers and would seem to eat only small amounts of the untasty shrub.

R. oppositifolia, which is also an invading shrub, was not consistently present on the farms where *valsiekte* occurred.

Failure of the dosing trials does not exclude the possible involvement of either of these plants, as the low morbidity of *valsiekte* militates against its experimental reproduction in small numbers of sheep. Of the 2 plants, *C. tenuifolia* would be the more probable causative agent in view of the apparent coincidence of *valsiekte* and *kaalsiekte*, and the constant presence of the plant during outbreaks.

ACKNOWLEDGEMENTS

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BACTERIOPHAGE TYPING OF *STAPHYLOCOCCUS AUREUS* STRAINS ISOLATED FROM BLOEMFONTEIN DAIRY HERDS

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ABSTRACT: Swartz R.; Jooste P.J.; Novello J.C. **Bacteriophage typing of *Staphylococcus aureus* strains isolated from Bloemfontein dairy herds.** *Journal of the South African Veterinary Association* (1985) **56** No. 2, 69-73 (En). Department of Dairy Science, Faculty of Agriculture, University of the Orange Free State, P.O. Box 339, 9300 Bloemfontein, Republic of South Africa.

Bacteriophage typing was performed on 88 coagulase positive *Staphylococcus aureus* strains isolated during a survey of subclinical mastitis in Bloemfontein dairy herds. Phage typing was performed using two basic international phage typing sets, i.e. the human isolate phage set (HPS) and the bovine isolate phage set (BPS). The results clearly indicated that the BPS could be successfully applied for the phage typing of bovine mastitis *S. aureus* strains. The majority of the strains was typed as BPS phage group 1V (78,4 %) and HPS group III (47,7 %). The high prevalence of BPS group IV strains is in agreement with other studies. The prevalence of non-typable strains was 3,4 % for BPS and 28,4 % for HPS. Phages 102, 117, 107, 81, 47 and 6 had high lytic activity. BPS group IV patterns (102/107/117 and 102/117) dominated. The incidence of unique phage patterns was 12,5 % for BPS and 26,0 % for HPS. A relatively high proportion (71,3 %) of the strains was typable with the HPS. As these strains were of possible human origin it indicated the possibility of mutual human-animal transfer of the pathogens. No relationship could be found between phage groups on the one hand and multiple antibiotic resistance on the other, and no phage groups dominated within herds.

Key words: *Staphylococcus aureus*, bacteriophage typing.

INTRODUCTION

Bacteriophage typing provides a useful method for differentiating between strains of *Staphylococcus aureus* that are not obviously different when subjected to conventional morphological and biochemical tests. Phage typing can also be used for epidemiological studies of *S. aureus* infections and to trace the origin and distribution of infections, for example mastitis infections, in a dairy herd. It can also indicate whether a *S. aureus* strain is of human or animal origin[13].

Cultures of *S. aureus* are classified according to their susceptibility to a set of phages chosen to make as many epidemiologically valid distinctions between strains as possible. It is therefore a method of bacterial classification based on a single class of characters. Phage typing allows the staphylococci to be divided into a series of *phage groups* which include strains lysed only by one or more of a restricted set of phages, and the phages are classified into corresponding *lytic groups*[3 13]. The function of the International Subcommittee for *Staphylococcus* Phage-typing formed in 1953, is the standardization of methods and development of methods and basic phage sets to meet changing needs. The *Staphylococcus* Reference Laboratory of the British Public Health Laboratory Service (Central Public Health Laboratory, Colindale, London) became the international reference centre, and in 1961 was recognized as the World Health Organization Centre for *Staphylococcus* Phage-typing[13].

Initially only one basic phage set for typing staphylococci from human origin was available for the typing of all staphylococci. This phage typing system proved unsuitable for the classification of staphylococci from a number of other mammals for one or both of the following reasons[13]: either most of the animal strains were not lysed by the "human" phages at routine typing dilution (RTD), or those that were typable gave wide patterns that were unstable and difficult to interpret. A separate basic set of phages for typing bovine staphylococci was therefore developed after intensive investigations[3 13]. Oeding[12] emphasized the need for separate phage typing systems for staphylococci isolated from different animal species. A phage typing system aimed at coagulase-negative staphylococci has also been developed[18] and has already been applied by several workers[7 17].

Phage typing in the current study was performed using two basic international phage typing sets for coagulase positive staphylococci, i.e. the set for staphylococci of human origin (human isolate phage set = HPS) and the set for staphylococci of bovine origin (bovine isolate phage set = BPS). The object was to determine the most applicable phage typing set for the mastitis staphylococci and to indicate the percentage of strains of possible human origin. Phage lysis and patterns, the incidence of phage groups within herds, and the relation between phage patterns and multiple antibiotic susceptibility were investigated. Although extensive information exists on the phage typing of human staphylococcal isolates, corresponding information on bovine staphylococci is relatively scarce. Nevertheless, and attempt has been made to compare results with relevant studies. The only other South African study[6] was of primary importance in this regard.

MATERIALS AND METHODS

The propagation and testing of typing phages, technique of typing, media and interpretation of results was performed according to the recommendation of the *Staphylococcus* Reference Laboratory, as well as those of Blaire & Williams[1] and Parker[13]. The basic set of typing phages for bovine staphylococci was obtained from the Central Veterinary Laboratory, Ministry of Agriculture, Fisheries and Food, Weybridge, Surrey, England. In accordance with the recommendation of Blair & Williams[1] the recommended standard methods were closely followed. The phage typing results obtained could therefore be compared with results from other laboratories.

S. aureus Strains

Phage typing was performed on 88 coagulase positive *S. aureus* strains isolated during a previous study[15].

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Antibiotic Susceptibility Testing

Antibiotic susceptibility was determined by the Kirby-Bauer disc diffusion method[11] as described previously[16].

Typing Phages

Typing of *S. aureus* strains using the HPS was performed at the S.A. Institute for Medical Research, Johannesburg. The HPS comprised the following:

	Phages				
Lytic group I	29	52	52A	79	80
Lytic group II	3A	3C	55	71	
Lytic group III	6	42E	47	53	54
Miscellaneous	75	77	83A	84	85
	81	94	95	96	

The BPS contained the following lytic groups:

	Phages				
Lytic group I	29	52A			
Lytic group II	3A	116			
Lytic group III	6	42E	53	75	84
Lytic group IV	102	107	117		
	78	118	119		

Phage typing with the additional BPS phages not included in the HPS was performed at the Department of Dairy Science, University of the Orange Free State, Bloemfontein.

Propagation and Testing of Typing Phages and Routine Typing Technique

The procedures followed for the propagation and testing of typing phages and the routine typing technique are summarized in Fig. 1. Oxoid nutrient broth no. 2 (Oxoid Co.) was the medium used for suspending and

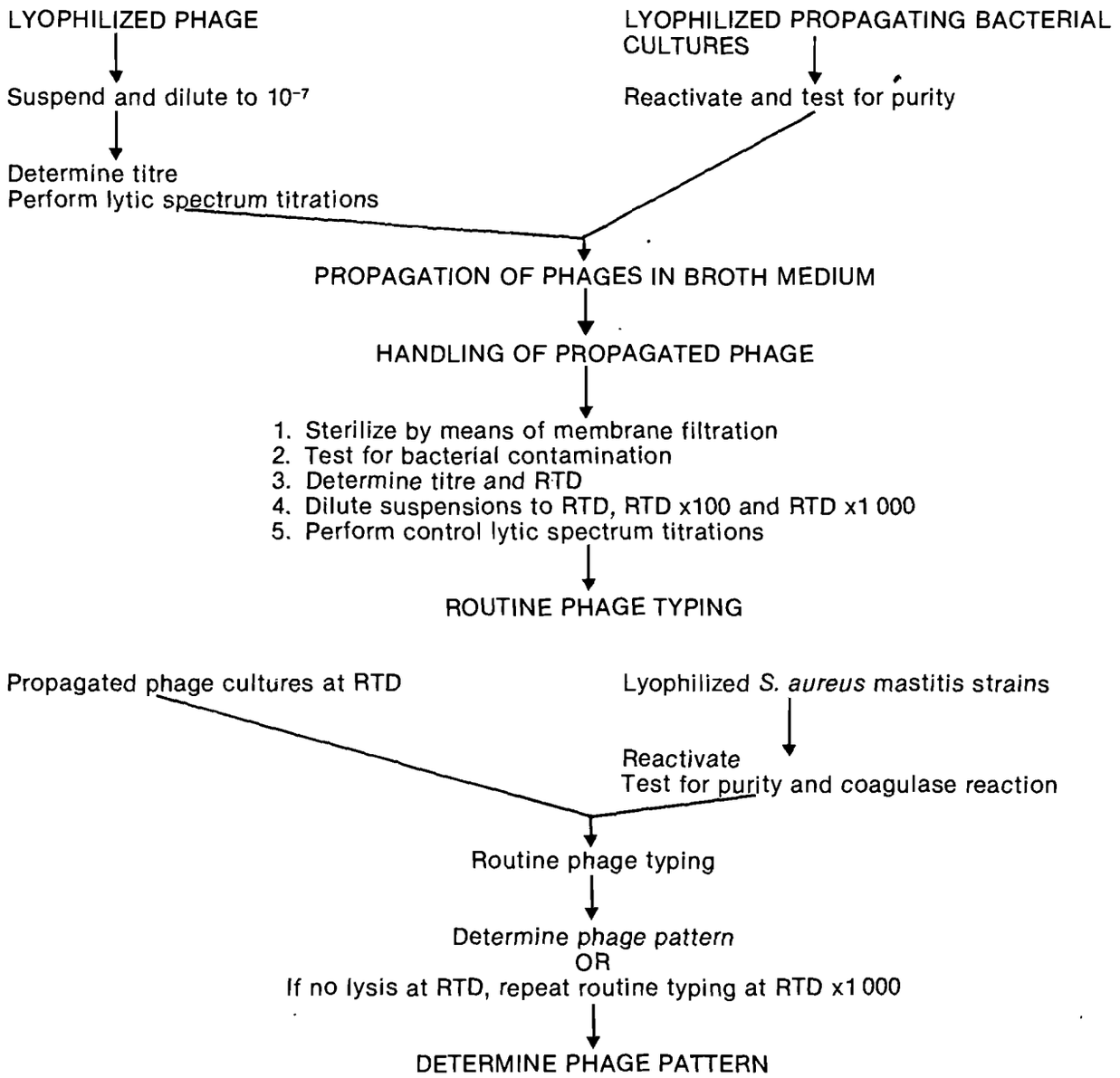

Fig. 1: Schematic representation of propagation and testing of typing phages and routine phage typing.

diluting of the phage cultures and cultivation of *S. aureus* strains. Oxoid nutrient agar enriched with 400 µg $CaCl_2$/ml was used for the preparation of typing agar plates. The quality of the phage cultures propagated from the lyophilized cultures was carefully monitored according to the procedure of Blair & Williams[1]. The proper testing of new lots of propagated phage cultures is regarded by these authors as the most important step in the production of typing material. Routine typing was completed within 5 days of phage propagation to ensure that the titre of phage suspensions was still high enough for typing.

Routine typing at routine typing dilution (RTD) was performed on all strains. Only in cases where no lysis could be observed at RTD, was typing repeated at RTD x100. RTD is defined as the highest dilution of phage that produces confluent lysis. Strong as well as weak positive reactions were regarded as positive, as recommended by Zierdt et al.[20]. To facilitate the subdivision into phage groups the reactions were classified according to the scheme of Blair & Williams[1]. The stronger (++) reactions thus carried more weight in the subdivision than the weaker lysis reactions.

RESULTS AND DISCUSSION

Phage Groups, Lysis and Patterns

Results regarding phage groups are given in Fig. 2 & 3 and phage lysis in Fig. 4 & 5. The majority of the strains was typed as BPS phage group IV (78,4 %) and HPS phage group III (47,7 %). The incidence of non-typable strains was 3,4 % for BPS and 28,4 % for HPS. Phages having a high lytic activity were phage 102 (83 % of strains susceptible), phage 117 (81 % of strains susceptible) and phages 107, 81, 47 and 6. The dominant phage patterns were 102/107/117 and 102/117, both belonging to BPS phage group IV. No phage pattern dominated for the HPS. The incidence of phage patterns seen only once was 12,5 % for BPS and 26,0 % for HPS.

The result indicate that the mastitis staphylococci could be more successfully phage typed by means of the BPS than the HPS. Several reasons can be cited for this phenomenon. In the first place, a higher incidence of non-typable strains was found for the HPS; secondly, the emergence of specific, dominant phage patterns for the BPS, while no phage patterns dominated in the case

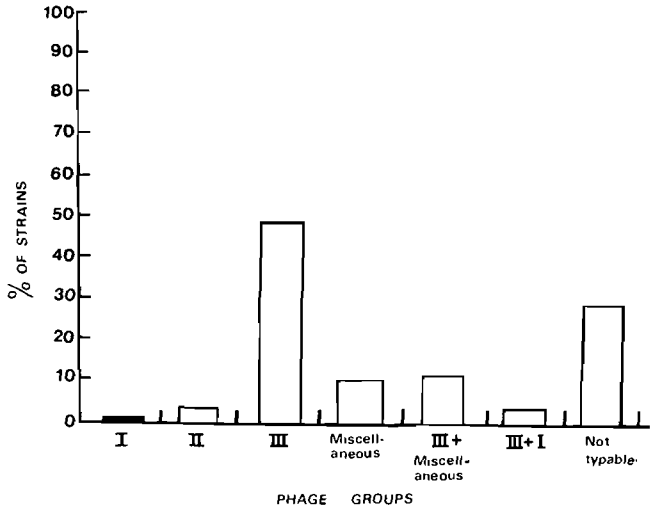

Fig. 2: Percentage of *S. aureus* strains in HPS phage groups.

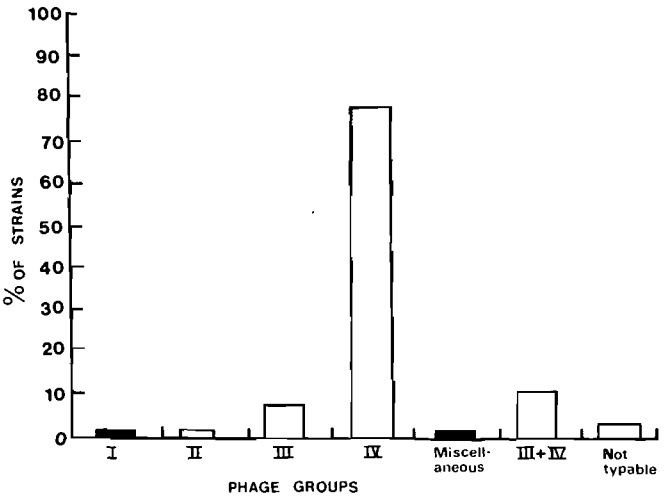

Fig. 3: Percentage of *S. aureus* strains in BPS phage groups.

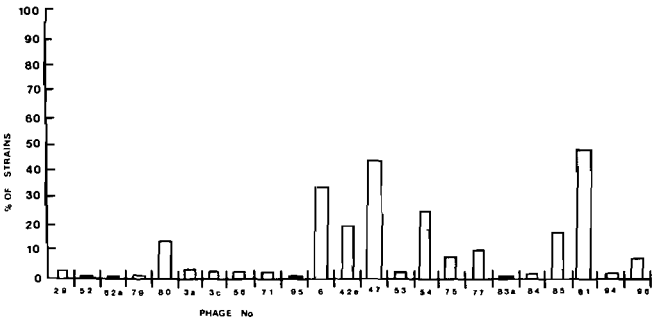

Fig. 4: Percentage of *S. aureus* strains lysed by HPS typing phages.

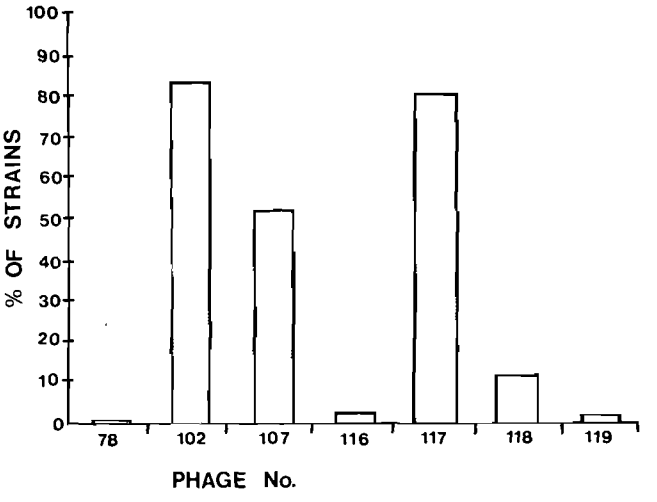

Fig. 5: Percentage of *S. aureus* strains lysed by BPS typing phages.

of the HPS, and finally, the high incidence of unique phage patterns obtained with the HPS.

The results of the current study as well as other relevant studies indicate that the BPS can still be successfully applied to bovine staphylococci. This is in contrast with the current tendency regarding the HPS typing of human staphylococci. Zierdt et al.[20] reports that over the last few years the proportion of unique strains and mixed phage group reactions have increased simultaneously. The mixing has reached such proportions that grouping on the basis of the current lytic groups has largely lost its significance.

Table 1: INCIDENCE OF ANTIBIOTIC MULTIRESISTANT *S. AUREUS* STRAINS IN BPS PHAGE GROUPS

Phage group	% Multiresistant strains
III	83 (6 strains)
IV	29 (69 strains)
III & IV	22 (10 strains)

Table 2: INCIDENCE OF ANTIBIOTIC MULTIRESISTANT *S. AUREUS* STRAINS IN HPS PHAGE GROUPS

Phage group	% Multiresistant strains
III	45 (42 strains)
Miscellaneous	0 (8 strains)
III & Miscellaneous	28 (25 strains)
Not typable	11 (9 strains)

It is of interest to compare the prevalence of the bovine mastitis staphylococci typable by the respective HPS and BPS sets. In the current study 96,6 % of the strains was typable by the BPS. This figure is in agreement with that obtained in the only other South African study[6], i.e. 93,6 % typable. The corresponding figure obtained using the HPS was, however, substantially higher (71,3 %) in the current study than in the previously mentioned study[6]. The corresponding figures of "human" strains causing bovine mastitis varies considerably in different studies: 40 %[2], 68 %[8], 0 %[9] and 91 % [10] human strains. The relatively high percentage of isolates of possible human origin in the current study is potentially hazardous[14]: raw milk can be a source of human *S. aureus* infections and humans can act as carriers of *S. aureus* strains capable of causing bovine infections.

The high incidence of BPS group IV staphylococci (78,4 %) in the current study corresponds well with the figures obtained in other studies[2 5 9 19] including the only other South African study (65 % group IV)[6]. The majority of strains in the study of Frost & Gradshaw[4] gave a complex pattern of lysis with group III and IV phages. In the only study deviating from the general pattern it was reported that phage groups III and 'Miscellaneous' dominated[8].

Phage Groups and Antibiotic Resistance

The incidence of multiresistant *S. aureus* strains are shown in Tables 1 & 2. Tables 1 & 2 can only be meaningfully interpreted when compared to the overall antibiotic resistance figure. Of the 88 strains tested 33 % was resistant to two or more antibiotics. The phage groups BPS III and HPS III contained the highest resistance figures for the respective phage sets. The finding of multiple resistance in the HPS III group is in agreement with current findings[6]. In the study of Giesecke et al.[6] no connection could be found between antibiotic resistance and lysis by the different phages. It should be noted, however, that the relatively low frequency of strains in each phage group makes it difficult to draw statistically reliable conclusions. A future increase in the proportion of phage groups containing multiple resistant strains could nevertheless present problems regarding antibiotic therapy of mastitis infections in the Bloemfontein region.

Incidence of Phage Groups within Herds

The incidence of phage groups within the 25 herds included in the study was investigated to determine whether specific phage groups dominated. No such conclusions could, however, be made as the distribution of phage groups within herds was similar to the overall distribution.

In summary, the most important finding of this study was that the BPS could be sucessfully applied to the typing of mastitis staphylococci. It was furthermore determined that the majority of the isolates belonged to phage group IV of the BPS. A disquieting finding was the high proportion of strains of possible human origin. This indicates the possibility of mutual transfer of *S. aureus* infections between humans and animals.

Future studies are essential for monitoring variations in phage patterns and for determining whether the BPS will remain applicable to the typing of *S. aureus* mastitis strains. Phage typing can also supply useful information for epidemiological purposes. It is clear therefore that future phage typing studies in South Africa and worldwide are needed to supplement the existing knowledge in this field.

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BOOK REVIEW

BOEKRESENSIE

REPRODUCTION IN THE DOG & CAT

Ib. J. CHRISTIANSEN

1st Edn. Bailliere Tindall, East Sussex BN 21 3UN, England. 1984 pp x and 309, illustrations 43 and tables 62, Price £12.75 (ISBN 0-7020-0918-0)

This is an extremely well presented and concise book dealing with reproductive physiology, gynaecology and andrology in the dog and cat. It is a soft cover edition in a format which will ensure its use as a textbook by pregraduate students and as a very handy reference guide by veterinary practitioners.

The book is divided into 2 sections. Part 1 deals with the dog and Part 2 with the cat. Both parts are divided into 10 similar chapters dealing with gynaecology of the normal female, breeding and mating, infertility and hormone treatment in the female, andrology of the normal male, infertility and hormone treatment in the male, artificial breeding and embryo transfer, limitation of fertility in the female and the male, pregnancy, parturition and neonates and finally dystocia, obstetrics and post-parturient problems.

The chapter on gynaecology of the normal female deals with the anatomy and physiology of reproduction including puberty, the breeding season and the oestrous cycle as well as the gynaecological examination of the female including the use of vaginal cytology and hormone profiles.

In the chapter on breeding and mating, the optimum time for breeding, the normal mating process and aberrations of mating behaviour are discussed. This is followed by a chapter on infertility and hormone treatment in the female in which anatomical and functional causes of infertility are briefly outlined as well as a brief discussion on hormone treatment.

The fourth and fourteenth chapters deal with the anatomy and physiology of the male and andrological examination including semen collection and evaluation. The fifth and fifteenth chapters give a very concise discussion of male infertility and hormone treatment while the sixth and sixteenth deal with artificial breeding and embryo transfer.

The subsequent chapter in each section provides a fairly detailed discussion on the limitation and artificial control of male and female sexual functions. Chapters eight and eighteen deal with pregnancy, foetal development, antenatal examinations and the diagnosis of pregnancy while chapters nine and nineteen deal with parturition, conception rates and litter sizes as well as care and pathological conditions of newborn puppies and kittens.

The last chapter in each section is devoted to a discussion of dystocia, obstetric care and postparturient problems.

This book should prove to be a valuable asset to the pregraduate veterinary students as well as to the practising small animal veterinarian and is recommended as such. The extensive reference list at the end of each chapter is considerably enhanced by the inclusion of a list of references for additional reading which should also facilitate the use of this book.

I can, however, not agree with the statement on the back cover that the book is also intended for ". . . breeders and owners with a scientific interest in their animals . . .". For that purpose I find it to be too scientific and precise, especially the chapters dealing with infertility and hormone treatment and limitation of fertility.

H.M. Terblanche

BOOK REVIEW BOEKRESENSIE

THE HENSTON VETERINARY VADE MECUM (LARGE ANIMALS)

A.H. ANDREWS and C.J. GILES

3rd Edn. Henston Limited, 1 Chilworth Mews, London W2 3RG. 1984 pp 608. Price £20.

This Veterinary Vade Mecum is distributed free to practising veterinarians in the United Kingdom and consists of two parts. Part I deals with diseases and conditions and part II lists therapeutic products. Numerous advertisements of pharmaceutical companies occur throughout the publication.

Part I is subdivided in the different large animal species but grouping sheep and goats together. For each species cross-indexed lists of diseases and conditions are given, that is then followed by an alphabetical list describing the disease profile, clinical signs, post mortem findings, principal differential diagnoses, treatment and control. As can be expected in a publication of this size and the wide field of interest, only a brief description can be given. The information given is, however, very useful and especially the differential diagnoses could be very handy to the practitioner. As the publication is intended for use in the United Kingdom many conditions important to the veterinarian in South Africa are not included and this will limit the practical use of the publication.

Part II which consists of 267 pages deals with the therapeutic products. Included also is information on the worldwide livestock population, oestrus control and other matters important to the local veterinarian.

The summary tables list in alphabetical order over 1300 therapeutic products. These have been compiled into tables according to their therapeutic class. The product name, the name of the manufacturing company, the active principle(s), presentation and the species where it is recommended are included. Where applicable, witholding time for meat and milk is given. The tables listing endoparasiticides and ectoparasiticides also give some information on the parasites affected by the active ingredient.

A wide spectrum of medicines are included but once again there is such a difference between the availability of products between our country and the United Kingdom that these lists will have limited value.

At the end of this part some information is listed on normal haematological data and units for clinical chemistry. The figures listing some physical and chemical drug incompatibilities and possible drug interactions, could be very useful for every veterinarian.

The Henston Vade Mecum is made up of two sections "bolted" together. The reason for this is that the information in Part I is unlikely to change as frequently as Part II. The publisher envisaged that only Part II will be amended annually which then could be replaced.

In summary it can be said that this book offers useful information but the veterinarian in this country should take note of the limitations.

A. Immelman

BOOK REVIEW BOEKRESENSIE

BIRDS: THEIR STRUCTURE AND FUNCTION

A.S. KING and J. McLELLAND

2nd Edn. Baillière Tindall, 1 St Anne's Rd., Eastbourne, East Sussex BN 21 2 UN, England. 1984, pp viii and 334, illustrations 123, Price £9.50 (ISBN 0-7020-0872-9)

This book is a revised and enlarged edition of the critically acclaimed Outlines of Avian Anatomy, first published in 1975 by the same authors. Although intended as a general introduction to avian anatomy for students of veterinary science, zoology and comparative vertebrate anatomy, the addition of many topics of general interest also make it of value to ornithologists.

The new edition employs a bolder print which makes it easier to read. Many of the original illustrations have been redrawn and new ones added to support and enhance the text. The bibliography for further reading has been updated and expanded, the relevant references being incorporated at the end of each chapter in alphabetical order. The anatomical nomenclature is now based on the Nomina Anatomica Avium, 1979, the Latin terms being converted into their English equivalents.

The text, supported by the illustrations, gives a very concise but clear description of the morphological features of birds. The first chapter deals with the evolution, energetics and classifications of birds, while the second deals with the external morphological characteristics of birds on a comparative basis. The first two chapters are of a general nature and will be of particular interest to the ornithologist. The rest of the book is devoted to a detailed description of the anatomical features of birds, including the lymphatic and nervous systems, with a chapter on the special sense organs. For those interested in greater detail, a complete alphabetical list of publications for further reading is given at the end of every chapter.

This book should be of great value to anyone interested or working in the field of avian anatomy.

A.J. Bezuidenhout

BLOOD CHEMICAL AND ELECTROLYTE CONCENTRATIONS IN THE OSTRICH *STRUTHIO CAMELUS*

J. VAN HEERDEN*, J. DAUTH**, M.J.F. JARVIS***, R.H. KEFFEN°, J.E.F.M. DENNY+, M.J. DREYER** and N.P.J. KRIEK+

ABSTRACT: Van Heerden J.; Dauth J.; Jarvis M.J.F.; Keffen R.H.; Denny J.E.F.M.; Dreyer M.J.; Kriek N.P.J. **Blood chemical and electrolyte concentrations in the ostrich *Struthio camelus*.** *Journal of the South African Veterinary Association* (1985) **56** No. 2, 75-79 (En). Department of Medicine, Faculty of Veterinary Science, University of Southern Africa, 0204 Medunsa, Republic of South Africa.

Serum levels of sodium, potassium, chloride, phosphorus, iron, total magnesium, total calcium, alkaline phosphatase, alanine transaminase, lactate dehydrogenase, creatine kinase, γ-glutamyltransferase, aspartate transaminase, urea, creatinine, total protein, albumin and plasma glucose were determined in 49 ostriches (*Struthio camelus*) kept under semi-extensive conditions.

Key words: Ostrich, *Struthio camelus,* blood analysis, blood chemical and electrolyte concentrations.

INTRODUCTION

The determination of the concentrations of blood constituents is widely used in veterinary science in the diagnosis of various metabolic, nutritional and organ disorders. The ability to use these parameters clinically depends on a knowledge of the concentrations in physiologically normal and healthy individuals.

This paper aims to present base-line values of certain blood constituents in ostriches (*Struthio camelus*).

MATERIALS AND METHODS

Blood specimens were collected from 49 ostriches from a commercial breeding unit. They were kept under semi-extensive conditions in a paddock with some natural vegetation and were fed commercial grower pellets (Senwesco) formulated for ostriches.

Most of the birds were 1-2 years old; 5 were 3 years of age. Twenty-nine of the birds were females and 20 were males.

The birds, which were accustomed to the presence of human beings, were herded into a small enclosure where most of them struggled when they were captured. The heads of the birds were covered with a hood to facilitate handling. They were physically restrained in a crush for the collection of blood specimens. Blood specimens were collected in evacuated tubes (Vac-u-test, Radem Laboratory Equipment) by venipuncture of the wing vein. After clotting, the blood was centrifuged and the serum was drawn off and stored in tubes in a cool bag during transport to the laboratory for approximately 2 hours. Blood specimens for glucose analysis were collected in evacuated tubes (Vac-u-test, Radem Laboratory Equipment) containing oxalate and sodium fluoride.

Serum specimens were analyzed by a Technicon™ SMA™ II system (Technicon Instruments Corp., Tarrytown, New York, USA) which used the following methodologies:

Sodium (Na^+) and potassium (K^+) levels were determined simultaneously with a flame photometer using lithium sulphate as an internal reference standard.

Chloride (Cl^-) levels were determined after addition of acidic mercury thiocyanate and ferric perchlorate reagents.

Phosphorus levels were determined using ammonium molybdate in a hydrochloric acid medium. Phosphomolybdic acid which formed was in turn reduced by stannous chloride hydrazine sulphate with the formation of molybdenum blue.

Iron (Fe^{3+}) levels were determined after liberation of iron from its carrier protein by hydrochloric acid. The liberated iron was then complexed with FerroZine in a sodium acetate buffer solution. In order to reduce protein and copper interferences, sodium chloride and neocuproine hydrochloride were added.

Urea levels were determined using a weak acid solution and a colour reagent which contained diacetylmonoxime and thiosemicarbazide.

Creatinine levels were determined using a modified Jaffe method.

Total protein (TP) was determined by using the Biuret reagent. A blank channel for the purpose of correcting for all intrinsic interferences was run simultaneously.

Albumin (ALB) levels were determined by using the Bromcresol Green (BCG) dye binding technique.

Except for Na^+ and K^+, colorimetric methods were used for the determination of all the above mentioned constituents.

Plasma glucose levels were determined with an ASTRA™ 8 discrete analyzer (Beckman Instruments Inc., Clinical Instruments Division, Brea, California, U.S.A.) utilizing the glucose oxidase method where the rate of oxygen consumption is measured.

Total serum calcium (t-Ca) and total serum magnesium (t-Mg) levels were determined with a Perkin-Elmer 5500 atomic absorption spectrophotometer (Perkin-Elmer Corp., Analytical Instruments Norwalk U.S.A.) using an air-acetylene flame (oxidizing) and a 0,16% (w/v) lanthanum oxide solution as diluent. The different wavelengths used for t-Ca and t-Mg determinations were 422,7 nm and 285,2 nm, respectively.

The activities of the following serum enzymes were measured at 30°C with a Flexigem centrifugal analyzer (Electro-Nucleonics Inc, Fairfield, New Jersey, U.S.A.) and all the reagent kits used for these determinations were Gemini™ manufactured for Electro-Nucleonics Inc. by E. Merck, Darmstadt, F.R. Germany utilizing the following methodologies:

Alkaline phosphatase (ALP): p-nitrophenylphosphate which is used as substrate is dissociated by ALP into p-nitrophenol and phosphate. The change in absorbance per time is measured at 405 nm.

Alanine transaminase (ALT): ALT reacts with α-ketoglutarate and L-alanine in a buffered solution with

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Table 1: THE MEAN ($\bar{X}$), STANDARD ERROR OF THE MEAN (SE), COEFFICIENT OF VARIATION % (CV%), VARIANCE (VAR) AND STANDARD DEVIATION (SD) FOR THE DIFFERENT CONSTITUENTS EVALUATED IN OSTRICH BLOOD.

	n	$\bar{x}$	range	SE	CV%	VAR	SD
Na mmol/ℓ	49	150,57	142 – 162	0,61	2,86	18,50	4,30
K mmol/ℓ	49	3,45	2,4 – 6,8	0,13	26,64	0,84	0,92
Cℓ mmol/ℓ	49	103,87	96 – 110	0,50	3,39	12,40	3,52
Phosphorus mmol/ℓ	49	1,73	0,98 – 3,64	0,06	23,62	0,16	0,40
tMg mmol/ℓ	49	0,81	0,54 – 1,34	0,02	18,90	0,02	0,15
tCa mmol/ℓ	49	2,51	1,88 – 2,80	0,02	6,50	0,02	0,16
Fe µmol/ℓ	49	16,15	2,2 – 90,0	3,23	140,11	512,58	22,64
ALP U/ℓ	48	478,97	150 – 1 384	36,23	52,41	63 015,25	251,02
ALT U/ℓ	49	2,85	0 – 12	0,36	88,08	6,33	2,51
LDH U/ℓ	48	1 041,35	240 – 2 187	57,62	38,34	159 383,35	399,22
CK U/ℓ	49	1 528,97	394 – 2 500	104,52	47,85	535 282,35	731,62
GGT U/ℓ	49	0,48	0 – 6	0,17	236,20	1,33	1,15
AST U/ℓ	49	236,97	100 – 892	19,48	57,54	18 591,60	136,35
Urea mmol/ℓ	49	0,49	0,2 – 1,2	0,03	43,48	0,04	0,21
Creatinine µmol/ℓ	49	36,57	6 – 64	1,86	35,61	169,62	13,02
TP g/ℓ	49	40,40	26 – 50	0,70	12,16	24,16	4,91
Alb g/ℓ	49	20,04	14 – 24	0,32	11,25	5,08	2,25
Glucose mmol/ℓ	48	9,70	3,6 – 14,6	0,34	24,52	5,66	2,38

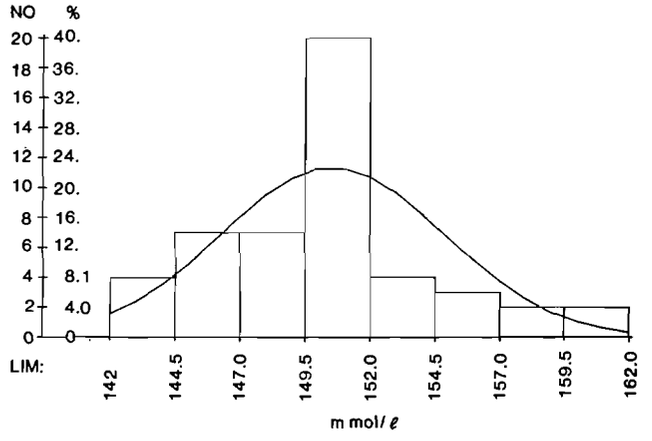

Fig. 1: The frequency distribution of sodium (mmol/ℓ)
LIM: = limit NO: = number of ostriches

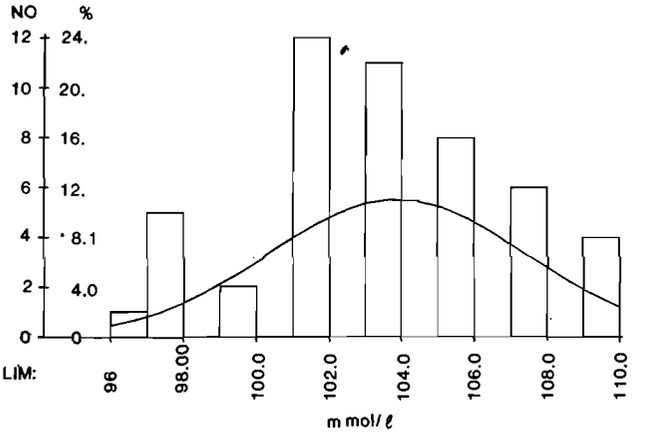

Fig. 3: The frequency distribution of chloride (mmol/ℓ)
LIM: = limit NO: = number of ostriches

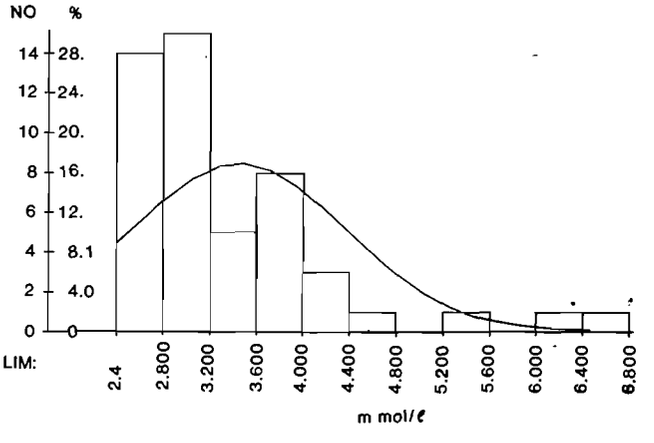

Fig. 2: The frequency distribution of potassium (mmol/ℓ)
LIM: = limit NO: = number of ostriches

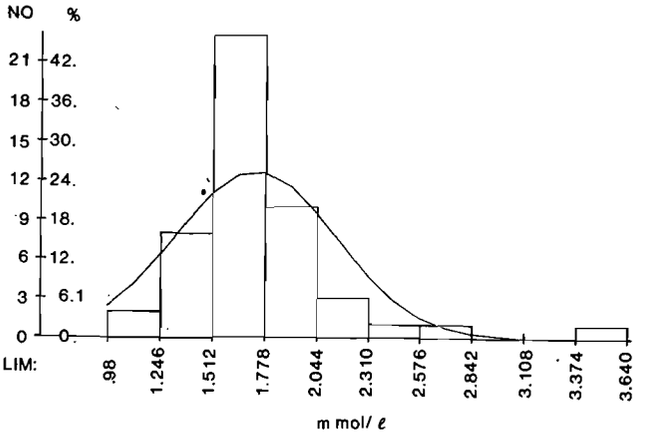

Fig. 4: The frequency distribution of phosphorus (mmol/ℓ)
LIM: = limit NO: = number of ostriches

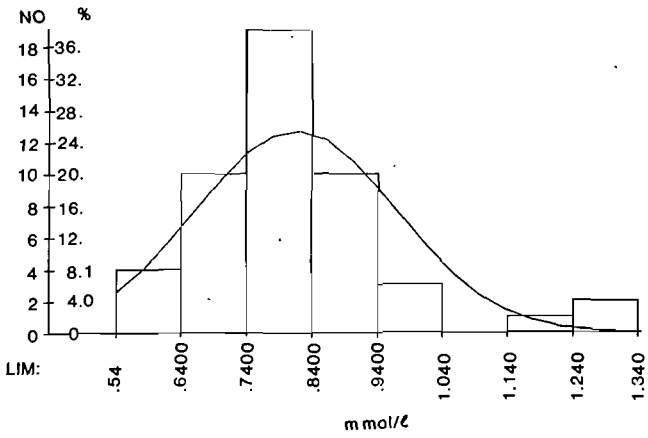

Fig. 5: The frequency distribution of total magnesium (mmol/ℓ)
LIM: = limit NO: = number of ostriches

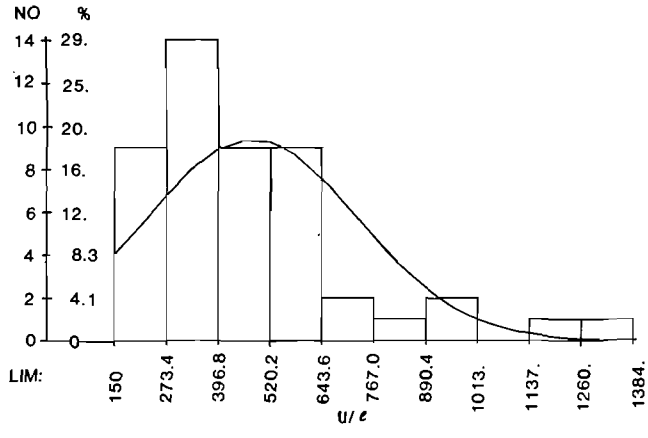

Fig. 8: The frequency distribution of alkaline phosphatase (U/ℓ)
LIM: = limit NO: = number of ostriches

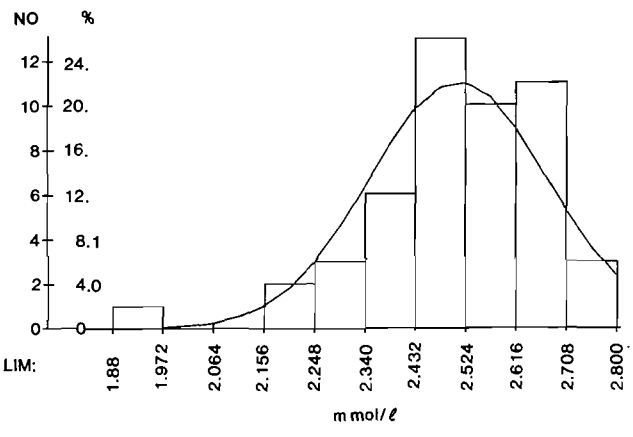

Fig. 6: The frequency distribution of total calcium (mmol/ℓ)
LIM: = limit NO: = number of ostriches

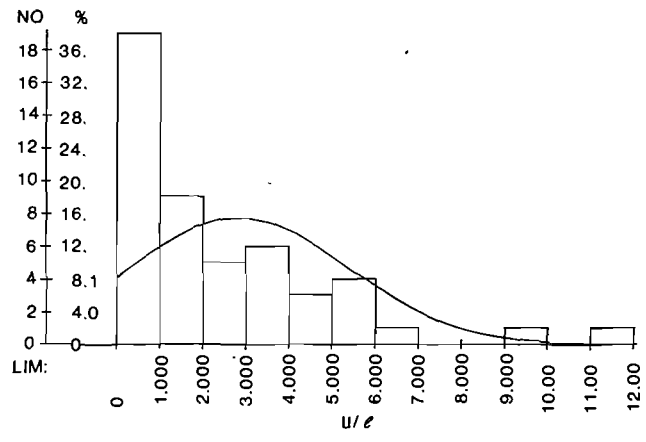

Fig. 9: The frequency distribution of alanine transaminase (U/ℓ)
LIM: = limit NO: = number of ostriches

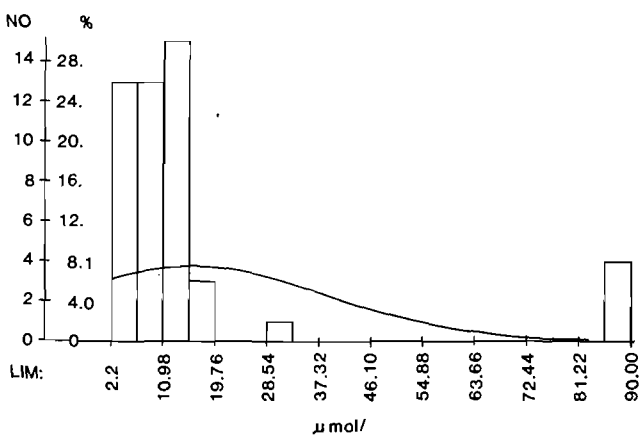

Fig. 7: The frequency distribution of iron (μmol/ℓ)
LIM: = limit NO: = number of ostriches

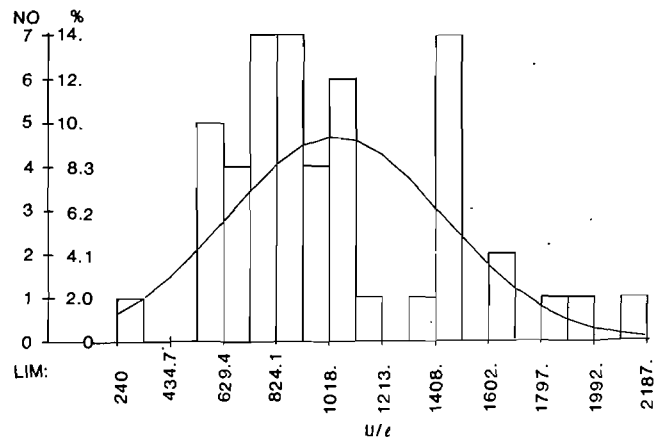

Fig. 10: The frequency distribution of lactate dehydrogenase (U/ℓ)
LIM: = limit NO: = number of ostriches

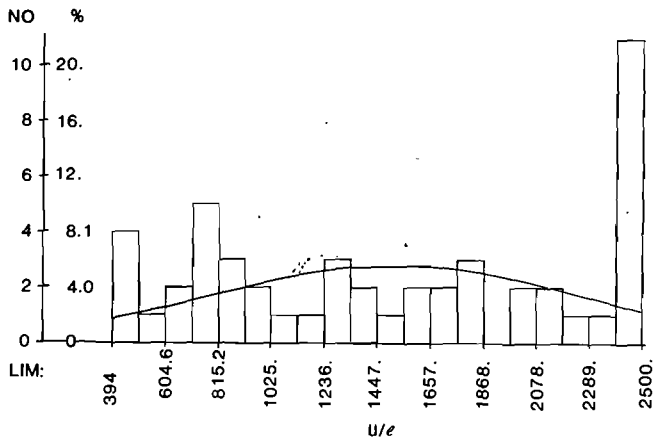

Fig. 11: The frequency distribution of creatine kinase (U/ℓ)
LIM: = limit NO: = number of ostriches

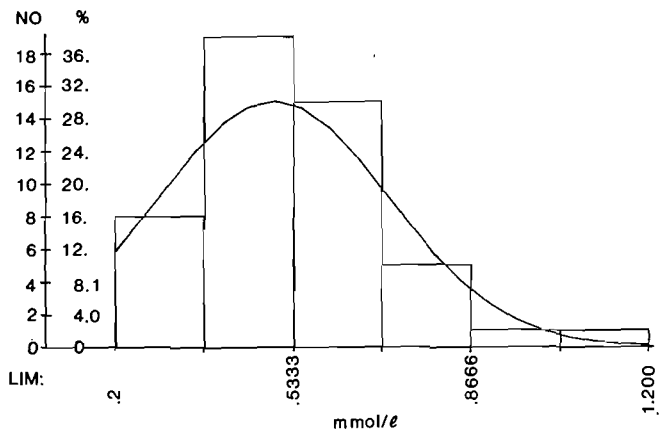

Fig. 14: The frequency distribution of urea (mmol/ℓ)
LIM: = limit NO: = number of ostriches

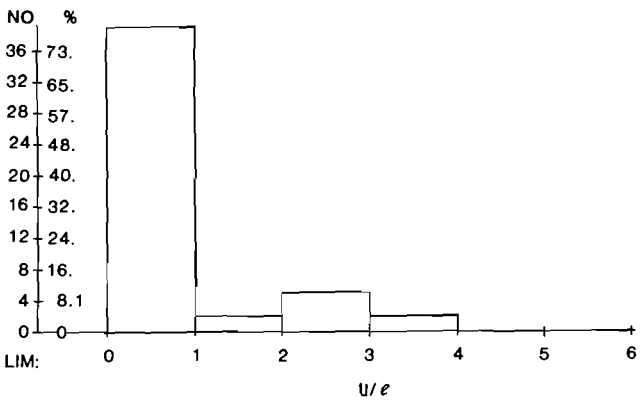

Fig. 12: The frequency distribution of γ-glutamyltransferase (U/ℓ)
LIM: = limit NO: = number of ostriches

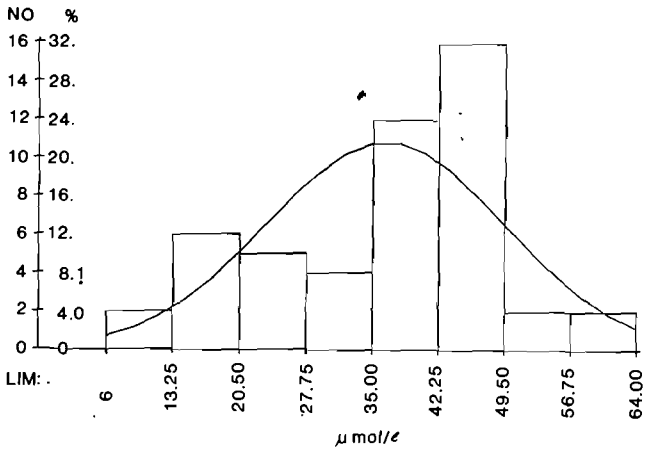

Fig. 15: The frequency distribution of creatinine (μmol/ℓ)
LIM: = limit NO: = number of ostriches

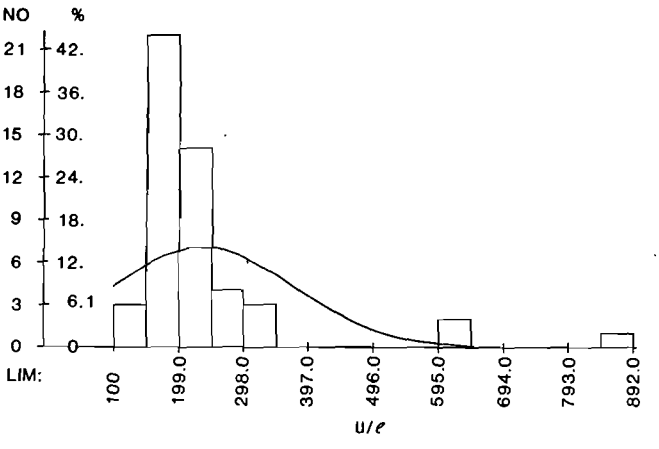

Fig. 13: The frequency distribution of aspartate transaminase (U/ℓ)
LIM: = limit NO: = number of ostriches

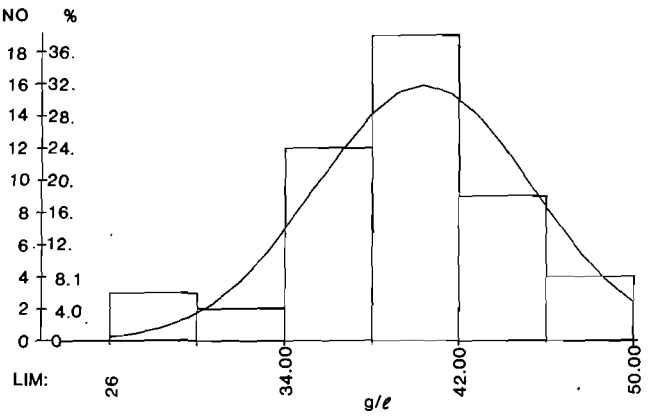

Fig. 16: The frequency distribution of total protein (g/ℓ)
LIM: = limit NO: = number of ostriches

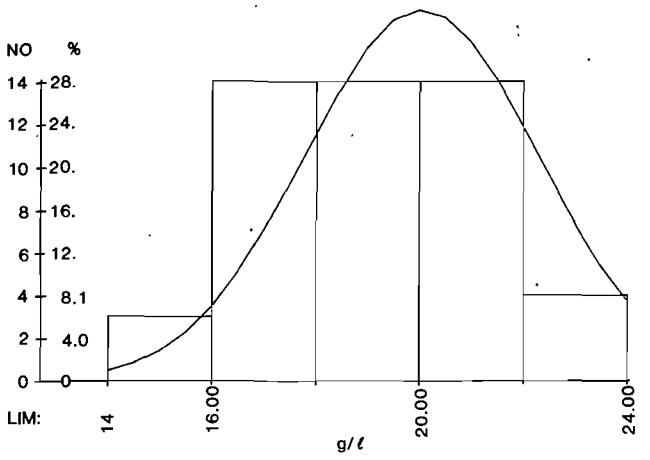

Fig. 17: The frequency distribution of albumin (g/ℓ)
LIM: = limit NO: = number of ostriches

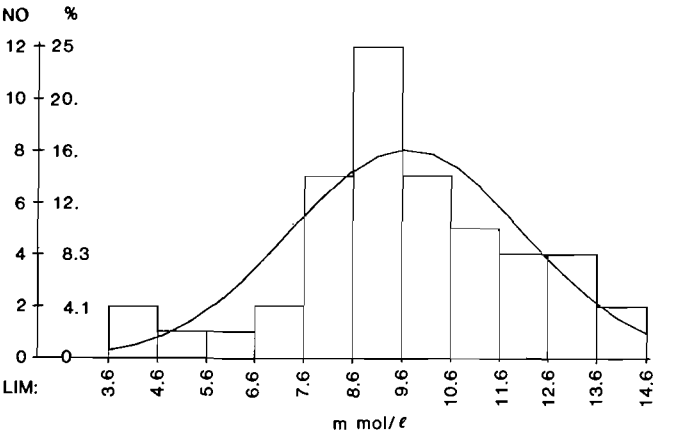

Fig. 18: The frequency distribution of glucose (mmol/ℓ)
LIM: = limit NO: = number of ostriches

the formation of glutamate and pyruvate. The resulting pyruvate is converted enzymatically in the presence of lactate dehydrogenase to lactate and NAD with the consumption of $NADH_2$ which is then measured via a decrease in absorption at 340 nm.

Lactate dehydrogenase (LD): LD reacts with pyruvate and $NADH_2$ with the formation of lactate and NAD. $NADH_2$ consumption is measured via a decrease in absorption at 340 nm.

Creatine kinase (CK): This determination is based on the following equations:

$$\text{Creatine phosphate} + \text{ADP} \xrightleftharpoons{\text{CK}} \text{creatine} + \text{ATP}$$

$$\text{ATP} + \text{Glucose} \xrightleftharpoons{\text{Hexo-kinase}} \text{ADP} + \text{Glucose-6-phosphate}$$

$$\text{Glucose-6-phosphate} + \text{NADP} \xrightleftharpoons{\text{Glucose-6-phosphate dehydrogenase}} \text{Gluconate-6-phosphate} + \text{NADPH}_2$$

The rate of increase of $NADPH_2$ is directly proportional to the CK activity in the serum.

γ-Glutamyltransferase (GGT): GGT catalyses the transfer of the amide-like bound γ-glutamyl residue onto an acceptor peptide. In the test 5-amino-2-nitrobenzoate, which absorbs at 405 nm is split off from the substrate (L-γ-glutamyl-3-carboxy-4-nitroanilide); the γ-glutamyl residue is transfered to glycylglycine. The change in absorbance per time is proportional to the rate of substrate splitting and thus to the enzyme activity.

Aspartate transaminase (AST): AST reacts in a buffered solution with α-ketoglutarate and aspartate with the formation of glutamate and oxalacetate. The oxalacetate is converted enzymatically in the presence of $NADH_2$ by malate dehydrogenase to malate and NAD and the rate of $NADH_2$ consumption is measured.

The mean ($\bar{x}$), standard error of the mean (SE), co-efficient of variation % (CV%), variance (Var) and standard deviation (SD) were determined for each of the measured parameters.

The Student's t test was applied in order to compare the values obtained between the 2 sexes.

RESULTS

The values of the different parameters analysed statistically are presented in Table 1 and Fig. 1-18.

No statistically significant differences were found between the values of the different constituents in the male and female ostriches.

In vitro haemolysis occurred in about 4% of blood specimens and in these samples K^+, inorganic phosphorus, t-Mg and Fe^{3+} concentrations were considerably higher than in the rest of the samples (Fig. 2, 4, 5 and 7).

Tremendous variation was recorded in the concentrations of the enzymes ALP, LHD, CK and AST (Fig. 8, 10, 11 & 13).

DISCUSSION

The presented values for the different blood consituents may be considered normal baseline values for the particular ostrich population. Haemolysis occurred in a number of specimens despite precautionary measures taken during specimen collection. This may have affected the levels of a number of constituents. Among these were K^+, phosporus, t-Mg and Fe^{3+}. S-Fe^{3+} levels in humans, however, are also influenced by additional factors such as stress where it has been shown that glucocorticoids and ACTH as well as adrenaline administration reduce the levels of transferrin and thereby lowering the levels of s-Fe^{3+}. Whether stress during capture of the birds played a role and thereby causing relatively low s-Fe^{3+} levels in the majority of them, is unknown.

ALP, CK and GGT activities in serum are not affected by haemolysis although the manufacturers of the Flexigem analyzer recommend the use of unhaemolyzed specimens. The elevated ALP levels found in a few individuals cannot be explained because iso-enzyme determinations were not done. Furthermore, the GGT serum activities did not aid in clarifying this finding. Higher than normal levels of ALP are seen in growing children and animals with levels returning to normal with onset of adolescence. Most of the birds in the group were between 1-2 years of age, and the possibility of raised levels or ALP due to growth should be considered.

The high CK and AST concentrations are probably due to muscular activity during the struggling and running that preceded the collection of serum specimens.

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BOOK REVIEW BOEKRESENSIE

LEONARD'S ORTHOPEDIC SURGERY OF THE DOG AND CAT

J.W. ALEXANDER

3rd Ed. W B Saunders Company, West Washington Square, Philadelphia, P A 19105. pp ix and 242, numerous figures, Price R74,79 (ISBN 0-7216-57222-2)

The object of the third edition is to update and revise the second edition of Leonard's Orthopedic Surgery of the Dog and Cat. Many of the basic principles, methods and techniques presented in the second edition have been retained. The book is not intended to be a complete text. It does, however, provide the student and practitioner with basic guidelines for the diagnosis, treatment and management of orthopaedic injuries, problems and diseases. A very useful addition is a review of basic aspects of the pathophysiology involved in the various clinical entities presented. A list of references is given at the end of each chapter.

The first chapters deal with bone as a tissue, fracture healing, osteomyelitis, preoperative and postoperative care. A chapter "Methods and Materials" describes various materials used for internal and external fixation. Indications and disadvantages of the various materials are discussed as well as application techniques. Bone grafts are discussed briefly with emphasis on types of bone grafts commonly used in clinical practice. Biomechanics of fractures and fracture classification with basic principles of their management are discussed. Fractures of the pelvic and pectoral limbs, pelvis, mandible and maxilla are presented on the basis of incidence, aetiology, pathology, signs, diagnosis, prognosis and treatment. Signs and management of malunions, delayed unions, non-unions and growth deformities are concisely presented. Subsequent chapters discuss amputations, tendon repair and basic principles of luxation management. Luxation of specific joints is also discussed with a seperate section on arthrodesis as a management technique. The last chapters in the book deal seperately with osteochondritis dissecans, elbow dysplasia, hip dysplasia, Legg-Calvé-Perthes disease, panosteitis, cranio-mandibular osteopathy, hypertrophic osteodystrophy, multiple cartilaginous exostosis, hypertrophic osteoarthropathy and arthritis.

Most of the chapters are well written and presented in a concise manner. Photographic reproductions are used extensively throughout the book but unfortunately their quality is sometimes poor and detail is not readily discernible. While this is not meant as a complete text on small animal orthopedic surgery, it is definitely a usefull asset to student and practitioner alike.

R.D. Gottschalk

BOOK REVIEW BOEKRESENSIE

PET LOSS AND HUMAN BEREAVEMENT

W.J. KAY, H.A. NIEBURG, A.H. KUTSCHER, R.M. GREY and C.E. FUDIN

1st Edn. The Iowa State University Press, Ames, Iowa 50010. 1984 pp xi and 198. Price $19,50 (ISBN 0-8138-1326-3)

Hierdie boek beskryf die omstandighede tydens die verlies van 'n troeteldier asook die rol van die persone wat betrokke is by so 'n ervaring. Twee-en-twintig outeurs van verskeie dissiplines lewer kort bydraes, elk met eie onderwerp en hoofstuk. Die hoofstukke word in die volgende drie afdelings gegroepeer: The Human/Companion Animal Bond, The Grieving Human Companion en Veterinary Medicine Perspectives.

Die boek laat die kollig val op die mens agter die troeteldier en dan spesifiek op die emosionele belewenis van die mens tydens sy troeteldier se afsterwe. Uit hierdie publikasie is dit weereens duidelik dat die troeteldier nie totaal los van sy eienaar hanteer kan word nie. Veral by aktiewe genadedood speel die veearts 'n belangrike rol en beide pasiënt en kliënt behoort in ag geneem te word.

Die groot aantal outeurs gee aan die eenkant 'n breë spektrum van sieninge oor die onderwerp, maar aan die anderkant versteur die fragmentasie 'n deurlopende gedagtelyn. Duplisering van idees kom ook voor.

Behalwe vir hierdie enkele tegniese kritiek is die boek sterk aan te bevele vir elke veearts wie se praktyk huisdiere hanteer.

Die volgende aanhaling uit die Voorwoord som die waarde van die boek vir die veearts as volg op: "With the ability to offer wise and informed counsel to bereaved clients, veterinarians will expand their professional role and enhance their image as caregivers".

J.S.J. Odendaal

DIFFERENT BANDING TECHNIQUES FOR THE STUDY OF BOVINE CHROMOSOMES

U. MÄRKI and D.R. OSTERHOFF*

ABSTRACT: Märki U.; Osterhoff D.R. **Different banding techniques for the study of bovine chromosomes.** *Journal of the South African Veterinary Association* (1985) **56** No. 2, 81-83 (En). Department of Zootechnology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

The different banding techniques R-, Q-, C-, NOR- and G-banding allow the identification of individual chromosomes in cattle with greater accuracy than with undifferentiated Giemsa staining. These techniques show characteristic bands on each chromosome and are therefore very useful and reliable for the detection of chromosomal abnormalities.

Key words: Chromosome, banding, bovine.

INTRODUCTION

A species is characterized by a specific chromosome complement, the karyotype, which is defined by the basic number, form and size of the chromosomes usually identified in mitotic metaphase. The species specificity of the karyotype makes chromosome studies useful in problems related to phylogeny and evolution. Chromosome studies are also useful in the detection of morphological changes leading to functional disorders. For this purpose cytogeneticists apply undifferentiated Giemsa staining or differential banding techniques.

In the species *Bos taurus* and *Bos indicus* all autosomes are acrocentric and very similar in form and size. Here, the different banding techniques are of special importance and their advantages and disadvantages are described.

BANDING METHODS

Bovine autosomes, in particular those of late metaphase stage, cannot be classified sufficiently by using conventional staining techniques. The limitation of this technique or even autoradiography in the identification of bovine somatic cell chromosomes has often resulted in changes in structural re-arrangements remaining unnoticed. Consequently, there was a serious need for better methods for the identification of individual chromosomes with greater accuracy.

Recently developed technical procedures, the C-, Q-, G-, R- and NOR-banding techniques, produce characteristic bands on each chromosome which are very useful and reliable for the identification of chromosomes. These banding techniques are especially successful in prometaphase when the chromosome is greatly extended. With knowledge and experience of the banding techniques, the individual chromosomes can be characterised by well-defined differential patterns which are helpful and efficient in accurate identification of the chromosomes. The G-, C-, Q-, R- and NOR-banding results are depicted in Fig. 1.

The banding techniques vary a great deal from laboratory to laboratory and are being improved continuously. They are most efficient when the metaphases are well spread, the chromosomes very long and not overlapping.

In 1971 Seabright[7] developed the rapid G-banding technique involving a trypsin digestion procedure (Fig. 2).

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The quinacrine fluorescence banding technique, or Q-banding, produces a preferential uptake of certain DNA-banding fluorescent substances which induces a constant and reproducible pattern in different chromosomes[2]. Under the ultraviolet microscope the chromosomes treated with quinacrine represent patterns of varying degrees, characteristic for each chromosome. The pale staining regions are caused by a decreased binding of quinacrine, predominantly due to non-histone proteins. The Q-bands are equivalent to the G-bands and this technique is not recommended for cattle chromosomes because of low resolution[5].

The R-bands are referred to as bands in reverse relation in expression to the G-bands. The excellent method demonstrating the R-bands was developed by a French research team led by Dutrillaux[4]. The R-bands are usually produced through the activation by fluorochrome acridine orange. Especially the bovine R-bands can be reliably described in great detail; they offer a better resolution than Q-bands[3]. This technique is strongly recommended for routine analysis of cattle chromosomes because the misclassification of chromosomes involved in structural abnormalities can almost entirely be avoided.

The centromeric heterochromatic chromosomes can be demonstrated with consistency using a special staining procedure with Giemsa, called the C-banding[1]. The C-bands contain a large number of repeated DNA sequences. A detailed analysis of the heterochromatin pattern is useful as an efficient criterium for identifying chromosomes which are morphologically difficult to differentiate. C-banding is the most useful as sequential banding following the fluorescence bandings Q and R because the acrocentric autosomes in cattle can be identified more easily, more accurately and more efficiently.

The nucleolus-organizing regions of chromosomes can be demonstrated by the NOR-banding method, which combines a silver treatment with Giemsa staining[6]. The NOR-bands represent certain structural non-histone proteins specially linked to nucleolar organizing regions and are especially useful in bovine chromosome analysis.

Bovine chromosome studies

For accurate chromosome analysis it is essential to use the proper banding method. Translocations (or any chromosomal change) can only be sufficiently diagnosed with the application of banding techniques. Giemsa stained chromosomes are only useful for counting the chromosomes and for recognizing any superficial chromosomal changes. An accurate identification of chromosomal disorder can only be performed by using

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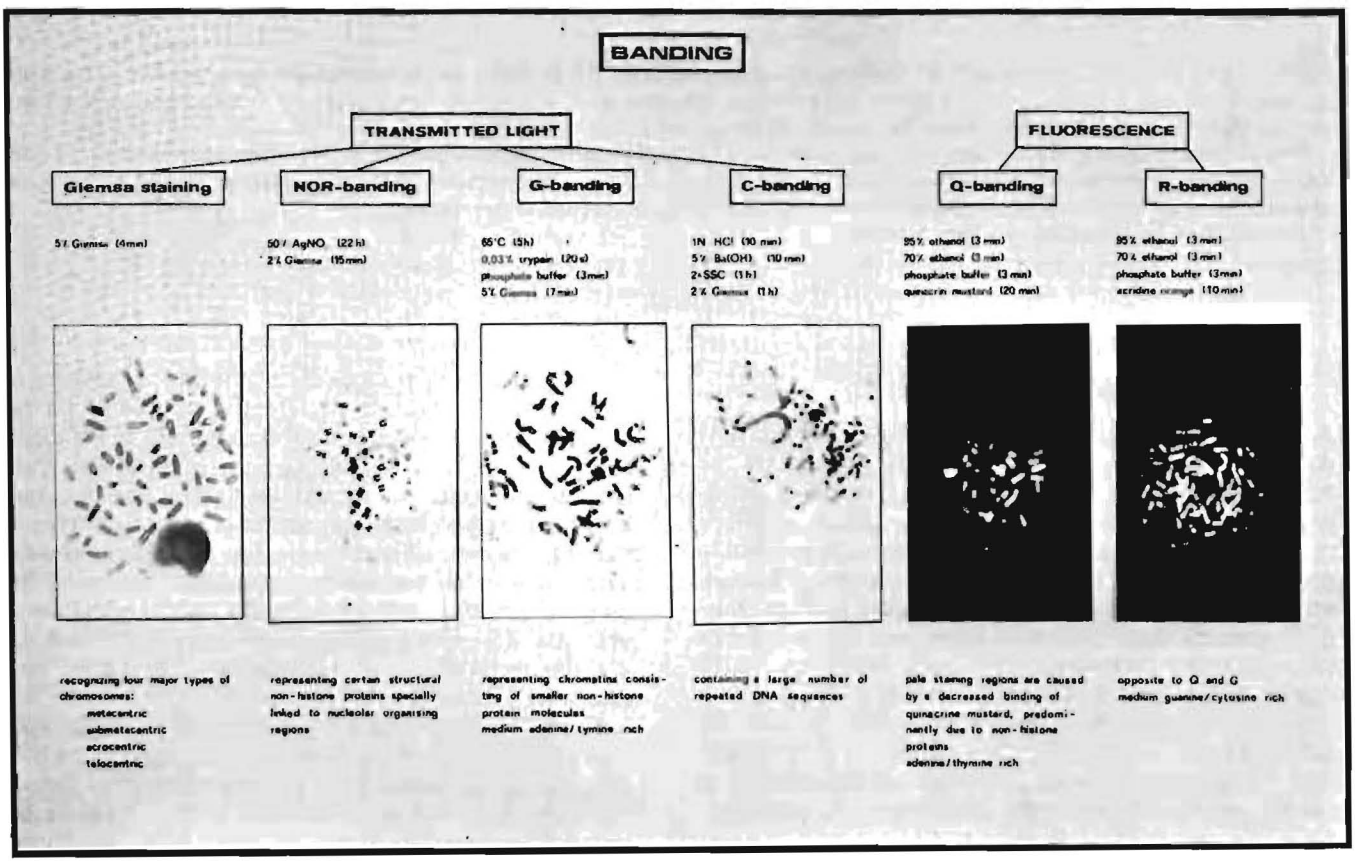

Fig 1: Giemsa staining and differential banding patterns of the bovine chromosomes: NOR-, G-, C-, Q- and R-banding techniques.

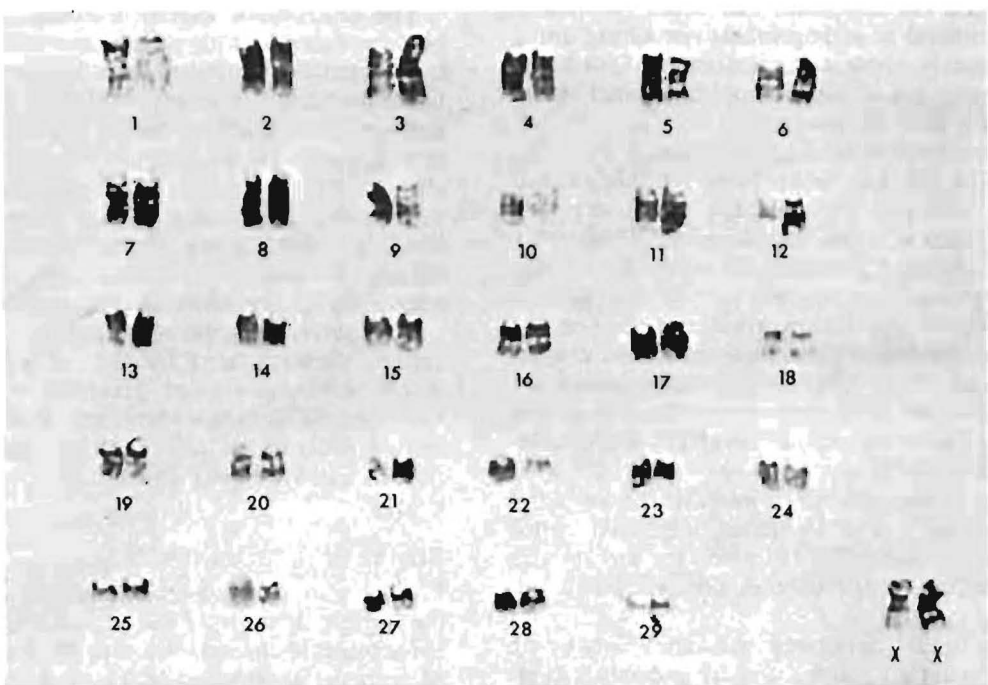

Fig 2: Representative G-banded karyotype of a Holstein/Friesian cow (2n = 60, XX).

banding methods. Whether G- or R- and Q-banding methods are used, is not really relevant. More important is the fact that cytogeneticists use a method which represents the most distinctive and clear bands as well as the greatest number of bands. Should insufficient representative bands be present, the use of a second or even a third method, to avoid a wrong diagnosis, is recommended. In our cytogenetics laboratory the Giemsa stained chromosomes are counted and a preliminary search for disorders is performed. If a chromosomal change is encountered some slides are treated with acridine orange (R) and some with trypsin (G). The detailed description of R-banded bovine chromosome[3] is of particularly great value in the identification of the R-banded chromosomes. The only disadvantage of R-banding is that not all chromosomes are activated at the same time. For this reason photographs of the same metaphase have to be taken after various time intervals. If it is difficult to recognize the centromere of the chromosomes in the R-banded chromosomes, as depicted in Fig. 1, an additional technique, the C-banding method is recommended.

The R- and G-banding methods in cattle are faster, more reliable and more accurate than the Q-banding procedures[3]. However, a great deal of experience is needed to ensure good and useful photographs of fluorescence metaphases.

CONCLUSION

Before any banding procedures can succeed, the cell culture must be timed and treated in such a way that most cells are blocked in the early stages of metaphase when chromosomes are extended, a condition which is optimal for representing a great number of bands. The different banding techniques will show up changes in the chromosomes and will avoid incorrect identifications. The undifferentiated Giemsa staining method should not be used for the analysis of morphological and structural disorders. Although the other banding techniques still have to be improved and possibly changed, the results are already satisfying and accurate for identification and study of the morphology of chromosomes. The best detailed description of bovine chromosomes is obtained by using the R-banding method, which is fast, reliable and accurate, and avoids misclassification of chromosomes involved in morphological and structural abnormalities.

ACKNOWLEDGEMENTS

The authors wish to express their appreciation to Dr. T. Robinson, Dept. of Zoology, University of Pretoria for his advice in preparing the paper. Gratefully acknowledged also are the contributions of H.H.C. Pretorius for technical assistance and L. de Kock for typing the manuscript.

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ABSTRACTS

ABSTRACT: Anderson, L.A.P., Joubert, J.P.J., Prozesky, L., Kellerman, T.S., Schultz, R., Anitra, Procos J. & Olivier, Philippa M., 1983. **The experimental production of krimpsiekte in sheep with *Tylecodon grandiflorus* (Burm. f.) Toelken and some of its bufadienolides.** *Onderstepoort Journal of Veterinary Research*, **50**, 301-307 (1983).

Six bufadienolides were isolated from *Tylecodon grandiflorus* (Burm. f.) Toelken. The paretic syndrome, krimpsiekte, could be induced in sheep either by repeated oral administration of small quantities of plant material or the intravenous injection of small quantities of certain bufadienolides. A mild to moderate, acute to subacute, multifocal cardiomyopathy was evident in sheep poisoned by both the plant and the bufadienolides.

ABSTRACT: Anderson, L.A.P., Schultz, R. Anitra, Joubert, J.P.J., Prozesky, L., Kellerman, T.S., Erasmus, G.L. & Procos, J., 1983. **Krimpsiekte and acute cardiac glycoside poisoning in sheep caused by bufadienolides from the plant *Kalanchoe lanceolata* Forsk.** *Onderstepoort Journal of Veterinary Research*, **50**, 295-300 (1983).

Three toxic bufadienolides, one characterized as hellibrigenin 3-acetate, have been isolated from *Kalanchoe lanceolata* Forsk.

Typical signs of cardiac glycoside poisoning, involving the gastro-intestinal, neuromuscular and cardiovascular systems, could be induced by drenching the milled plant to sheep. Such signs could also be induced by dosing the bufadienolides to sheep or by injecting them into both guinea-pigs (subcutaneously) and sheep (intravenously).

The specific paretic syndrome, krimpsiekte, on the other hand, was reproduced only by the repeated intravenous administration of smaller doses of the 2 unknown bufadienolides to sheep.

Histopathological examination revealed a mild to severe multifocal cardiomyopathy in sheep receiving plant material or bufadienolides.

ABSTRACT: Erasmus, J.A., 1983. **The application of numerical taxonomy in the classification of staphylococci from bovine milk.** *Onderstepoort Journal of Veterinary Research*, **50**, 291-293 (1983).

One-hundred-and-two isolates of staphylococci from bovine milk were each subjected to a battery of 19 different tests. With the application of numerical taxonomy these isolates could be classified into 1 genus and 3 different species. Although the majority of the coagulase negative organisms were grouped as 1 species, the biochemical differences within this group indicated that they should belong to at least 2 species. About 50% of these isolates could be designated *Staphylococcus epidermidis*. Possibly because of the small number of tests, a finer division into different species could not be made.

The coagulase-positive organisms could be divided into 2 species, the smaller group of which consisted of 3 isolates only. If used as the only method for identification, the coagulase test produces false positive results at a rate of about 2,5 % of cases and false negative results at a rate of about 1,7 %.

ABSTRACT: Scialdo-Krecek, Rosina C., Reinecke, R.K. & Biggs, H.C., 1983. **Studies on the parasites of zebras. III. Nematodes of the mountain zebra from the farm "Kelpie" and the Namib-Naukluft Park, South West Africa/Namibia.** *Onderstepoort Journal of Veterinary Research*, **50**, 283-290 (1983).

Twelve mountain zebra which were culled at monthly intervals on the farm "Kelpie" in South West Africa/Namibia were examined for helminths. The zebras varied in age from 1-15 years, the middle group of which, aged 4-7 years, had the highest worm burdens. Fourteen species of nematodes belonging to the families Atractidae, Strongylidae, Oxyuridae, Setariidae and Spiruridae were recovered. The highest worm burdens were those of *Crossocephalus* sp. with 692-61 066 680 and *Probstmayria vivipara* with 1 257 810-42 004 300. The predominance of the atractids is discussed. The nematodes consistently present were: *Cylicodontophorus* sp. and *P. vivipara*. Two new species, *Cylicostephanus longiconus* and *Cylicodontophorus* n. sp., were reported.

An additional 3 mountain zebra, culled in the Namib-Naukluft Park, were also examined for helminths. Of 3 zebras ranging in age from 2-7 years, the 2 older animals had the highest helminth burden. Ten species of the nematodes belonging to the same families mentioned above were recovered. The only Spiruridae present were 3 *Habronema majus* in 1 zebra. The highest worm burdens were those of *Crossocephalus* sp. with 64 052-883 070 and *P. vivipara* with 50 720-220 200. The nematodes consistently present were the same as those in the "Kelpie" zebra. In addition, a 2nd, new species of *Cylicodontophorus* was reported.

ABSTRACT: Jansen, B.C., 1983. **The epidemiology of bacterial infection of the genitalia in rams.** *Onderstepoort Journal of Veterinary Research*, **50**, 275-282 (1983).

The interrelationship between the various bacteria isolated from the genital tract of rams and their host animals was studied. The pathogenicity of the different isolates varied. Several of these bacteria could be cultured in a medium consisting of a suspension of pen floor debris solidified with agar, while many organisms survived in the suspension for 10 days. Epidemiological investigations showed that rams kept under intensive systems were subjected to large-scale invasion of their genitalia by bacteria which led to infection of the accessory glands and orchitis and epididymitis. Apart from the preputial cavity, some rams kept on open range were entirely free of bacterial infection of their genitalia, and those that did have bacteria in the deeper parts of their genitalia had a very significantly lower incidence of pathological lesions of their genitalia. Finding bacteria and neutrophils in semen is consistent with the epidemiological findings.

ABSTRACT: Jansen, B.C. & Hayes, Marianna, 1983. **Retardation of wool growth in Merino sheep caused by bacteria.** *Onderstepoort Journal of Veterinary Research*, **50**, 271-274 (1983).

A condition evidenced by retarded growth of wool with alteration of the yolk into a yellow, sticky, wax-like substance was investigated. The condition was associated with hyperaemia and cellular infiltration into the dermis in the affected areas. Three bacterial species, viz. *Enterobacter aerogenes, E. agglomerans* and *Hafnia alvei*, which could grow on the water-extractable component of wool-yolk, were incriminated as the cause of the condition.

VOORKOMS VAN KLINIESE GEVALLE IN 'N VOORSTEDELIKE PLAASDIER-PRAKTYK

G.H. RAUTENBACH*

ABSTRACT: Rautenbach G.H. **The incidence of clinical cases in a peri-urban farm animal practice.** *Journal of the South African Veterinary Association* (1985) **56** No. 2, 85-88 (Afrik). Department of Applied Veterinary Practice, Faculty of Veterinary Science, Medical University of Southern Africa, P.O. Box 170, 0204 Medunsa, Republic of South Africa.

The incidence of clinical cases in farm animals encountered in the ambulatory practice at the Faculty of Veterinary Science, University of Pretoria, is discussed. In a 3 year period 4 013 clinical cases were seen; these did not include cases seen during herd examinations. The diagnoses made are grouped under infectious diseases (19,6 %), nonspecific inflammatory conditions (17,3 %), gastro-intestinal conditions (9,7 %), gynaecological conditions and procedures (14,5 %), surgical conditions and procedures (26 %), toxicological conditions (3,7 %), metabolic diseases (1,5 %), nutritional abnormalities (2,4 %) and miscellaneous conditions.

Key words: Clinical incidence, farm animals, peri-urban practice.

INLEIDING

Literatuur wat handel oor die insidens van kliniese gevalle is 'n belangrike bron van inligting vir die epidemioloog. Tog is daar met enkele uitsonderings[1,3-5,9,15] maar min gepubliseer hieroor in die Suid-Afrikaanse veeartsenykundige tydskrifte. Daar is wel voorbeelde van siekte-insidensstudies deur opvolging van nadoodse ondersoeke[8], abattoir resultate[13], laboratoriumverslae[16-18] sensus en veldobservasiestudies, of 'n kombinasie van voorafgaande[2,6,10-12,14].

Die voorkoms van kliniese gevalle waargeneem deur die praktiserende dierearts mag drasties verskil van bogenoemde gegewens, veral in toestande met 'n lae morbiditeit en mortaliteit. Hierdie skynbare gebrek aan basiese epidemiologiese gegewens bemoeilik doelgerigte navorsing en opleidingsbeplanning.

PRAKTYK EN METODES

'n Opname is gemaak oor die voorkoms van gevalle in die ambulatoriese praktyk van die Fakulteit Veeartsenykunde, Universiteit van Pretoria. Die praktyk bedien hoofsaaklik voorstedelike kleinhoewes wat in 'n omtrek van ongeveer 25 km om Onderstepoort geleë is. Die opname is gemaak vanaf 1979-1982 en sluit slegs enkelpasientgevalle in; gevalle wat gesien is tydens kuddebesoeke is uitgesluit. Diagnoses is gemaak deur kliniese ondersoeke, in sommige gevalle gerugsteun deur kliniespatologiese bepalings, nadoodse ondersoeke en ander diagnostiese hulpmiddels wat binne 24 uur afgehandel kon word. Resultate van diagnostiese toetse wat nie binne 'n redelike tyd beskikbaar gestel is nie kon nie in alle gevalle gekorreleer word met die kliniese diagnoses nie.

Elke geval is slegs by een afdeling ingedeel en waar die etiologie onbekend was, is 'n patologies-anatomiese diagnose gemaak, soos byvoorbeeld pneumonie in teenstelling met pasteurellose. Dit moet dus duidelik wees dat sieketoestande wat maklik uitgeken word meer gediagnoseer word en sodoende 'n invloed het op die waarneembare voorkoms van die siekte.

Een van die funksies van hierdie praktyk is die opsporing van kliniese materiaal vir die fakulteit se opleidingshospitaal en dus is meeste van die moeiliker gevalle vir opname verwys. Hierdie gevalle maak dus ook nie deel van hierdie verslag uit nie.

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Die gevalle is in die volgende afdelings ingedeel:

Infeksiesiekte – bakteriese siektes
– virussiektes
– protosoiese siektes
– Chlamydia/Rickettsiële siektes

Nie-spesifieke inflammatoriese toestande
Primêre spysverteringskanaal toestande
Geslagskundige toestande en prosedures
Chirurgiese toestande en prosedures
Vergiftigings
Metaboliese toestande
Voedingsabnormaliteite
Ander toestande

RESULTATE

Vierduisend-en-dertien kliniese gevalle bestaande uit 3 040 beeste, 321 perde, 112 varke, 509 skape en 31 bokke is ondersoek en/of behandel vanaf 1979-1982.

Die aantal diagnoses in elke afdeling word in Tabel 1 weergee.

Tabel 1: SIEKTE TOESTANDE GEDIAGNOSEER EN PROSEDURES UITGEVOER OP 4 013 KLINIESE GEVALLE GEDURENDE DIE TYDPERK 1979-1982

	Aantal gevalle	%
Infeksiesiektes:		
Bakteriese siektes	280	7,0
Virussiektes	131	3,3
Protosoiese siektes	137	3,4
Chlamydia/Rickettsiële siektes	236	5,9
Nie-spesifieke inflammatoriese toestande	695	17,3
Primêre toestande van die spysverteringskanaal	390	9,7
Geslagskundige toestande en prosedures	581	14,5
Chirurgiese toestande en prosedures	1070	26,0
Vergiftigings	148	3,7
Metaboliese toestande	62	1,5
Voedingsabnormaliteite	98	2,4
Ander	185	4,6

In 784 gevalle kon die oorsaak van die kliniese tekens aan 'n spesifieke infeksieuse oorsaak toegeskryf word (Tabel 2). Daar is 29 verskillende infeksiesiektes geïdentifiseer waarvan dié met die hoogste insidens in Tabel 3 weergee word.

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Seshonderd-vyf-en-negentig (17,3 %) nie-spesifieke inflammatoriese toestande is gediagnoseer waar die oorsaak nie klinies of klinies-patologies geïdentifiseer kon word nie (Tabel 4). As die gevalle van diarree en metritis of piometra wat onder ander afdelings ingedeel is hier bygetel word, vermeerder die getal na 987 (24,6 %).

Primêre spysverteringsprobleme is gediagnoseer in 390 gevalle (9,7 %). In Tabel 5 word 'n uiteensetting gegee van die belangrikste toestande in hierdie groep.

Daar is 581 (14,5 %) geslagskundige toestande en prosedures gehanteer (Tabel 6). Dragtigheidsondersoeke, enkel of op 'n kuddebasis, is nie hierby ingesluit nie.

Tabel 2: INFEKSIESIEKTES GEDIAGNOSEER: TOTAAL 784

	Aantal gevalle	%
Bakteriese siektes	280	35,7
Virussiektes	131	16,7
Protosoïese siektes	137	17,5
Chlamydia/Rickettsiële siektes	236	30,1

Daar is 1070 (26 %) chirurgiese gevalle gesien en prosedures uitgevoer (Tabel 7).

Tabel 3: MEES ALGEMENE INFEKSIESIEKTES GEDIAGNOSEER: TOTAAL 784

	Bees	Perd	Skaap en Bok	Vark	Totaal	%
Vrotpootjie-kompleks	229	—	—	—	229	29,2
Anaplasmose	121	—	—	—	121	15,4
Hartwater	98	—	17	—	115	14,7
Babesiose	87	9	—	—	96	12,2
Perdesiekte	—	42	—	—	42	5,4
Drie-dae-stywesiekte	39	—	—	—	39	5,0
Koksidiose	17	—	8	—	25	3,2
Aansteeklike bees rinotrageïtis	20	—	—	—	20	2,5
Ander	73	2	16	6	97	12,4

Tabel 4: NIE-SPESIFIEKE INFLAMMATORIESE TOESTANDE: TOTAAL 695

	Bees	Perd	Skaap en Bok	Vark	Totaal	%
Mastitis	211	2	27	6	246	35,4
Keratitis/oftalmie	133	8	20	—	161	23,2
Absesse	82	16	25	7	130	18,7
Respiratoriese infeksies	58	6	7	—	71	10,2
Sellulitis	6	5	2	—	13	1,9
Artritis/poliartritis	8	3	—	2	13	1,9
Ander	38	9	11	3	61	8,8

Tabel 5: PRIMÊRE SPYSVERTERINGSKANAALPROBLEME: TOTAAL 390

	Bees	Perd	Skaap en Bok	Vark	Totaal	%
Diarree	84	7	21	5	117	30
Rumenasidose	74	—	19	—	93	23,8
Rumenstase	53	—	7	—	60	15,9
Traumatiese retikulitis	48	—	—	—	48	12,3
Koliek	—	30	—	—	30	7,7
Timpanie	23	—	—	—	23	5,9
Ander	9	3	7	—	19	4,9

Tabel 6: GESLAGSKUNDIGE TOESTANDE EN PROSEDURES: TOTAAL 581

	Bees	Perd	Skaap en Bok	Vark	Totaal	%
Metritis/piometra	167	3	5	—	175	30,1
Plasentale retensie	118	—	—	—	118	20,3
Distokie	106	—	9	2	117	20,2
Oestrus induksie	30	—	—	—	30	5,1
Aborsies	22	—	1	1	24	4,1
Vaginale prolaps	17	—	—	—	17	2,9
Keisersnee	15	—	1	—	16	2,7
Anoestrus	15	—	—	—	15	2,6
Ander	50	4	14	1	69	11,9

Tabel 7: CHIRURGIESE PROSEDURES EN GEVALLE: TOTAAL 1 070

	Bees	Perd	Skaap en Bok	Vark	Totaal	%
Orgidektomie	111	35	100	63	309	28,9
Onthoringing	275	—	11	—	286	26,7
Trauma	49	33	36	2	120	11,2
Absesse dreineer	59	6	26	2	93	8,7
Wonde heg	40	23	25	1	89	8,3
Klou/hoef-versorging	16	9	6	—	31	2,9
Rumenotomie/laparotomie	28	—	2	—	30	2,8
Speenoperasies	25	—	—	—	25	2,3
Ander	50	24	13	—	87	8,1

Honderd-agt-en-veertig individuele gevalle van vergiftiging is gediagnoseer en behandel. Van die organiese gifstowwe was organofosfate vir die meeste vergiftigings verantwoordelik terwyl *Lantana camara* vergiftiging die mees algemene plantvergiftiging was.

Daar is 'n totaal van 62 gevalle met 'n metaboliese siekte gediagnoseer bestaande uit 29 "downer" koeie, 21 melkkoorsgevalle en 11 gevalle met ketose. Slegs een van die ketose gevalle is as primêre ketose gediagnoseer, terwyl die ander sekondêre ketose gehad het met ander kompliserende faktore.

Agt-en-negentig gevalle met primêre voedingsabnormaliteite is geïdentifiseer waarvan die meeste verhongering was as gevolg van onkunde van die eienaars. Veertien gevalle van afosforose is gediagnoseer.

Honderd-vyf-en-tagtig gevalle het nie onder die voorafgaande afdelings ingepas nie. Parasitiese toestande (152) vorm die vernaamste onderafdeling in hierdie groep. Veertien gevalle van sweetsiekte is geïdentifiseer oor die 3 jaar periode.

BESPREKING

Die infeksiesiekte met die hoogste voorkoms was die vrotpootjie-kompleks, gevolg deur anaplasmose, hartwater en babesiose. Die vrotpootjie gevalle was meestal afkomstig van groter intensiewe melkkuddes en is 'n byna onbekende siekte onder diere op kleinhoewes. Die 20 gevalle van aansteeklike bees rinotrageitis is almal gedurende een uitbraak geïdentifiseer. Geen gevalle van besnoitiose in beeste is gediagnoseer nie, maar een geval is in 'n perd gesien. Vyf gevalle van serebrale theileriose is deur nadoodse ondersoeke bevestig en 'n verdere 9 gevalle was klinies tentatief as serebrale theileriose gediagnoseer maar nie bevestig nie. Drie van die gevalle was afkomstig van Haakdoringboom, 'n plaas waar gevalle voorheen aangeteken is[7]. Slegs 'n enkele geval van serebrale babesiose is in 'n bees gedurende die 3 jaar gediagnoseer.

Wat die nie-spesifieke inflammatoriese toestande betref, was mastitis en keratitis belangrik, selfs in die kleiner kuddes en in enkeldiere wat op kleinhoewes aangehou word. Positiewe identifisering van die oorsaak in hierdie toestande is dikwels bemoeilik deur die voorafbehandeling met 'n antibiotiese middel.

Die besondere hoë voorkoms van kliniese rumenasidose kan waarskynlik toegeskryf word aan onkunde van die eienaars. Vagusindigestie is in 7 gevalle gediagnoseer terwyl 3 gevalle met 'n verplaasde abomasum gesien is.

Van die 133 distokie gevalle is 16 (23,3 %) deur middel van 'n keisersnee verlos. Manlike geslagsprobleme het min voorgekom, waarskynlik as gevolg van die feit dat min eienaars 'n bul aanhou en eerder van kunsmatige inseminasie of toevallige dekkings deur beskikbare manlike diere gebruik maak.

Die siekte-insidens in hierdie praktyk word grootliks deur bosluisbesmetting beïnvloed. Daar is 374 (9,3 %) bosluis-oordraagbare siektes geidentifiseer. Dit sluit die verskillende toestande in wat veroorsaak word deur die *Babesia-*, *Theileria-*, *Anaplasma-* en *Rickettsia-*organismes en ook die bosluistoksikoses soos sweetsiekte. Verder speel bosluisbesmetting 'n groot maar moeilik bepaalbare rol in die voorkoms van absesse, speenmisvormings, otitis eksterna, epidurale absesse en prolaps van die voorhuid. Selfs baie van die mastitis gevalle begin waarskynlik primêr as gevolg van besmette bosluisbyte aan die spene en uier.

SAMEVATTING

Die veearts in die voorstedelike praktyk sien waarskynlik gevalle wat sy kollegas in die platteland min bereik. As gevolg van die onkunde van baie van die voorstedelike kleinboere is die spektrum van gevalle en probleme wat gesien word wyd. Voorkomende geneeskunde en gesonde bestuurspraktyke is, soos vir die groter boer ook belangrik vir die kleinhoewe eienaar. Die voorstedelike area het eiesoortige veeartsenykundige probleme en dit word voorsien dat dit 'n belangrike werksomgewing sal word vir die stedelike praktisyn wat belangstel in plaasdiere.

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NEUROCHEMICAL CHANGES IN THE BRAIN AND SPINAL CORD OF SHEEP: A BASIS FOR THE IMMOBILIZING ACTION OF ETORPHINE

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ABSTRACT: Kania B.F. **Neurochemical changes in the brain and spinal cord of sheep: a basis for the immobilizing action of etorphine.** *Journal of the South African Veterinary Association* (1985) **56** No. 2, 89-92 (En). Department of Pharmacology and Toxicology, Veterinary Faculty, University of Lubumbashi, Republic of Zaire.

Clinical and pharmacological analyses showed a full immobilizing action of etorphine in adult sheep ± 7 min. after a deep i.m. injection of the drug at a dosage rate of 20 μg/kg. Neurochemical analyses of motoric CNS-structures done within 30 min. after i.m. injection of etorphine in an immobilizing dose, showed the following:

(a) A significant decrease of dopamine (DA) and homovanilic acid (HVA) concentrations in the corpus striatum, the frontal motor cortex, cerebellum and in the lumbo-sacral portion of the spinal cord.
(b) A decrease of DA-concentration with a simultaneous increase of HVA-concentration in the pons.
(c) A significant decrease of noradrenaline (NA) concentration in the cerebellum and lumbo-sacral portion of the spinal cord and an insignificant decrease of amine concentration in the pons.
(d) A significant increase of adrenaline (A) concentration in the frontal motor cortex.
(e) A significant decrease of 5-hydroxytryptamine (5-HT) concentration in the frontal motor cortex and cerebellum and an equally significant increase in its concentration in the pons.

Key words: Etorphine, immobilization, sheep, neurochemical changes.

INTRODUCTION

Etorphine is a narcotic analgesic exceeding the analgesic action of morphine by 1000-2000 times[6]. It has been used for 16 years alone or in neuroleptic-analgesic combinations for immobilizing free-ranging and domestic animals[11 12 23 30].

Because of the strength and rapidity of its immobilizing action, the lack of harmful side effects, a high margin of safety and the fact that infallible competitive antogonists (cyprenorphine and diprenorphine) are available, the author has used this drug in different animals with good results[15-17].

During the immobilizing action of both etorphine itself (M 99, Oripavin) as well as its neuroleptic-analgesic compounds (Immobilon), the following side effects have been observed in animals: rigidity, spastic flexion and body tremors[12 21 27].

Similar symptoms have been observed in idiopathic Parkinson's disease[8 13] parkinsonoidal postneuroleptic syndrome in man[13] and in postneuroleptic and postetorphine catalepsy in rats[18 20 28].

Following the finding of biochemical changes in Parkinson's disease in man[4], attention was paid to the role of the extrapyramidal system in the mechanism of cataleptic and immobilizing activity of etorphine in rats. It was observed that etorphine, similarly to morphine and fentanyl[9 28 29], increases the dopamine (DA) synthesis and turnover in the central structure of the extrapyramidal system, i.e. the striatum. Similarly changes also occur in cases where neuroleptic drugs are administered in cataleptic doses.

The difference between postetorphine and postneuroleptic catalepsy according to Sharman[28], lies in the fact that the newly synthetized DA of the striatum is diverted by the etorphine and other narcotic analgesics from the storage sites to the catabolic sites in the presynaptic endings of neurones. Neuroleptics which block the postsynaptic DA-receptors of the striatum increase the synthesis, release and metabolism of DA. This results from the activation of transsynaptic feedback mechanisms from postsynaptic to presynaptic structures due to a deficiency or total lack of amine in the postsynaptic receptors.

Until now, there has been no explanation of the central mechanism of the immobilizing action of etorphine in non-laboratory animals especially in ruminants and thus in sheep. In ruminants the use of narcotic drugs for analgesic purposes is contraindicated, thus the undertaking of such a study appeared to be of interest. It was decided to determine the dependence between the immobilizing action of etorphine and the changes in DA, homovanilic acid (HVA), noradrenalin (NA), adrenalin (A), 5-hydroxytryptamine (5-HT) and 5-hydroxyindole acetic acid (5-HIAA)-concentrations in the corpus striatum, motor frontal cortex, pons, cerebellum and lumbo-sacral portion of the spinal cord in sheep.

MATERIALS AND METHODS

The experiments were done in Warsaw on 12 adult wethers of unknown origin and body mass of 32-45 kg. The animals were divided into 2 groups of 6 animals each. The experimental group was given deep i.m. injections of etorphine hydrocloride (M 99, Reckitt & Colman, South Africa Division) at a dosage rate of 20 μg/kg. The animals were killed by decapitation 30 min. after the injection. The control group were given deep i.m. injections of etorphine solvent at the same volume dose and the animals were decapitated 30 min. after the injection.

Immediately after the decapitation, cranial trepanation was performed and the brain was removed. At the same time the lumbo-sacral part of the spinal cord was removed as well. The tissues were dried on special lignin, the meninges removed and the exposed tissue placed on dry ice. Proper dissection was then done immediately and the corpus striatum, motor frontal cortex, pons and cerebellum removed. According to the weight and size of isolated brain structures, each part was cut into 3 monomial fragments and immediately placed in a solution of absolute alcohol and dry ice. The mean interval from decapitation to the time of freezing was c. 5 min. The frozen CNS-structures were weighed, put in properly marked metal foil and placed in dry ice where they were stored until assayed.

The concentrations of DA, HVA, 5-HT and 5-HIAA

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were assayed with spectrofluorometric methods and the concentrations of catecholamines (DA, NA, A) with methods previously described[18]. Samples for determination of HVA-concentrations were prepared according to Juorio et al.[14] and were oxidized according to Anden et al.[1].

The method of Curson & Green[7] was used to detect 5-HT and 5-HIAA. Statistical analyses were done with the Student's *t* test at $p < 0,05$.

RESULTS

In sheep an optimal immobilizing dose of etorphine hydrochloride by deep i.m. injection was determined as 20 µg/kg. Full immobilization was observed in c. 7 min. after the injection. The immobilizing action lasted about 100 minutes. Before full immobilization, a motoric excitation phase, lasting approximately 30 sec., was observed.

During full postetorphine immobilization the following signs were observed: spastic flexion of hind leg muscles, rigidity of the neck and foreleg muscles and recurring body tremors.

In the neurochemical examination it was observed that etorphine in its full immobilizing action caused a statistically significant decrease in DA-concentration in the corpus striatum ($p < 0,05$) and pons (51 %) and an increase in the cerebellum (35 %), lumbo-sacral part of the spinal cord (60%) and especially in the motor frontal cortex (122 %) (Table 1).

Etorphine caused insignificant changes in NA-concentrations in the corpus striatum and motoric frontal cortex, clearly decreased its concentration in the pons (43 %) and significantly decreased the levels in the cerebellum (64 %) and lumbo-sacral part of the spinal cord (46 % at $p \leq 0,025$) (Fig. 1).

The results obtained from adrenalin determinations do not point clearly to any specific tendency since levels could not be determined in all structures nor could it be found in all control animals. It can only be said that etorphine administered in immobilizing doses decreased the A-concentrations in the corpus striatum (17 %), pons and lumbo-sacral part of the spinal cord and increased it in the cerebellum and significantly so in the motor frontal cortex only ($p \leq 0,025$) (Fig. 1).

No effect of etorphine on 5-HT-concentration in the corpus striatum was observed, however, a significant decrease of 5-HT-concentration was noted in the motor frontal cortex (48 %) and cerebellum (46 %) at $p < 0,01$. Etorphine increased 5-HT concentration in the pons (53 %, $p < 0,01$) and in the lumbo-sacral part of the spinal cord (36 %) (Fig. 1).

It should be added that an exceptionally low standard error in this group of notations resulted in the fact that with the differences in concentrations amounting to ± 15 %, the results differed significantly, while in other series of notations of biogenic amine concentrations, the results were only significant when the differences amounted to ± 45 % and more.

Etorphine caused a significant decrease of HVA-concentration in the corpus striatum in sheep (40 % $p < 0,05$) and an equally significant increase of the concentration of this acid in the motor frontal cortex (310 %, $p < 0,025$), pons (59 %), cerebellum (90 %) and lumbo-sacral part of the spinal cord (138 %) (Table 1; Fig. 1).

At the time of full immobilizing action, a decrease of 5-HIAA-concentration in the lumbo-sacral part of the spinal cord (48 %) and an increase in the concentration of this acid in the cerebellum were observed. The lack of statistical calculations for the data obtained is due to the fact that only single results were obtained in the analyses (Table 1).

DISCUSSION

A detailed analysis of the obtained results points to the fact that the main cause of the immobilizing action of etorphine in sheep is a significant decrease of DA and HVA-concentrations in the main structure of the extrapyramidal system viz. the corpus striatum. During postetorphine immobilization a deficiency or lack of DA on postsynaptic receptors of the striatum were observed. This leads to an intensified inhibitory effect of the striatum on motoric activity in animals and finally to their immobilization. The main cause of postetor-

Table 1: EFFECT OF ETORPHINE ON DOPAMINE (DA), HOMOVANILIC ACID (HVA), NOR-ADRENALINE (NA), ADRENALINE (A), SEROTONIN (5-HT) AND 5-HYDROXYINDOLE ACETIC ACID (5-HIAA) CONCENTRATIONS (µg/g – TISSUE) IN DIFFERENT CNS REGIONS OF SHEEP COMPARED TO THE CONTROL GROUP (C). ALL VALUES ARE MEAN ± SE (n = 6)

CNS regions	DA		HVA		NA		A		5-HT		5-HIAA	
	C	E	C	E	C	E	C	E	C	E	C	E
Corpus striatum	3,762 ±0,062	3,046[a] ±0,222	0,733 ±0,100	0,440[b] ±0,092	0,099 ±0,005	0,094 ±0,003	0,024 ±0,004	0,021 ±0,006	0,295 ±0,007	0,307 ±0,011	0,500	0,480
Cortex cerebral	0,188 ±0,035	0,418 ±0,097	0,346 ±0,068	1,418[b] ±0,295	0,070 ±0,021	0,076 ±0,019	0	0,021[b] ±0,006	0,332 ±0,014	0,218[c] ±0,016	0,210	0,210
Pons	0,143 ±0,038	0,070 ±0,020	0,245 ±0,086	0,391 ±0,048	0,152 ±0,041	0,080 ±0,014	0,011 ±0,005	0,009 ±0,004	0,237 ±0,009	0,363[c] ±0,024	0,610	0,710
Cerebellum	0,104 ±0,022	0,139 ±0,033	0,169 ±0,059	0,322 ±0,064	0,129 ±0,015	0,045[c] ±0,006	0,027 ±0,007	0,032 ±0,007	0,282 ±0,012	0,170[c] ±0,013	0	0,100
Spinal cord	0,148 ±0,025	0,237 ±0,045	0,307 ±0,075	0,730 ±0,423	0,060 ±0,009	0,033[a] ±0,007	0,010 ±0,004	0	0,170 ±0,008	0,232 ±0,029	0;210	0,100

Significance (Student's *t* test): a = $p \leq 0,05$, b = $p \leq 0,025$, c = $p \leq 0,01$
C = Control
E = Etorphine

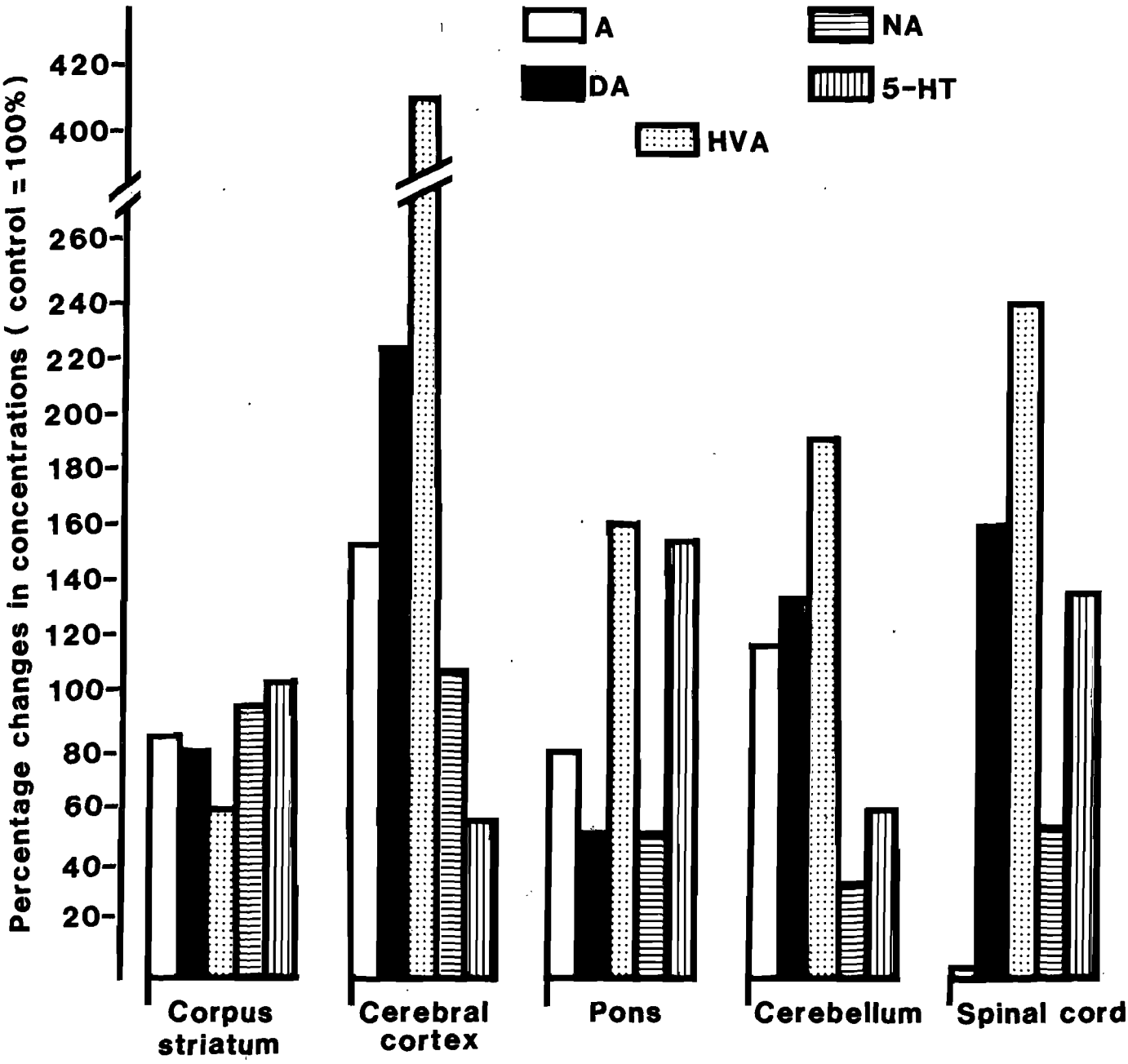

Fig. 1: The percentage changes of A, DA, HVA, NA and 5-HT concentrations after i.m. etorphine injection in sheep motor structures of the CNS.

phine inhibition of synthesis of the main mediator of the central structure of the extrapyramidal system, is a stimulating effect of the drug on presynaptic DA-receptors. According to Kebabian et al.[22] stimulation of these receptors leads to inhibition of tyrosine hydroxylase activity – an enzyme responsible for the transformation of tyrosine to dihydroxy-phenyl-alanine (DOPA) which is the direct precursor of DA.

Comparative analysis of the results of neurochemical investigations obtained during postetorphine catalepsy in rat striatum contradicts the results obtained at the time of full postetorphine immobilization in sheep. The author's earlier results[18] point to significant postetorphine increases in DA-concentration and the results of Sharman[28] to a simultaneous significant increase of HVA-concentration in rat striatum. It could be a confirmation of the existence of specific differences in reaction to narcotic analgesics as suggested in 1864 by Claude Bernard[3] in relation to morphine and later confirmed by Mayenert[26].

Summing up this part of the discussion it should be stressed that etorphine diminished the rate of DA synthesis in the corpus striatum of sheep while in rats the DA turnover in this structure is significantly increased. Similar effects are observed in relation to other drugs from the morphine group including morphine when administered in cataleptic doses[9 10 24 28].

In its full immobilizing action in sheep, etorphine increased DA and HVA-concentrations in the motor frontal cortex, cerebellum and lumbo-sacral spinal cord. These actions could be the result of the post-etorphine blocking of postsynaptic DA-receptors. However, according to Sharman[28] and later Kuschinsky & Hornykiewicz[24] and Freye & Kuschinsky[9], an increase of HVA-concentration is a result of the diversion of newly synthesized DA from the storage to the catabolic sites

and not an action similar to that of the neuroleptics. This hypothesis is confirmed by our assumption concerning the presynaptic mechanism of etorphine activity because Kebabian et al.[22] claim that the stimulation of presynaptic DA-receptors inhibits its release from the presynaptic elements of neurons. The fact that the postsynaptic DA-receptors are free at the same time, has been confirmed by the antietorphine and anticataleptic activity of apomorphine in rats[19].

Postetorphine neurochemical changes in the lumbosacral part of the spinal cord in sheep are worth noticing. At the time of full postetorphine immobilization it has been observed in this part of the spinal cord, as it was already mentioned, that DA and HVA-concentrations increased significantly and NA concentration decreased equally significantly (Fig. 1). This finding could testify to the inhibition of metabolic transformation of DA into NA and simultaneously it confirms an increased metabolism of the latter or it proves the participation of noradrenergic receptors of the spinal cord in the production of cataleptic and immobilizing effects by etorphine and other narcotic analgesics[2 5 25].

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RESEARCH NOTE **NAVORSINGSNOTA**

PROFILES OF SEROLOGICAL REACTIONS FOLLOWING ADULT COW INOCULATION WITH STANDARD DOSE *BRUCELLA ABORTUS* STRAIN 19 VACCINE

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ABSTRACT: Herr S.; Te Brugge L.A. **Profiles of serological reactions following adult cow inoculation with standard dose *Brucella abortus* strain 19 vaccine.** *Journal of the South African Veterinary Association* (1985) **56** No. 2, 93-96 (En). Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa.

Experimental use of the standard dose ($4-12\times10^{10}$ viable organisms per dose) *Brucella abortus* strain 19 vaccine (S_{19}) in adult cattle led to a serological response in the complement fixation test (CFT) within 2 weeks. The antibody titres recorded could not be distinguished from those arising from infection for a period of 6 – 11 months. This was in the absence of the booster effect of *Brucella* antigen from either field or vaccine strains. The illegal or unintentional use of S_{19} in adult cattle may be recognized by a sudden steep antibody titre response in the CFT which will decline to negative levels within 6 – 11 months provided no booster *Brucella* antigen is present.

Key words: *Brucella abortus*, strain 19 vaccine, serology, adult cow inoculation.

INTRODUCTION

The standard dose ($4-12\times10^{10}$ viable organisms per dose) of *Brucella abortus* strain 19 (S_{19}) vaccine has been used for over 40 years[1,5]. Persistent serological reactions, following the use of this vaccine, are well documented. Greater problems are experienced in agglutination tests than in the complement fixation test (CFT)[5]. The problems are aggravated when animals are inoculated with this product after reaching maturity, although some workers found that CFT titres returned to the negative status within 6 months[13]. Although official policy in South Africa prohibits the indiscriminate use of this vaccine in adult animals, persistent titres resulting from its suspected illegal or unintentional use are still a problem in brucellosis serology (S Herr 1984 Veterinary Research Institute Onderstepoort, Republic of South Africa, unpublished data). In such cases a knowledge of how soon serological reactions may follow on the inoculation and the magnitude and duration of the reaction are all of importance. Although previous work[13] indicated that the reactions in the CFT, in adult cattle, had disappeared by 6 months post-inoculation it is known that the sensitivity of the CFT may vary widely depending on the methods employed[10]. Therefore it was deemed prudent to re-investigate the profiles of serological reactions following standard dose inoculation in typical adult cattle, employing the serological methods used at present.

MATERIALS AND METHODS

Animals and Specimens

Ten adult cows of mixed breeding were used. The previous vaccination history was unknown. None of the cows was pregnant. Blood was collected from the jugular vein on a weekly basis for 4 weeks prior to inoculation and for 5 weeks post-inoculation. Subsequent blood samples were collected on a 2 – 3 monthly basis up to 11 months post-inoculation. The serum was harvested and subjected to the serological procedures described below within 24 h of specimen collection. For the period of the trial the animals were kept in an environment where they were not exposed to a *Brucella* antigen stimulus either from field or vaccine strains.

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Serological Methods

All the sera were subjected to the following 3 serodiagnostic procedures:

The rose bengal test (RBT) was done in haemagglutination plates using a standardised method[2] as modified by Herr[6].

The serum agglutination test (SAT) was carried out in microtitration plates[7]. Titres were recorded in International Units/ml (IU/ml) from a table (Table 1) based on a standard serum containing 1 000 IU/ml giving an end-

Table 1: THE CONVERSION OF SAT END-POINT REACTIONS TO IU/ml ON A SCALE WHERE 50% AGGLUTINATION IN A 1/500 FINAL DILUTION IS EQUIVALENT TO 1 000 IU/ml

Serum[a] dilution	Final[b] dilution	End-point Reactions		IU/ml[c]
		Reading	% Agglutination	
1/5	1/10	1 – +	25	17
		2 – ++	50	20
		3 – +++	75	23
		4 – ++++	100	27
1/10	1/20	1 – +	25	34
		2 – ++	50	40
		3 – +++	75	47
		4 – ++++	100	53
1/20	1/40	1 – +	25	67
		2 – ++	50	80
		3 – +++	75	93
		4 – ++++	100	106
1/40	1/80	1 – +	25	134
		2 – ++	50	160
		3 – +++	75	186
		4 – ++++	100	212
1/250	1/500	2 – ++	50	1 000

[a] Serum dilution = dilution factor with phenol-saline diluent only

[b] Final dilution = dilution factor after the addition of an equal volume of antigen

[c] IU/ml – International Units per millilitre

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point reading of 50% agglutination in the 1/500 final dilution (after addition of all the reagents) of this serum. Twofold serial dilutions of a 1/5 serum dilution were used in a 4 well test giving final dilutions of 1/10 to 1/80 after addition of antigen. A 100 % agglutination in the 1/80 final dilution would give a maximum reading of 212 IU/ml in the test.

The CFT was performed in microtitration plates as previously described[7]. Twofold serial dilutions of sera were used with the 1/2 serum dilution being used as a control for anti-complementary activity. The serum dilutions in the CFT proper extended from 1/4 to 1/128. Titres were recorded in IU/ml based on a table (Table 2) where 1 000 IU/ml represented a ++ or 50 % haemolysis reaction in the 1/220 serum dilution. The highest titre that could be recorded in the CFT would be a ++++ reaction representing 0 % haemolysis in the 1/128 serum dilution and being equivalent to a serum containing 784 IU/ml.

Table 2: THE CONVERSION OF CFT END-POINT REACTIONS TO IU/ml ON A SCALE WHERE 50% HAEMOLYSIS IN A 1/220 SERUM DILUTION IS EQUIVALENT TO 1 000 IU/ml

Serum[a] dilution	Final[b] dilution	End-point Reactions		IU/ml[c]
		% Haemolysis	Reaction	
1/4	1/20	75	1 – +	15
		50	2 – ++	18
		25	3 – +++	21
		0	4 – ++++	24
1/8	1/40	75	1 – +	30
		50	2 – ++	36
		25	3 – +++	43
		0	4 – ++++	49
1/16	1/80	75	1 – +	60
		50	2 – ++	72
		25	3 – +++	86
		0	4 – ++++	98
1/32	1/160	75	1 – +	120
		50	2 – ++	145
		25	3 – +++	172
		0	4 – ++++	196
1/64	1/320	75	1 – +	240
		50	2 – ++	290
		25	3 – +++	344
		0	4 – ++++	392
1/128	1/640	75	1 – +	480
		50	2 – ++	581
		25	3 – +++	688
		0	4 – ++++	784
1/220	1/1 100	50	2 – ++	1 000

[a] Serum dilution = dilution factor with veronal buffer only
[b] Final dilution = dilution factor after all reagents are added
[c] IU/ml – International Units per millilitre

It should be noted that the term "serum dilution" is commonly used in the CFT. It refers to the initial twofold serial dilution of serum with veronal buffer and does not take into account the further dilution factor brought about by the addition of the balance of the reagents used in the test. In the SAT, in contradistinction, the term "final dilution" is commonly used and refers to the dilution of the serum after the addition of all the reagents. The use of the IU/ml system in both these tests obviates the confusion that may arise by using either of these terms.

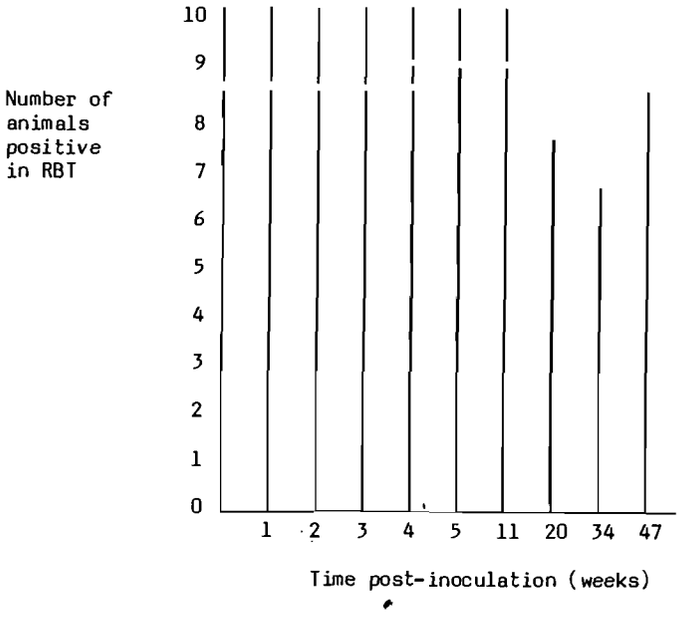

Fig. 1: Serological response of animals in the RBT post-inoculation with S_{19}.

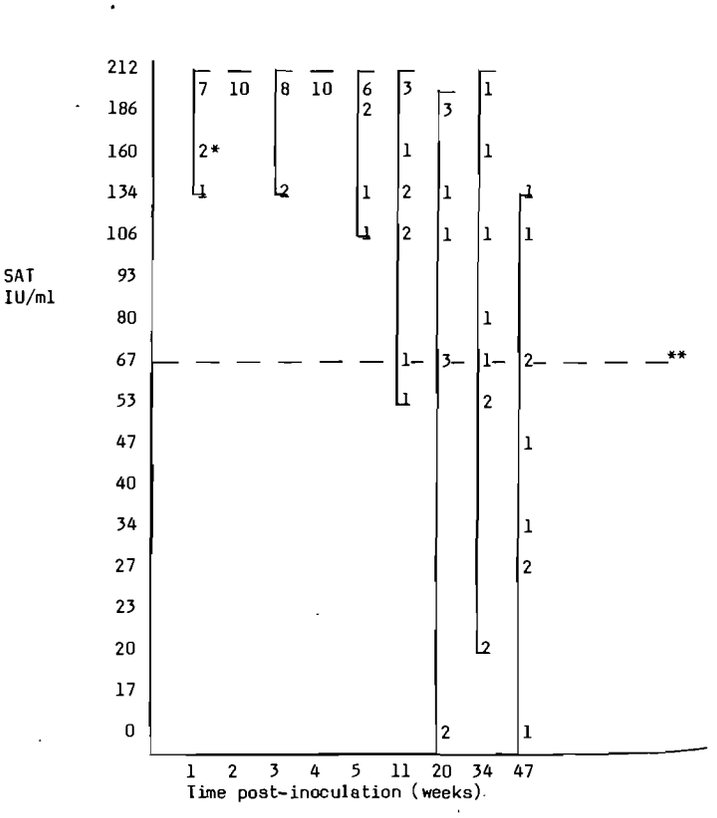

Fig. 2: The range of serological titres in the SAT after inoculation with S_{19}.

*Numerals indicate the number of animals reacting at the antibody concentration expressed in IU/ml; vertical bars represent the range
**67 IU/ml considered as a significant titre in the SAT

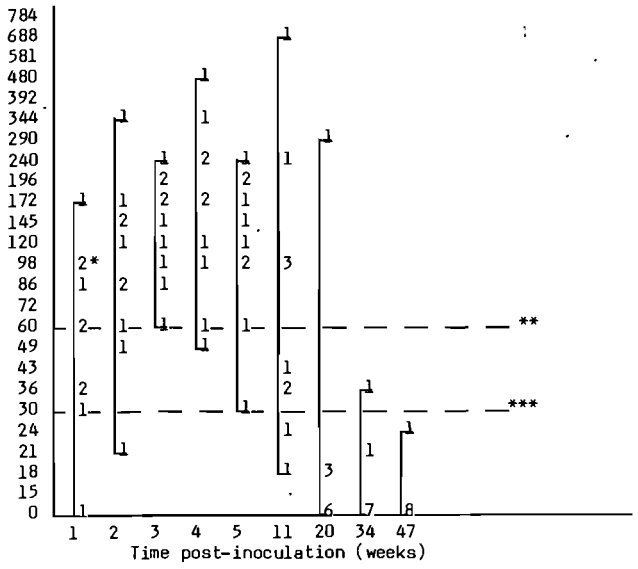

Fig. 3: The distribution of serological titres in the CFT after inoculation with S_{19}.

*Numerals indicate number of animals reacting at the antibody concentration expressed in IU/ml; vertical bars represent the range

**60 IU/ml or higher represents a positive reaction in adult inoculated animals

***30 IU/ml or higher represents a positive reaction in all other animals

Inoculation

Each animal was inoculated subcutaneously with 2 ml of S_{19} (Veterinary Research Institute, 0110 Onderstepoort), containing $4-12\times10^{10}$ viable organisms per 2 ml dose.

Interpretation of serological results

The CFT is used as the definitive serological test in South Africa. In animals that have never been inoculated, in those that have been inoculated as heifers (3 – 11 months of age) and tested as adults (over 18 months of age), and in animals where the inoculation history is unknown, an antibody titre of 18 to 24 IU/ml is regarded as suspicious and 30 IU/ml or higher as positive. In animals that have been inoculated as adults, 30 to 49 IU/ml is interpreted as a suspicious reaction and 60 IU/ml or higher as positive (P Bosman 1979 Department of Agricultural Technical Services Memorandum, File Ref 12/1/8/6/B of 1979-05-11).

The SAT results are not used to decide on the status of the animal, but are used to check the results of the RBT and CFT. Nevertheless, an SAT antibody titre of 67 IU/ml or greater has been shown to be of significance[7].

RESULTS

Eight of the ten animals were negative to all the tests (RBT, SAT and CFT) on the 4 tests prior to inoculation. One animal showed a positive reaction in the RBT on the day of, but prior to, inoculation. Another animal recorded a low titre of 36 IU/ml in the CFT at the same time. The subsequent serological profiles of these 2 animals did not differ from those of the other 8 animals in the trial.

All the animals responded serologically, in the RBT and SAT, within 1 week of inoculation (Fig. 1 & 2). Nine of the 10 animals seroconverted within 1 week in the CFT and all had done so by the 2nd week (Fig. 3). One animal was not available for testing during the 34th week post-inoculation and another was absent during the final (47th) week of the trial, so that only 9 animals were tested during these weeks.

In the RBT all the animals remained positive up until the 11th week post-inoculation. Two animals reverted to negative in the 20th and 34th weeks post-inoculation but were once again positive in the 47th week (Fig. 1).

In the SAT all the animals recorded antibody titres of 134 IU/ml or more for the first 4 weeks of the trial (Fig. 2). During the 2nd and 4th weeks all the animals showed a maximal antibody titre of 212 IU/ml. By the 20th week post-inoculation all but 4 of the animals recorded titres below 134 IU/ml. In the final week of the trial (47th week) the serological reactions had, in all cases, declined to 134 IU/ml or less (Fig. 2).

In the CFT (Fig. 3), 9 out of 10 animals showed serological reactions of 30 IU/ml or higher during the first 2 weeks of the trial. From the 3rd to the 5th week all the animals had reactions of this magnitude. By the 11th week post-inoculation the serological titres of 2 animals had fallen below 30 IU/ml. By the end of the trial 8 animals recorded no reaction at all in the CFT and the remaining 1 was less than 30 IU/ml (Fig. 3). When the 60 IU/ml or higher antibody concentration was considered, all the animals showed serological reactions of this magnitude only during the 3rd week post-inoculation. By the 20th week 9/10 animals had fallen below this antibody concentration and by the 20th week post-inoculation all the animals had titres of below 60 IU/ml (Fig. 3). In this trial no animal had the maximum antibody titre (784 IU/ml) attainable in this test. Between the 2nd and 20th weeks of the trial from 1 to 4 animals showed serological reactions in excess of 196 IU/ml (Fig. 3).

DISCUSSION

The serological reactions observed in this trial were uninfluenced by contact with extraneous sources of *Brucella* antigen. In the field such sources may come from contact with infected animals, from S_{19} vaccine residues in syringes[3] or from the repeated use of S_{19} vaccine. Where such booster effects occur it would be reasonable to expect the antibody titres to be higher and to persist for longer periods.

The reactions in the RBT persisted the longest, with all the animals showing a positive reaction 47 weeks post-inoculation. The persistent RBT reactions have a great nuisance value as this test is used as a screening test and all reactors to the test must be subjected to the CFT. The latter test is technically more exacting and the laboratory is not designed to deal with high percentages of CF tests in routine serum samples.

Although one of the disadvantages of the SAT is that reactions are generally delayed longer than those in the CFT following infection[9], this is not equally true for reactions in the SAT following S_{19} inoculation[13]. The latent and incubation periods of up 2 years as described in infection[4,12] are not seen following S_{19} inoculation[4,13]. The finding that the SAT antibody titres recorded in this trial peaked for the greater part before those seen in the CFT is in accordance with the findings of other workers[8,13].

In the CFT the reactions post-inoculation were

delayed for no longer than 3 weeks and tended to peak at 5 to 11 weeks. They had returned to negative status by 47 weeks which is in agreement with the findings of other workers[11 13]. The magnitude of the CFT reactions (Fig. 3) was such that a high proportion of the animals would be classified as positive serological reactors for at least 11 weeks post-inoculation.

In sera coming from the field one may suspect the illegal or unintentional use of the standard dose S_{19} if a high proportion of animals suddenly develops positive serological reactions. These reactions, especially in the CFT, would wane within the next 6 to 11 months. At the same time there should be not concomitant *Brucella* abortions on the farm. Where the booster effect from either field infection or repeated contact with vaccine antigen occurs greater difficulty in differentiating between vaccination and infection would be expected.

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THROMBOEMBOLIC MENINGOENCEPHALITIS DIAGNOSED IN NATAL

J.P. KITCHING*, G.C. BISHOP* and P.H. MAPHAM**

ABSTRACT: Kitching J.P.; Bishop G.C.; Mapham P.H. **Thromboembolic meningoencephalitis diagnosed in Natal.** *Journal of the South African Veterinary Association* (1985) 56 No. 2, 97-98 (En). Allerton Regional Veterinary Laboratory, P/Bag X9005, 3200 Pietermaritzburg, Republic of South Africa.

Nine feedlot cattle showed clinical signs consistent with those expected in thromboembolic meningoencephalitis. These signs included pyrexia, ataxia, posterior paresis, paralysis and coma. Brown necrotic foci with haemorrhagic borders were observed in the brains of three animals that had died. In these foci vasculitis, thrombosis, infarction and neutrophil infiltration were observed during microscopical examination. *Haemophilus somnus* was isolated in pure culture from the brains.

Key words: Thromboembolic meningoencephalitis, TEME, *Haemophilus somnus*, cattle.

INTRODUCTION

Thromboembolic meningoencephalitis (TEME) caused by *Haemophilus somnus*[3] has, to our knowledge, not previously been reported in the Republic of South Africa. The disease is frequently an acute, septicaemic condition characterized by a high mortality rate and occurs primarily in yearling feedlot cattle[4].

Early clinical signs include temperatures of up to 42°C and uni- or bilateral blindness[1,3,4]. Later clinical signs include ataxia, circling, muscle tremors, paralysis and coma. Humphrey et al[3] consider respiratory signs an integral part of TEME.

During autopsy the lesions found are haemorrhagic infarcts in various regions of the central nervous system[1-4]. Microscopically TEME is characterized by severe vasculitis, thrombosis, infarction and a cellular exudate consisting mainly of neutrophils.

Adhesion of *H. somnus* to the vascular endothelial cells followed by exposure of subendothelial collagen may initiate the thrombosis, vasculitis and ischaemic necrosis[5].

The causative bacterium, *H. somnus*, is an exacting microaerophile which requires enriched media for primary isolation[3].

HISTORY AND CLINICAL SIGNS

An outbreak of disease in feedlot cattle from a local Pietermaritzburg farm was investigated in the period covering June 1984 to August 1984. These animals, all aged between 12 and 18 months, exhibited marked neurological signs. Temperatures of up to 40,5°C were recorded. Congested mucous membranes and ataxia with knuckling of the hind fetlocks were consistent features. As the disease progressed, the temperatures returned to normal and posterior paresis set in which very rapidly progressed to paralysis, coma and then death. Affected animals had been in the feedlot for more than 30 days. The stockman routinely treated animals with imidocarb diproprionate (Forray-65, Coopers) as the farm is situated in a known babesiosis and anaplasmosis endemic area. Because other diseases considered initially were listeriosis, heartwater and pasteurellosis, intravenous treatment with oxytetracycline HCl (Engemycin, Wellcome) was instituted. A good response to treatment was obtained in the febrile stage of the disease in six animals showing clinical signs similar to those seen in the three cattle that died.

PATHOLOGY

Autopsies were performed on 3 cattle. Minimal gross pathological changes were found other than those in the brain. Brown to grey foci 1 – 12 mm in diameter surrounded by haemorrhagic borders and distributed at random in the brain tissue, were evident in two of the animals. In the third case similar lesions were confined to the medulla oblongata. Lesions seen on the brain surface extended inward in a cone shape.

Microscopically the brain lesions were characterized by vasculitis and thrombosis with infarction and haemorrhage. Adjacent areas were densely infiltrated mainly by neutrophils and a few round cells. In two of the cases Gram negative bacilli were seen in sections of the brain lesions.

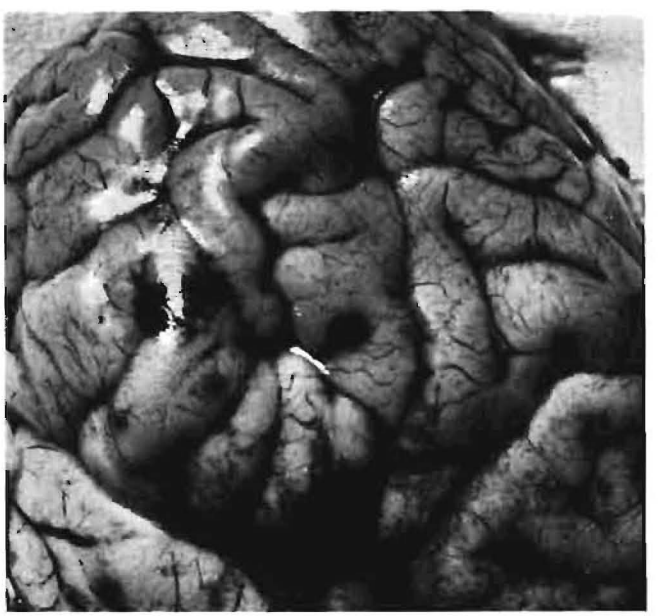

Fig. 1: Focal haemorrhagic lesions on brain surface.

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BACTERIAL ISOLATION

Approximately 5 g of brain tissue including lesions was homogenized for 1 minute in about 20 ml sterile physiological saline. Aliquots of 0,1 ml were spread over the surface of Columbia Agar (Bacto-Columbia Blood Agar Base, Difco) plates containing 7 % whole citrated sheep blood and were incubated at 37°C for 72 hours in an atmosphere containing 10 % CO_2.

The growth of *H. somnus* after 48 hours incubation appeared as grey-yellow convex, circular, entire colonies about 1 – 2 mm in diameter. The yellow colony colour was enhanced when the colonies were scraped off the agar surface and raised using a bacteriological loop. No haemolysis was evident. Pure cultures of the pathogen were obtained from the brain specimens in all 3 cases.

The staining, biochemical and morphological characteristics of these isolates conformed to those of *H. somnus*[3,4]. Although recovery of the pathogen from the brain was successful, no isolates were made from the lungs of the three animals.

DISCUSSION

The condition observed in the Pietermaritzburg area of Natal resembled very closely the description of TEME recorded in the literature[1-4]. The staining and biochemical reactions as well as the cellular and colonial morphology of the bacterium also conformed to those described in the literature. Although it has been claimed that the respiratory tract is consistently involved in TEME[3], we failed to isolate *H. somnus* from the lungs. This was probably due to the profuse overgrowth by *Pasteurella haemolytica* and *Corynebacterium xerosis* and the effect of previous antibiotic treatment. A clearer knowledge of the prevalence of TEME will depend on the more routine use of bacteriological methods suitable for the isolation of *H. somnus*. Since such methods are not often routinely applied, the importance of TEME in South Africa may have been underestimated up to this stage.

ACKNOWLEDGEMENTS

The authors express their sincere appreciation to Miss Colleen Dale and Mrs Beverley Coetser for their technical assistance, to Mrs Ann Johnstone for typing the manuscript and to the Director of Veterinary Services for permission to publish this article. The support and assistance in the identification of this pathogen given by Dr M. Henton of the Veterinary Research Institute, Onderstepoort, is gratefully acknowledged.

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BOOK REVIEW — BOEKRESENSIE

DISEASES OF POULTRY

M.S. HOFSTAD, H.J. BARNES, B.W. CALNECK, W.M. READ, H.W. YOUDER, Jr. (EDS)

8th Edn. The Iowa State University Press, Ames, Iowa 50010. 1984 pp 830, illustrations 570. Price $75 (ISBN 0-8138-0430-2)

This book describes the diseases of fowls, ducks and turkeys in considerable detail with particular emphasis on the aetiology, pathogenesis, symptoms and pathology but in some cases is somewhat brief on the treatment and control. The book comprises 34 chapters, each written by a single or pair of authors, all accepted authorities in their respective fields.

Several new diseases, egg drop syndrome in the chapter on adenovirus infections, rota virus infections and turkey coryza are included in this edition. Although femoral head necrosis, acute death syndrome and kinky-back are mentioned in the preface, these conditions do not appear in the index. A chapter on bone diseases which are of considerable economic importance in broilers, would be a welcome addition in any future publication. Although this edition is shortened by approximately 100 pages, accomplished by eliminating conditions which are never encountered, it contains a large amount of new knowledge on diseases previously described. The style of presentation is good with an extremely detailed list of references following each disease described. The illustrations are clear and the book is printed on high quality glossy paper. The authors and editorial committee are to be congratulated on producing a volume even better than those of the past.

There is no need to emphasize the value of this book as it is well established as the best English language textbook on the subject, far exceeding any other.

This book can be unreservably recommended to avain pathologists, researchers, diagnosticians and university personel involved in poultry.

L. Abrams

SHORT COMMUNICATION KORT MEDEDELING

GUIDELINES FOR BACTERIAL COUNTS ON CARCASES AT CATO RIDGE ABATTOIR

ANNE SELMER-OLSEN*

ABSTRACT: Selmer-Olsen Anne. **Guidelines for bacterial counts on carcases at Cato Ridge abattoir.** *Journal of the South African Veterinary Association* (1985) **56** No. 2, 99-100 (En). Division of Veterinary Services, Department of Agriculture, P.O. Box 206, 3680 Cato Ridge, Republic of South Africa.

The agar sausage technique was used to make an assessment of the surface aerobic bacterial levels of refrigerated beef, mutton and pork carcases at Cato Ridge abattoir by taking agar imprints from selected sites on the surfaces of carcases.

It was shown that half of the 297 beef carcases examined had less than 200 aerobes cm^{-2}, half of the 298 mutton carcases less than 250 aerobes cm^{-2} and half the 299 pork carcases less than 134 aerobes cm^{-2}. These counts are utilised as guidelines to identify breakdowns in hygiene at the abattoir.

Key words: Aerobic bacterial counts, refrigerated carcases.

INTRODUCTION

The hygiene control programme at Cato Ridge abattoir includes bacteriological monitoring of fixtures, equipment, personnel and carcases using the agar sausage technique[5].

High counts of bacteria can be used as an indication of poor hygiene[1 3 4], and whilst guidelines for levels of bacteria cm^{-2} using the agar sausage technique are available for fixtures and equipment, few are available for carcase monitoring purposes[1 2 6 7].

It is known that on any one day, the range of bacterial counts between carcases and even between sites on one carcase is very large[4 8]. Nevertheless, the range of aerobic mesophilic bacterial counts cm^{-2} on chilled carcases at Cato Ridge abattoir would help to establish guidelines for hygiene standards.

MATERIALS AND METHODS

Agar imprints from selected areas of the surfaces of 297 beef, 298 mutton and 299 pork carcases were taken in groups of 5 of each species, respectively, per week over a period of approximately 60 weeks. Carcases were chosen at random and all had been refrigerated for 18 – 24 h between 0° and 2°C.

The agar sausage technique[3 5], using Tryptone Glucose Extract Agar (Biolab), was employed. Agar sausage imprints were taken after the carcase had been out of the chiller for approximately 2½ h and had been hanging in an ambient temperature of 10°C or less. Twelve agar sausage imprints (6 per side) were taken from the outside of each carcase. On beef carcases the sites examined were the hip, lumbar region, posterior thorax, shoulder, sternum and the belly. On mutton and pork carcases agar imprints were taken from the rump, lumbar region, dorsal neck region, shoulder, sternum and the belly.

The agar sausage slices were incubated aerobically for 24 h at 37°C and the results were interpreted according to Olgaard's method as given by Hobbs[3]. Olgaard's table for "The mean bacterial count cm^{-2} from the mean point value" was adapted by a factor of 1,59 to allow for the smaller sausage diameter of 2,7 cm which we used.

RESULTS

Variations in bacterial counts cm^{-2} on carcases were considerable (see Table 1) and some sites e.g. the shoulder and the neck regions consistently yielded higher counts than other sites.

Table 1: RANGE OF CARCASE SURFACE BACTERIAL COUNTS cm^{-2}

Species	Lowest count cm^{-2}	Highest count cm^{-2}
Beef	16	1 128
Mutton	9	1 749
Pork	14	874

Bacterial counts cm^{-2} from all carcases tested were converted to $\log_e$ values and were analysed by Dr. K.C. Ryan (Department of Statistics and Biometry, Faculty of Agriculture, Natal University).

From the data, it was possible to determine that 96% of beef, mutton and pork carcases had yielded total aerobic mesophilic counts cm^{-2} of 898, 1075 and 655 or less organisms, respectively. Half of the beef, mutton and pork carcases yielded bacterial counts cm^{-2} of 200, 250 and 134 or less, respectively.

DISCUSSION

The results of data compiled at Cato Ridge abattoir show that under routine conditions of hygiene at this abattoir, total aerobic mesophilic bacterial surface counts on 50% of refrigerated beef, mutton and pork carcases should be 200, 250 and 134 organisms or less cm^{-2}. This represents a satisfactory situation.

If over a period of time, 50% of carcases yield higher counts, then the reason for the general rise in bacterial contamination levels should be sought and eliminated.

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CASE REPORT **GEVALVERSLAG**

A HAEMORRHAGIC SYNDROME IN RECENTLY WEANED PIGS ASCRIBED TO HYPOVITAMINOSIS K

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ABSTRACT: Newsholme S.J.; Cullen J.S.C.; Nel P.W.; Reyers F. **A haemorrhagic syndrome in recently weaned pigs ascribed to hypovitaminosis K.** *Journal of the South African Veterinary Association* (1985) **56** No. 2, 101-102 (En). Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa.

An outbreak of a haemorrhagic syndrome involved recently weaned, mixed-breed pigs in a large piggery. The pigs were fed a pelleted complete ration containing antibacterial drugs. Affected pigs failed to grow, became pale and developed large, subcutaneous haematomas. Some pigs became lame and one had epistaxis. The monthly mortality rate in the weaner house, which was previously less than 2 %, exceeded 6 % during the outbreak. Coagulation time, activated partial thromboplastin time and prothrombin time were prolonged in blood from some of the pigs. The outbreak resolved promptly after supplementation of the diet with vitamin K_3.

Key words: Pigs, hypovitaminosis K, haemorrhagic syndrome.

INTRODUCTION

Sporadic outbreaks of a porcine haemorrhagic syndrome have been reported from the United States of America[5]. The disease was most severe in recently weaned pigs, and in most outbreaks the pigs had received pelleted, complete rations containing various antibacterial drugs. Clinical signs included lethargy, anorexia, lameness and swellings over the hams, hind legs and ventral abdomen. Anaemia, subcutaneous haematomas and intramuscular haemorrhage and oedema were evident at necropsy. On occasion, the syndrome has been reproduced by feeding pigs the rations from farms where outbreaks occurred.

Evidence strongly suggests that the syndrome is a manifestation of vitamin K deficiency or antagonism. Blood coagulation times and prothrombin times were prolonged, deficiency of clotting factors VII and X was indicated and there was no evidence of hepatic injury. The underlying causes of this vitamin K deficiency or antagonism have not been identified. Coumarin compounds could not be demonstrated in the feed. Possible causative factors included an antivitamin K mycotoxic agent and reduction of intestinal microbial vitamin K synthesis by antibacterial drugs[4].

Similar outbreaks have been described in France[2], New Zealand[3] and Japan[6], but the syndrome has not been reported in South Africa. In this report we describe an outbreak of a haemorrhagic syndrome in recently weaned pigs in South Africa. The outbreak closely resembles those described elsewhere.

HISTORY OF OUTBREAK

The outbreak affected a large piggery in the Warmbaths district of the Transvaal. The breeding-stock consists of Landrace and Large White sows and boars, and some Duroc boars. Piglets are injected with iron (Gleptosil, Maybaker) at 3 days old and are weaned at 5 weeks. At 6 weeks old, they are removed from the farrowing house to the weaner house, where the litters are pooled, and kept in open-topped wire cages with wire-mesh bottoms.

Home-mixed meal rations were provided throughout the piggery, but a commercially-prepared, pelleted, complete ration had been substituted from time to time. Antibacterial drugs were incorporated into the meal and pelleted rations. Different antibacterials had been used at various times and had included oxytetracycline (TM 100, Pfizer), neomycin (Neomix, Upjohn) and tylosin and sulphur drugs (Tyla Sulf, Elanco).

From February to May 1984, a pelleted ration containing oxytetracyline (TM 100, Pfizer) had been fed. In early May, the owner noticed that the mortality rate in the weaner house was increasing. Previous monthly mortality rates in the weaner house had not exceeded 2 %, but during May, 57 out of 947 weaners (>6 %) died. Increased mortality rates continued into June. Pigs that died were between 7 and 15 weeks old. The owner observed that the pigs became pale before death and that many had swellings on the legs and body. Some became lame. Many pigs in this age group were not growing. No association between the affected pigs and particular litters or parents was apparent.

Necropsies done on the premises showed that the carcasses were pale and that the swellings were large haematomas. Whole blood, collected into tubes from live, pale pigs, took longer than 30 min to coagulate. These findings initially suggested that the pigs had been exposed to a coumarin-containing rodenticide, but detailed inquiry and close inspection of the premises indicated that this was most improbable.

MATERIALS AND METHODS

Pathology

Necropsies were done on 6 weaners which were regarded as typical cases. Various tissues, including liver, were fixed in buffered 10 % formalin. Paraffin sections were prepared from these tissues, stained with haematoxylin and eosin according to routine methods and examined by light microscopy.

Clinical pathology

Blood was collected into citrated tubes from the *vena*

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cava of each of 5 weaners which were pale and had haematomas. Activated partial thromboplastin time (APTT) (PTT Reagent, Boehringer Mannheim) and prothrombin time (PT) (Hepato Quick Boehringer Mannheim) were measured for each sample. Citrated blood from a healthy, aged-matched Duroc pig was used concurrently as a control. Blood smears from each pig were also examined microscopically.

Vitamin K supplementation

In mid-June, 8 g/ton of vitamin K_3 (Vitamin K_3 mix, Panvet) was mixed into the feed.

RESULTS

Pathology

The carcasses of all 6 pigs were pale. Multiple, large, subcutaneous haematomas were situated in various sites, but were most common at the axilla and over the hock and stifle joints. A submandibular haematoma was present in 2 pigs. Smaller haemorrhages were scattered in the musculature, and most of the lymph nodes were haemorrhagic. Haemarthrosis was observed in both hock joints of one pig, and there was evidence of previous epistaxis in another.

Light microscopical examination confirmed the haemorrhagic nature of the lesions, but no other significant changes were observed. No evidence of hepatic injury could be seen.

Clinical pathology (Table 1)

In 4 of the 5 blood samples, both APTT and PT were prolonged, compared to the control. In 3 of them PT was substantially prolonged. Platelet numbers appeared to be adequate in blood smears.

Table 1: ACTIVATED PARTIAL THROMBOPLASTIN TIMES AND PROTHROMBIN TIMES IN AFFECTED PIGS AND CONTROL

Pig	1	2	3	4	5	Control
Activated Partial Thromboplastin Time(s)	25	30	26	23	13	15
Prothrombin Time(s)	38	53	48	120	30	30

Vitamin K supplementation

No more pigs on the premises became pale after vitamin K supplementation. The mortality rate in the weaner house fell abruptly to less than 2 %, and feed conversion efficiency promptly improved.

DISCUSSION

The age of the pigs affected in this outbreak and the clinical and pathological features closely resemble those in the outbreaks described elsewhere, which were related to the feed[2,3,5,6]. From the available evidence, this outbreak was ascribed to hypovitaminosis K. The prolonged coagulation times, APTT and PT, absence of evidence of hepatic injury, and adequate platelet numbers are compatible with hypovitaminosis K. The diagnosis was supported by the prompt disappearance of the syndrome following vitamin K_3 supplementation.

As in the other outbreaks reported, specific underlying causes were not identified. Access to rodenticide was most improbable. When the outbreak occurred, the pigs had been receiving a pelleted complete ration containing oxytetracycline, but the same feed was also being used concurrently in a number of other large piggeries in which the disease did not occur. It has been suggested that susceptibility to vitamin K deficiency in pigs may be genetically influenced, since most of the pigs affected in one outbreak were consanguineous[6]. The pigs affected in our outbreak, however, were of mixed breeding, and were not associated with particular litters or parents.

The piggery involved is somewhat unusual in having wire-bottomed cages in the weaner house. Such cages would minimize the opportunity of the pigs for coprophagia. It has been shown that coprophagia can prevent haemorrhagic disease in young rats fed a vitamin K-deficient diet[1]. Since coprophagia, however, apparently does not occur in normal pigs[7], it seems unlikely that the wire-bottomed cages influenced this outbreak.

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CASE REPORT GEVALVERSLAG

INFERTILITY IN A MALTESE POODLE AS A RESULT OF A SPERM MIDPIECE DEFECT

E.E. OETTLÉ* and J.T. SOLEY**

ABSTRACT: Oettlé E.E.; Soley J.T. **Infertility in a Maltese poodle as a result of a sperm midpiece defect.** *Journal of the South African Veterinary Association* (1985) 56 No. 2, 103-106 (En). Department of Genesiology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

A 2½-year-old Maltese poodle was examined for breeding soundness following a series of unsuccesful matings to fertile bitches. He was found to have only 8% normal sperm in his ejaculate when Spermac-stained smears were examined under the light microscope. The defect most frequently encountered involved the midpiece attachment, and the various manifestations of disintegration found in this region are described and illustrated. Transmission and scanning electron microscopy confirmed light microscopical findings.

Key words: Infertility, dog, sperm midpiece defect.

INTRODUCTION

A variety of sperm defects, known to adversely influence fertility in domestic animals, have been well documented in the bull, boar, and ram[2-9 11]. In the dog, however, little work has been done to establish which abnormalities significantly affect fertility, and thus much of our knowledge is derived from comparison with defects described in other species[8].

CASE PRESENTATION

A 2½-year-old Maltese poodle was presented at our clinic with a history of numerous matings with various bitches of proven fertility, none of which produced a litter. Clinical examination revealed that the dog was in good health, and, on palpation of the genital system, no gross abnormalities could be detected. Semen was collected by digital manipulation into a warm, sterile glass funnel and collecting tube[10]. The dog showed good libido.

SEMEN EVALUATION

A motility evaluation of the sample was arrived at by placing a drop of semen on a warm slide under a coverslip and examining it under a light microscope on a warm stage at 100x and 400x magnification. Thin smears were prepared and stained with "Spermac Stain" (Stain Enterprises, P.O. Box 12421, 0110 Onderstepoort) for light microscopy for the detection of morphological abnormalities. A portion of the original sample was fixed in 4% phosphate-buffered glutaraldehyde, post-fixed in similarly buffered 1% osmium tetroxide and processed for scanning (SEM) and transmission electron microscopy (TEM), using standard techniques.

The sample displayed macroscopic characteristics typical of normal dog semen. Under the coverslip, 40% of the sperm showed progressive motility, 35% showed erratic local motility, and 25% were non-motile.

The most significant finding was on morphological examination where 80% of spermatozoa examined, showed defects of the head-base/midpiece region. Other abnormalities accounted for 12% and only 8% appeared morphologically normal.

The defect was manifested as various degrees of disruption of the midpiece attachment to the head, and this was often noted to be associated with a narrowed head-base (Fig. 1a-g). In the mild form, the midpiece appeared to be swollen (Fig. 1b) while in more severely affected sperm, the midpiece was bent (Fig. 1c) or kinked (Fig. 1d).

In some sperm, one side of the kinked midpiece seemed to rupture (Fig. 1d & e) and in the most severe form the whole midpiece was pinched off (Fig. 1f), the resultant loose head displaying an extremely narrowed head-base (Fig. 1g). Transmission electron micrographs confirmed these findings.

In Fig. 2a, there is a unilateral absence of mitochondria in the *pars ascendens* and the head-base is noticeably damaged and narrowed. In Fig. 2b, there is a severe disruption of the alignment and attachment of the structures in the neck region which appears to have caused the kinking of the midpiece.

The greater detail revealed by TEM studies indicate a defective or incomplete formation of the complex neck region of the sperm, particularly the segmented columns and coarse fibres of the connecting piece, as a primary factor in the development of this abnormality (manuscript in preparation).

Other abnormalities that were noted with light microscopy were lipped acrosomes (Fig. 1e), misshapen heads and double tails (Fig. 1h) and coiled tails characteristic of the "Dag-defect"[2] (Fig. 1i).

Phagocytosis of defective sperm was a prominent feature. Many of the abnormal spematozoa found on TEM were within phagocytes (Fig. 2c). A similar phenomenon in an oligospermic dog has been described[1].

DISCUSSION

Midpiece defects are well documented in bulls. Coubrough & Barker[5] described in detail the ultrastruc-

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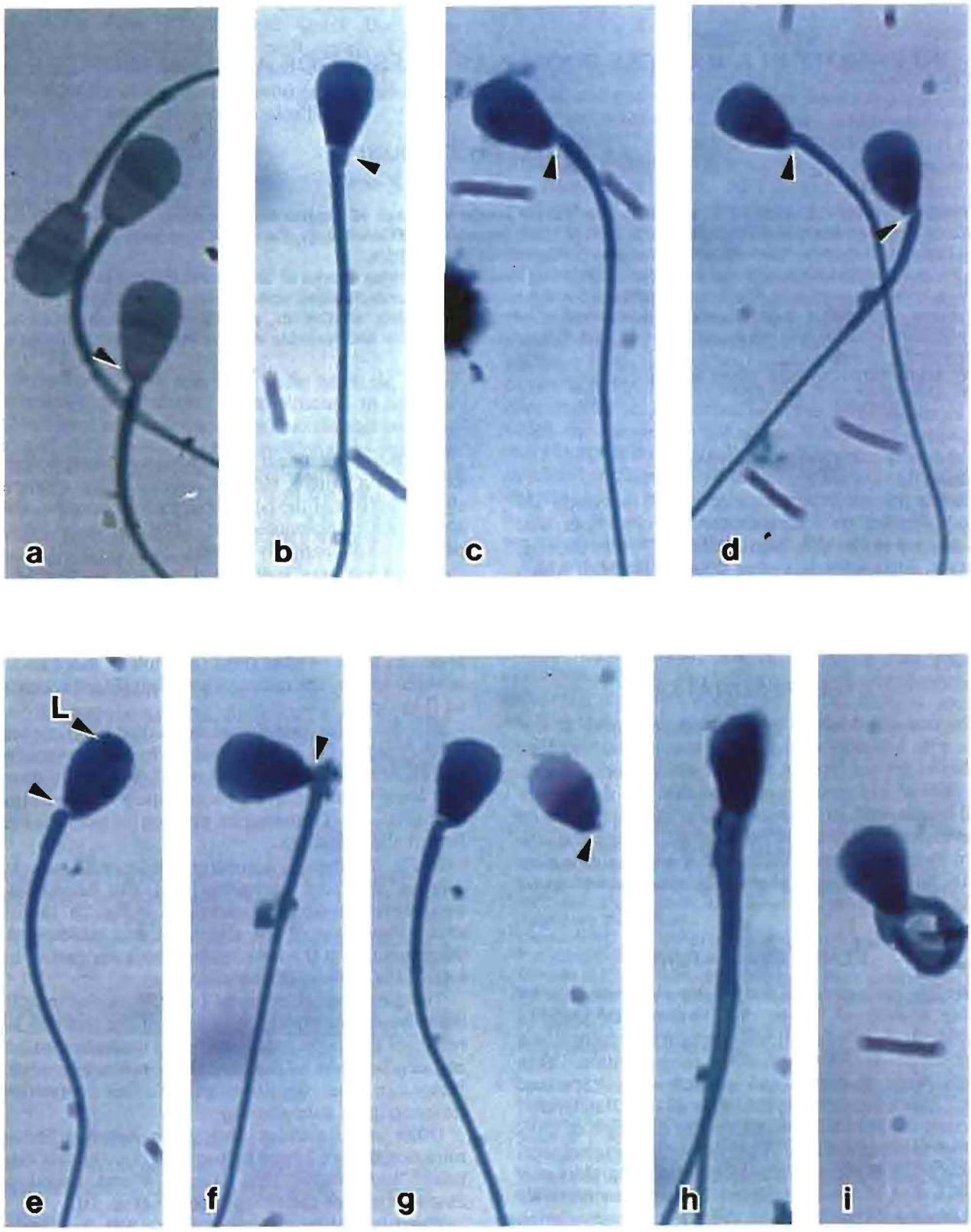


Fig. 1: Spermac-stained thin smears showing degrees of midpiece disintegration. 1 000 x
a) Normal sperm. Note width of head base.
b) Swelling of the midpiece.
c) Bending of midpiece. Note rounding of head base.
d) Kinking.
e) Rupture of one side of the kinked midpiece with narrowed, skew head base. Note acrosomal lipping also present (L).
f) Pinching off of the midpiece. Note severely narrowed head base.
g) Extreme disintegration – loose head, with narrow base.
h) Abnormal head, staining very dark, with double midpiece and tail.
i) "Dag-defect" of the tail.

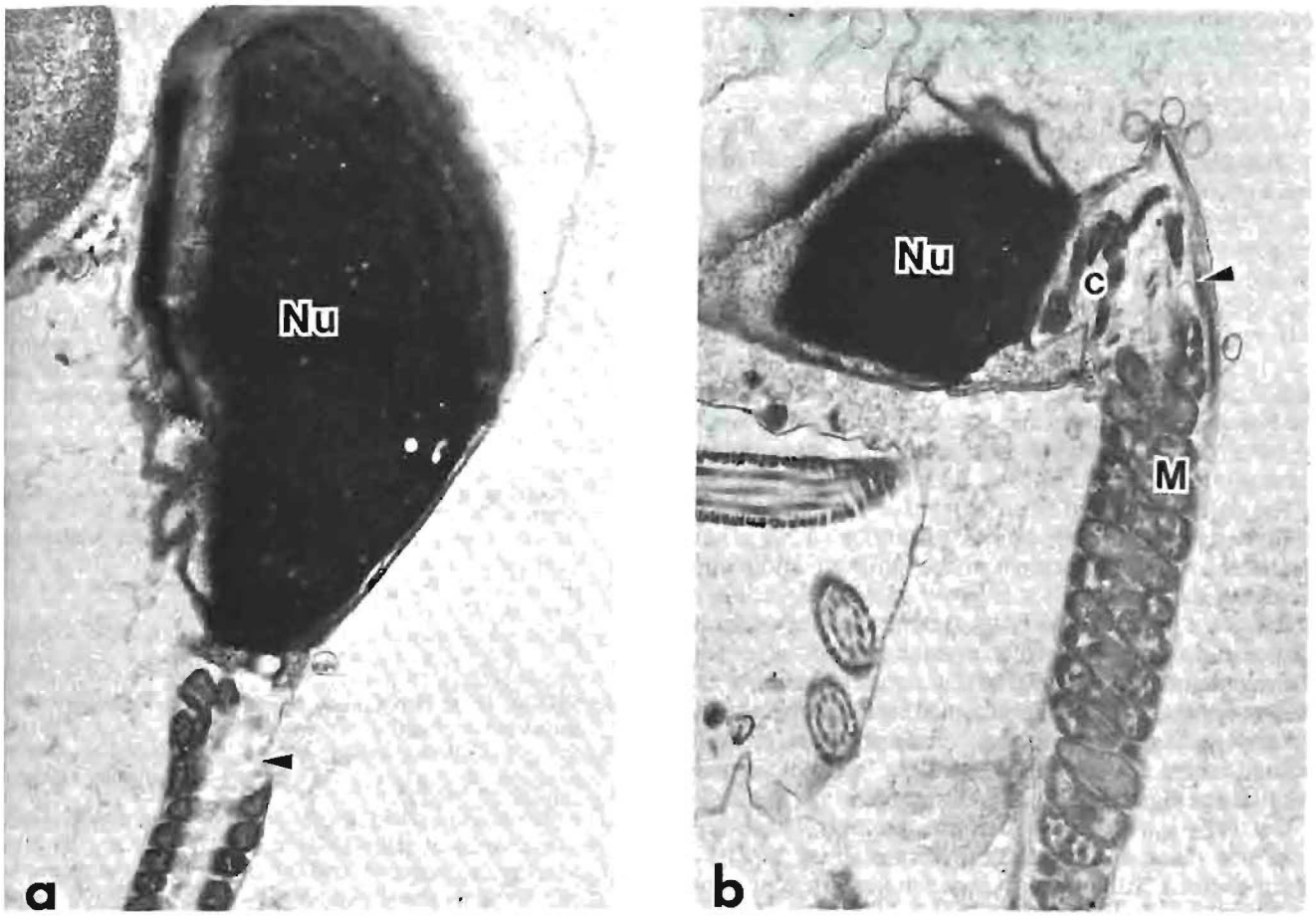
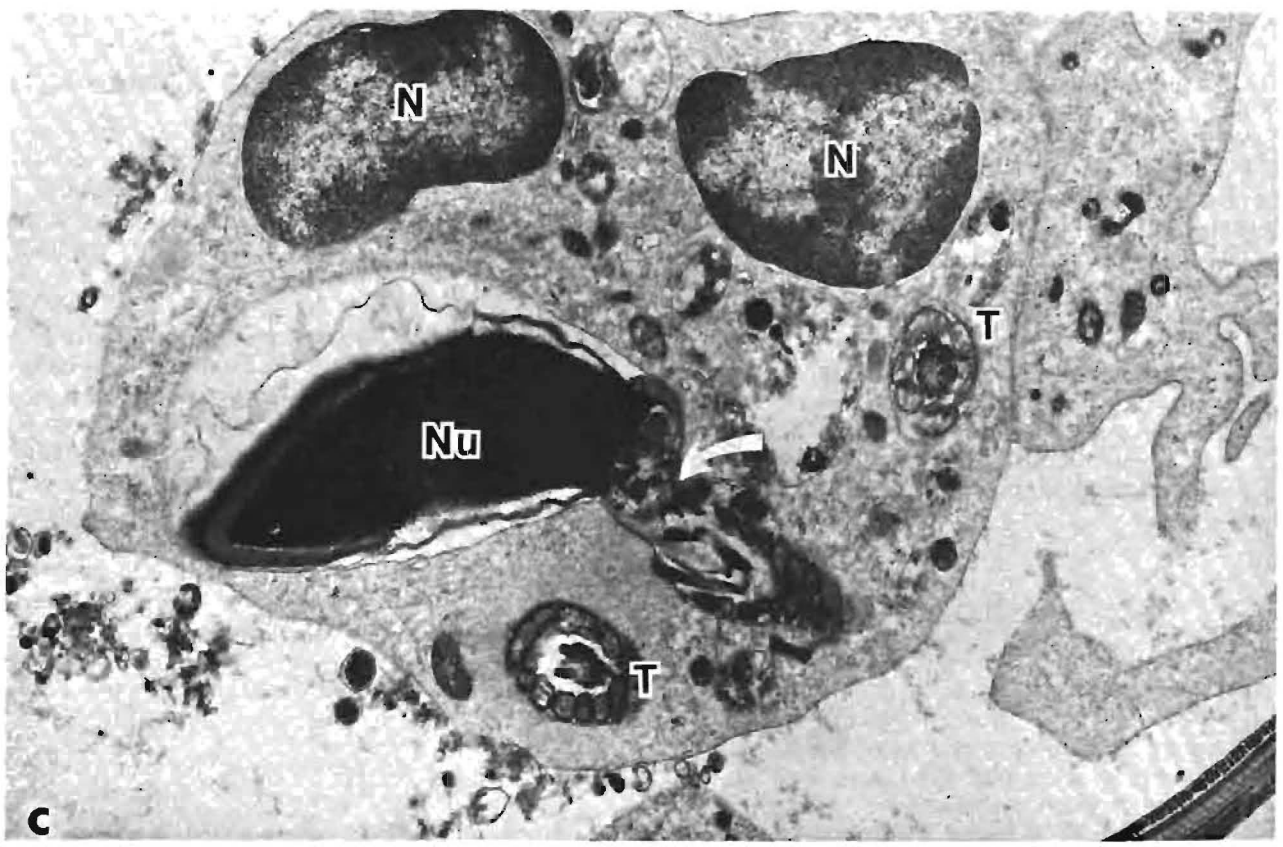


Fig. 2: Transmission electron micrographs.

a) Planar view of a sperm head showing a narrowed head base with unilateral absence of mitochondria in the pars ascendens (arrow). Nucleus (Nu).

b) Oblique section through a sperm head showing severe disruption of the head base attachment, with kinking of the midpiece. (Compare with Fig. 1f) (Note the nucleus (Nu), proximal centriole (c), mitochondria (m) of the *pars spiralis* and the misplaced coarse fibres (arrow) lying beneath the cell membrane. 23 000 X)

c) A section through a macrophage. Note profile of sperm head nucleus (Nu), with kinked midpiece (arrow), tails (T) and lobes of the macrophage nucleus (N). 18 000 X



ture of disintegrated midpieces. They found that the midpiece was very short and the tail was absent. The abnormality appeared to be progressive, and involved varying degrees of mitochondrial disorganisation and fragmentation of the axial filament complex. Settergren & Nicander[11] described an abnormality in sterile bulls similar to the one presented here. They noted a narrowing of the head-base in some sperm, but in general the midpiece was narrowed, with various abnormalities of the implantation region. They also found a high percentage (50%) of loose heads associated with the defect.

Blom & Birch-Andersen[3] described a heritable condition in Guernseys which they termed the "decapitated sperm defect", where there were 100% loose heads in the ejaculate. Testicular and epididymal samples revealed misalignment of the midpiece, kinking, and gradual disintegration of the midpiece attachment.

The defect described in this paper is essentially similar to those described in the bull by the above authors, and clearly shows a derangement of the midpiece and its attachment to the head.

This case report emphasizes the importance of morphological evaluations of spermatozoa in assessing genital soundness of dogs. If a cursory motility examination only had been performed, the cause of the infertility could not have been determined. With only 8% normal sperm present in the ejaculate, it is not surprising that the dog was found to be sterile.

Correctly stained smears for morphological examinations are valuable aids to the practitioner as part of a routine semen evaluation, to broaden thus the scope and quality of services offered to the clientiele.

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CASE REPORT **GEVALVERSLAG**

SUSPECTED TYZZER'S DISEASE IN TWO FOALS

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ABSTRACT: Van der Lugt J.J.; Coetzer J.A.W.; Jordaan P.; Marlow C.H.B. **Suspected Tyzzer's disease in two foals.** *Journal of the South African Veterinary Association* (1985) **56** No. 2, 107-108 (En). Section of Pathology, Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa.

Tyzzer's disease was diagnosed histologically in two foals, a 4-week-old Thoroughbred cross and a 6-week-old Arabian foal. Clinically both foals were in good health prior to a short illness lasting only a few hours. The liver lesions in both foals were characterized microscopically by multiple foci of necrosis. Variable numbers of elongated slender intracytoplasmic bacilli resembling *Bacillus piliformis* were demonstrated within hepatocytes bordering the necrotic foci.

Key words: Tyzzer's disease, hepatic necrosis, foals.

INTRODUCTION

Tyzzer's disease in foals is characterized by multifocal hepatic necrosis and is caused by *Bacillus piliformis,* a Gram-negative, spore-forming[10], obligate intracellular bacillus[8].

The organism was initially associated with diarrhoea in mice[10]. Subsequently Tyzzer's disease has been reported world-wide in a variety of animals including rodents, rhesus monkeys, domestic cats, dogs and horses in Canada[8 12], the United States of America[4-6 9], England[11], Australia[1] and New Zealand[2].

In these species, the disease is clinically characterised by episodes of diarrhoea, debility and high mortality, especially in young animals[3]. In laboratory animals losses may be extensive, while in foals the disease occurs sporadically affecting individual animals, between 7-42 days of age.

In this paper we report the first cases of an illness suspected to be Tyzzer's disease in foals in South Africa.

HISTORY AND MACROSCOPICAL PATHOLOGY

Tissues from two foals, fixed in 10% buffered formalin, were submitted from the Regional Veterinary Laboratory, Middelburg, Cape Province to the Veterinary Research Institute, Onderstepoort for histological examination.

Foal 1:

A 4-week-old Thoroughbred-cross foal, was found in a collapsed state, had a temperature of 40,5°C and showed congested mucous membranes and icterus. Despite intensive treatment it died within 4 hours after first being observed. At necropsy multiple, variable sized haemorrhages were seen in the epicardium, diaphragm, gastric mucosa, peritoneum and the renal pelvis. Multiple yellowish foci, 1-3 mm in diameter, were observed in the liver. Liver, spleen, kidney and lung specimens were submitted for microscopical examination.

Foal 2:

A 6-week-old Arabian foal, was observed to be depressed in the morning and was found dead 2 hours later. At necropsy numerous greyish-yellow, slightly raised foci, 1-10 mm in diameter were scattered throughout a reddish-brown discoloured liver (Fig. 1). Copious amounts of blood were found in the abdominal cavity as a result of rupture of the liver. The kidneys were moderately enlarged. Liver, spleen, kidney, heart and brain specimens were submitted for microscopical examination.

No specimens were submitted for bacterial culture.

MICROSCOPICAL PATHOLOGY

Multiple foci of necrosis were evident throughout the livers in both foals (Fig. 2). Some of the necrotic foci coalesced to involve entire lobules while others extended into the portal triads and Glisson's capsule. In Foal 1, fibrin was observed in some of the necrotic foci. A spectrum of hepatic lesions ranging from acute to chronic were observed in Foal 2. An inflammatory exudate consisting primarily of neutrophils and a few macrophages and lymphocytes surrounded many of the necrotic foci. Some foci showed evidence of fibroplasia. Fibrosis around central veins and in portal triads occurred in some lobules.

At the periphery of the necrotic foci, numerous bacilli were demonstrated in the cytoplasm of intact hepatocytes with the Warthin-Starry staining method[13] (Fig. 3). The organisms were long slender rods, 0,5 μm x 6-10 μm, sometimes faintly banded and arranged in a parallel or haphazard criss-cross pattern. Organisms were rarely seen extracellularly in sinusoids. The bacilli were not demonstrable in sections stained with haematoxylin and eosin (HE), Giemsa, periodic acid-Schiff (PAS) or Gram staining methods.

No lesions were observed in the other tissues.

DISCUSSION

The morphology of the organisms and the associated microscopical changes in the liver resemble those described for Tyzzer's disease[1-6 8 9 11 12]. Although no myocardial lesions were seen in our cases, minute foci of necrosis and myocarditis have occasionally been reported in foals[1 11]. Specimens for bacterial isolation were not submitted. It must, however, be pointed out that *B.*

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Fig. 1: Enlarged and mottled liver. Note rupture of capsule.

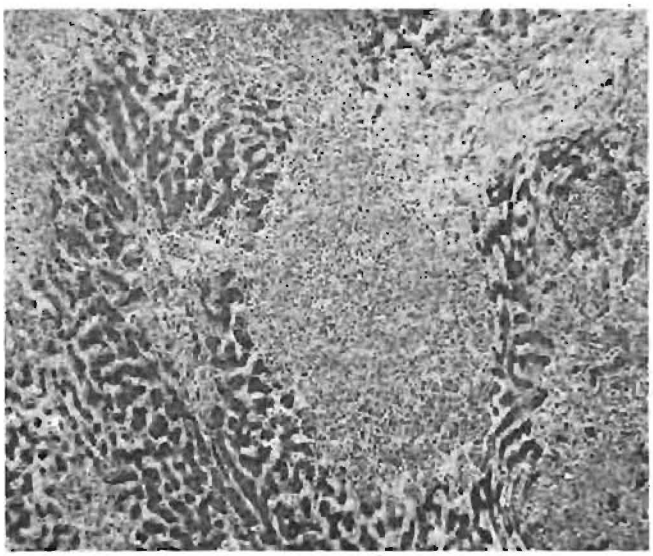

Fig. 2: Randomly distributed foci of hepatic necrosis. HE X 70

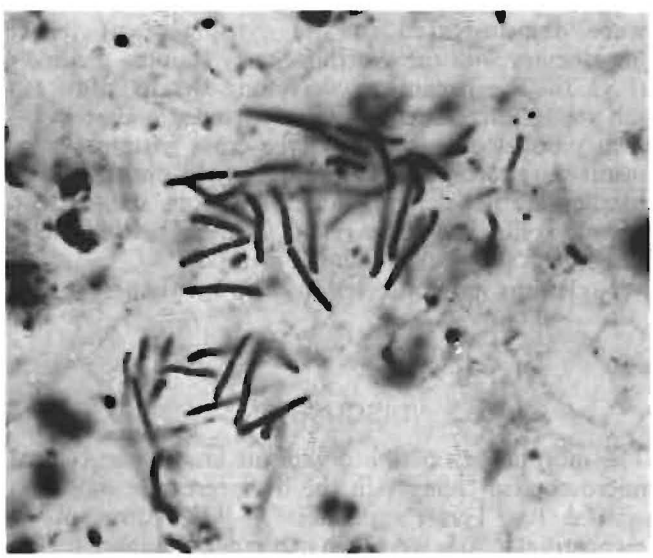

Fig. 3: Organisms resembling *Bacillus piliformis*. Numerous elongated rods in and adjacent to necrotic foci in the liver. Warthin-Starry X 980

piliformis is difficult to isolate by conventional methods[3,7] but that it does grow in embryonated chicken eggs[8]. Though the morphological changes are considered to be sufficiently characteristic for diagnostic purposes, final confirmation of the presence of this disease in South Africa awaits isolation and characterisation of the organism.

Whitwell[11] speculated that apparently normal foals my be suffering subclinical liver necrosis for some time before clinical signs develop. Successive cycles of hepatocellular necrosis after the inital infection of the liver may explain the acute and more chronic changes of the lesions in Foal 2.

Little is known about the epidemiology of the disease in horses. Swerczek[7] has shown that foals may become infected by organisms that are shed in the faeces of adult carrier horses. Environmental contamination may possible also result from other susceptible species such as rabbits, rats, mice and cats[6]. As foals are naturally coprophagous during the first few days after birth, the most likely route of infection would seem to be per os[1]. No sex, breed or other predisposing factors have been identified in foals[9].

Practitoners should consider Tyzzer's disease as a possible cause of death in foals following a short course of illness, especially under stud conditions where a foal is regularly exposed to the faeces of its dam[11].

ACKNOWLEDGEMENTS

The authors wish to thank the staff of the Sections of Pathology and Photography, Veterinary Research Institute, Onderstepoort, for the preparation of the histopathological sections and photomicrographs, respectively.

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CASE REPORT GEVALVERSLAG

ENDODONTIC THERAPY ON AN AFRICAN LION (*PANTHERA LEO*)

R.H. KEFFEN*

ABSTRACT: Keffen R.H. **Endodontic therapy on an African lion (*Panthera leo*).** *Journal of the South African Veterinary Association* (1985) **56** No. 2, 109-110 (En). State Veterinarian, Private Bag X1005, 0302 Mogwase, Bophuthatswana.

The endodontic therapy used on an African lion was similar to procedures performed on humans and other exotic animals with the same condition. The procedure involved removal of the pulpal tissue to the apex of the root in three canine teeth. The internal aspect of the root canal was sterilized and filled with a rubber-like material (gutta percha) which was cemented in place. Chrome cobalt alloy crowns were fabricated and cemented to the top two canine teeth. The lower canine tooth received a silver amalgam restoration.

Key words: Endodontic therapy, African lion, *Panthera leo*.

INTRODUCTION

Endodontic therapy on exotic species is not a new concept and bears great promise in tooth restoration of affected animals. Fractured or worn teeth will expose the sensitive dental pulp to invasion by micro-organisms from the oral cavity and result in abscessation, bone resorption, and eventual evulsion of the tooth. The severe pain upon mastication results in anorexia and loss of body condition. Endodontic therapy is required to preserve the affected teeth, reduce the pain, and provide a functional dental arcade to perform the masticatory needs of the animal. With variations to the basic technique, endodontic therapy has been performed on such exotic animals as the Siberian tiger (*Panthera tigris altaica*)[10], polar bear (*Thalarctos maritimus*)[4], sun bear (*Helarctos malayanus*)[2], White handed gibbon (*Hylobates lar*)[1], capuchin monkey (*Cebus* sp.)[7], orangutan (*Pongo pygmaeus*)[3], and gorilla (*Gorilla gorilla*)[9].

HISTORY

A nineteen year old African lion (*Panthera leo*) had fractured an upper left canine tooth and it was noticed that the lion had lost considerable weight and was salivating excessively. For the two months prior to this observation, the lion had had a precarious appetite. This was thought to be the result of fractured canine teeth acquired from constant chewing on the wire of the enclosure. This habit may have been the result of a need for peridontal stimulation which was lost as a result of the domestic diet or as a form of release of frustration brought about by a static environment.

CLINICAL FINDINGS

Close examination of the lion required tranquilization with the use of a remote delivery system containing 1.3 g of ketamine (Ketalar, Parke Davis), and 300 mg of xylazine (Rompun, Bayer). The oral examination revealed fractures of the two upper canine teeth and the lower left canine tooth. The fractures of the two upper canine teeth occurred two centimeters above the gingival margin and the lower left canine was fractured at the gingival margin. All the fractures exposed the sensitive dental pulp to the oral cavity. Erosions and ulcerations of the gingiva were extensive around the canine and cheek teeth as well as the hard palate. Radiographs of the dental arcade indicated that the canine teeth appeared sound, but could not rule out the possibility of periapical abscesses of the maxillary and mandibular cheek teeth. In retrospect an apisectomy should have been considered in order to rule out this possibility, however, this was not done. A moderate amount of dental tartar was evident on all teeth and was removed.

DIAGNOSIS

A diagnosis of pulpitis of the upper canine teeth and lower left canine tooth was based on the clinical examination and impaction of the pulpal canal with food particles. The history of excessive salivation and anorexia also aided in the diagnosis. Gingivitis and mucosal ulcerations were evident.

TREATMENT

Metronidazole (Flagyl, Maybaker) was given orally (50 mg/kg) for five days after the clinical examination. Following treatment the excessive salivation ceased. Two weeks after the clinical examination, the lion was again tranquilized with 1.3 g of ketamine (Ketalar, Parke Davis) and 300 mg of xylazine (Rompun, Bayer), and this time brought to the veterinary college for root canal therapy. Under tranquilization the lion was intubated (20 mm tube) and maintained on gas anaesthetic (1-2% Halothane) for the entire endodontic procedure. At this time the ulcers of the palate and the gingivitis were no longer evident. The radiographs taken at the time of the clinical examination were used to determine the length and degree of curvature of the root canal of each canine tooth.

A 5 cm dental file was inserted through the exposed root canal, and in a push pull motion, was used to extract the pulpal tissue and file the walls of the canal to a predetermined size. A considerable amount of bleeding ensued and a combination of suction and flushing of the root canal with hydrogen peroxide brought about haemostasis. The use of hydrogen peroxide also aided in flushing out the dental filings. A progression from small

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to large files resulted in an increase in root canal diameter. After filing was completed, the canal was cleaned and dried using numerous sterile pipe cleaners. Each tooth was flushed with a sodium hypochlorite solution to aid in the removal of proteinaceous substances left in the canal and to sterilize the canal. After flushing with the hypochlorite solution, the canine tooth was again cleaned and dried using the pipe cleaners.

The final result was a sterilized root canal which is able to receive the final filling agent. A master gutta percha cone (an enantiomer of the natural rubber – isoprene) was dipped in a dental sealer cement and placed into the root canal. The master cone was then packed tightly against the apex of the tooth using a root canal spreader. A second smaller cone of gutta percha was inserted into the canal and packed tightly. This was repeated until the root canal was completely filled with gutta percha.

Each canine tooth was cleaned, ground, and shaped to remove tartar and rough edges in order to prepare them for the impression casts. Impression casts were made of the upper canine teeth and lower left canine tooth, and were used as moulds to fabricate the caps for the teeth. After the casts were taken, all three canine teeth were given temporary fillings in order to protect the root canal until the caps could be made and fitted at a later date.

A period of one month had lapsed between the root canal therapy and capping of the canine teeth. Chrome-cobalt alloy crowns were applied after the removal of the temporary filling and grinding of the enamel of each canine tooth in order to remove edges and allow a tighter fit. A zinc cement was used to firmly attach the crowns to the canine teeth. The fracture line of the lower left canine tooth was too close to the gingival margin to allow a stable crown to be applied. A silver amalgam restoration or permanent filling was substituted for the crown. A positive contour had been placed on the caudal aspect of each crown in order to avoid "locking" of the teeth if the lion bit the wire mesh of his enclosure.

OUTCOME

After the endodontic therapy, an increase in appetite resulted in an appreciable weight gain and improvement in general body condition of the lion. It can be concluded that the therapy was successful in this particular dental problem.

DISCUSSION

Endodontic therapy has been performed most frequently in humans and dogs[6,8]. The basic procedure, however, has been applied in exotic animal medicine with very beneficial results[1-5,7,9-11]. Root canal therapy was performed on a Siberian tiger[40]. The protocol was similar to the one described here, however, the root canal was filled with RC-2B mixed with RCTM liquid which is a formula containing obtundants, bacteriocidals, and anti-inflammatory agents. A zinc oxide, eugenol, and formocresol mixture was used as a successful filling agent for exotic carnivores[5,11]. Adaptic (Johnson & Johnson) (an epoxy/plastic combination with a quartz filler) sets very quickly and has found enormous success as a filling material in exotic animals[2]. The human dentists working on this case used gutta percha as a filling material and found it to be a successful agent. Chrome cobalt caps[11], silver amalgams[2,9], and Nuvaseal (Caulk Co.)[1,10] (a quartz fill composite which requires ultra violet light for hardening) have been reported to be used as final restorations for teeth. One unique difference between the procedure described here and any other procedure described in the literature, is the use of sterile pipe cleaners for cleaning and drying of the root canal.

ACKNOWLEDGEMENTS

I would like to thank the anaesthesia staff of the Department of Clinical Studies, Western College of Veterinary Medicine, Saskatoon, Saskatchewan, for their assistance during the endodontic surgery. I would also like to thank the Department of Dentistry for performing the operation and providing free equipment and advice. Thanks to Dr J Haigh of the Department of Clinical Studies, Western College of Veterinary Medicine for his critique of this paper.

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TO THE EDITOR — AAN DIE REDAKSIE

TREATMENT OF MALIGNANT EPULIS IN THE DOG

I read with interest Dr J S J Odendaal and Dr J D E Cronje's article in the Journal of the South African Veterinary Association (1984) Vol 55, 209-210 on a case of a malignant epulis in a dog treated by irradiation. I would like to comment on some aspects of this publication.

The title of this article suggests that a considerable number of cases have been treated, but in fact only one case is described.

In the introduction, the authors point out that an epulis is a benign growth of the periodontal rests of squamous epithelial cells. This is most confusing. The only epithelial cells present in the periodontal ligament are the epithelial rests of Malassez, being the remnants of Hertwig's epithelial root sheath[2]. Tumours originating from these (cuboid) cells include ameloblastoma, odontogenic adenomatoid tumour and calcifying epithelial odontogenic tumour[13 15]. The bulk of the periodontal ligament and of an epulis consists of connective tissue[2 3 10]. Although an epulis is covered by epithelium and usually contains strands of epithelial tissue[3 4 10], it is incorrect to describe an epulis as a condition mainly originating from the epithelial component of the periodontal ligament.

The true nature of an epulis remains a matter of controversy. In the dental literature an epulis is usually regarded as a form of gingival hyperplasia induced by local irritation[10 15]. In the veterinary literature epulis is classified as a benign oral growth or neoplasm[1 3 4 12 14] Dubielzig et al.[3] divided epulides in 3 types, namely fibrous, ossifying and acanthomatous epulis, the latter being infiltrating in bone. The term "malignant epulis" has not been used, except in one publication where an ameloblastoma was considered to be the "malignant counterpart" of an epulis[11]. There is, however, unanimity as far as the localization of an epulis is concerned[1 3 4 6 10 12 14 16]. By definition an epulis can only arise on the gingiva and not "usually in the oral mucosae", as stated by the authors. An epulis occurs directly against the gingival margin of one or more teeth. In my limited experience, it would appear that the tooth associated with the epulis usually shows signs of strictly localized periodontitis. Whether this is the cause or the result of the epulis is questionable.

Dr Odendaal and Dr Cronje further state that it is suspected that an epulis could be the initial stage in the development of a carcinoma or sarcoma, hereby presumably referring to Theilen & Madewell[16] (reference given in the first half of the sentence involved). The exact words of the latter authors are: "Whether epulis is a premalignant change that may subsequently progress into carcinoma or sarcoma has yet to be scientifically examined"[16]. The difference may appear subtle but is of importance. I have found no evidence in the literature reviewed that there is an indication or a suspicion that an epulis could be considered as a premalignancy.

The case presented by the authors involved a growth on the rostral side of the hard palate. From the accompanying follow-up picture however it would appear that the caudal part of the hard palate was involved. The authors do not mention, nor does the picture suggest that the mass originated from the gingiva. In fact it would appear from the picture and the text that the mass originated from the palatal mucosae. An epulis can only arise on the gingiva and more specific at the gingival margin, because it is believed to be of periodontal origin[3 10 15]. Therefore it would seem highly unlikely that the tumour presented here was in fact an epulis. The histological findings included osteoid formation, epithelial rests, round cell infiltration and granulation tissue. None of these findings is specific or indicative for a neoplasm. The main characteristic of an epulis is its dense cellular stroma consisting of stellate cells and collagen[3].

The clinical staging of neoplastic conditions is of prime importance[16]. The TNM classification, based on tumour characteristics, lymphnode involvement and metastasis, is accepted world-wide and used in the literature. This is of academic interest for the correct interpretation of treatment results and the exchange of information. More important, however, is that it is beneficial to the patient, as it forces the clinician to do a complete clinical examination and aids in choosing the correct method of treatment.

A tumour involving the palate is an excellent example of a case where a complete clinical work-up is absolutely necessary, before any method of treatment is even considered. It should be determined how deeply the tumour infiltrates, and if so, if the underlying bone is affected. In fact it is not uncommon to find a nasal tumour eroding the palate and presenting as an oral tumour. An intra-oral radiograph is indicated. If the bony palate is completely eroded, drastic surgery, cryosurgery or irradiation is likely to result in an iatrogenic oronasal fistula. This complication is disastrous because it is extremely difficult or even impossible to correct.

Irradiation is an accepted method of treatment for acanthomatous epulides and its use has been well described by Thrall[19], based on a large number of cases. The scientific value of reporting on one isolated case is questionable. Complications have also been described and should have been mentioned by the authors in their discussion. Malignant tumour formation at the site of previously irradiated acanthomatous epulides has been recorded[18]. Osteo-radionecrosis is another serious complication that may be progressive and may result in an oronasal fistula[17 18].

In the discussion the authors mention adamantinoma as a possible differential diagnosis. It should be noted, however, that the term "adamantinoma" is not accepted and is replaced by "ameloblastoma"[5 13], since the introduction of the WHO classification of odontogenic tumours, jaw cysts and allied lesions in 1971[13]. It is interesting that this classification is largely based on the publications of the same Robert J. Gorlin[6 7 8 9], referred to indirectly by Dr Odendaal and Dr Cronje.

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LETTER TO THE EDITOR — AAN DIE REDAKSIE

OOPGESPREK OOR KWAADAARDIGE EPULIS

Baie dankie vir Dr Verstraete se menings oor "Die behandeling van kwaadaardige epulis in die hond". Een van die doelstellings van 'n publikasie is om deur middel van hierdie wetenskaplike gesprek te kommunikeer, en daarom is dit soms meer frustrerend om geen reaksie, as om kritiek te ontvang. Ongelukkig publiseer baie wetenskaplikes nie, omdat hulle die kritiek vrees en só gaan baie waardevolle inligting verlore.

Ons antwoord op die gesprek puntsgewys, en in die volgorde van Dr Verstraete se brief:

1. Die titel suggereer nie dat 'n aansienlike aantal gevalle behandel is nie. "Die hond" dui op die spesie en is verkies bo "'n hond" omdat die patoloog die outeurs verseker het dat die diagnose buitengewoon is vir die spesie.
2. Oor die kwessie van uit watter selle die epulis nóu presies sou ontstaan, is dit Moulton versus Bhaskor. Dr Verstraete se stelling dat 'n epulis hoofsaaklik uit bindweefsel bestaan en wat hy met drie aanhalings staaf, is ooreenstemmend met wat in die artikel "fibromateuse epulis" genoem word. Daar bestaan dus geen teenstrydigheid in die verband nie.
3. Omdat die ware aard van 'n epulis 'n omstrede saak is, is 'n artikel soos hierdie van groot waarde, want dit sit die gesprek rondom die onderwerp voort. Daar staan dan ook in die artikel "die oorsaak van gewasse van die mondholte is nog grootliks onbekend". Ons verskeie differensiële diagnoses bevestig ook hierdie feit. Een van die oorwegings om die artikel te plaas was juis die "omstrede" diagnose.

   Dr Verstraete sê dat sommige patoloë meen dat 'n epulis slegs "hiperplasties van aard" is, maar dit is presies wat in die artikel aangehaal word.

   Wat die kwaadaardigheid van die epulis betref, wil ons graag die volgende inligting verskaf:

   (i) Die diagnose is deur 'n spesialis patoloog gedoen wat haar toelê op diagnose van gewasse. Sy was oortuig dat hierdie 'n besondere diag-

nose was en dít alleen was genoeg rede vir ons as klinici om dit so te aanvaar. Ons het egter, weens die omstredenheid van die diagnose, 'n tweede spesialis patoloog gekonsulteer en hy het ons verseker dat hy die saak met die eerste patoloog sou bespreek omdat hulle in dieselfde Departement werksaam is. Ons het geen kritiek oor die diagnose terug ontvang nie. Nadat die publikasie vir 'n lang tyd agterweë gehou is, is uiteindelik besluit dat die artikel 'n bydrae kan lewer tot die gesprek rondom epulis. Hierin het ons met Dr Verstraete se reaksie geslaag.

(ii) Die patoloog het die outeurs, selfs per brief, verseker dat sy 'n artikel gaan publiseer wat net oor die patologie van hierdie diagnose handel. Dit het ons aangemoedig om die kliniese resultate te publiseer omdat die artikel oor die patologie die omstredenheid van die diagnose sou opklaar. Die beloofde artikel het egter nog nie verskyn nie.

(iii) Ons het self in die artikel gemeld dat "epulis gewoonlik 'n goedaardige groeisel is". Die "gewoonlik" laat die moontlikheid oop vir die diagnose wat ons van die patoloog ontvang het.

(iv) Dr Verstraete sal saamstem, dat al is daar nie ondersteuning in die literatuur vir die patologiese diagnose nie, daar altyd 'n moontlikheid is van 'n eerste keer.

4. "Mondslymvlies" was 'n ongelukkige vertaling van "gingiva" en moes gelees het "tandvleis". Daar word egter duidelik in die artikel gemeld dat "die letsel *vanaf* die pre- en molare tande gestrek" het. Verder kan die tandvleis tog ook as 'n spesifieke deel van die totale mondslymvlies beskou word.
5. Ons kon geen belangrike en/of subtiele verskil opmerk tussen 'n "vermoede" en "wat nog wetenskaplik ondersoek moet word" nie. Ons kon ook nie die doel van Dr Verstraete se kritiek in die verband snap nie.
6. Ons stem saam dat die term "rostraal" onvanpas is, maar die term is deur die beoordeelaar van die artikel aangebring. Die foto toon 'n pigmentlose litteken, ná genesing. Dit dui dus slegs op die finale resultaat. Die foto toon nie die oorspronklike groeisel wat wel vanaf die tandvleis ontwikkel het nie. Ons het reeds gemeld dat die letsel vanaf die tande gestrek het. Die res van Dr Verstraete se afleidings oor die ligging van die epulis is dus nie van toepassing nie. Dr Verstraete haal bevindings van die histopatologiese verslag aan en sê dan dat dit nie op 'n gewas dui nie. Hy laat egter die deel uit wat meld dat die weefsels "neoplasties" was.
7. Ons is ten volle op hoogte van die TNM-klassifikasie van gewasse en al ons gevalle, insluitend die epulis, is volgens hierdie klassifikasie in ons rekenaar aangeteken. In die artikel is die T volledig beskrywe en N en M was 0. Dr Verstraete meen dat die weglating van die letters TNM slegs van akademiese belang is en ons stem daarmee saam. Die voordeel wat die pasiënt uit so 'n ondersoek en klassifikasie kon haal, het sy inderdaad gekry.
8. Dr Verstraete meen dat 'n volledige kliniese ondersoek nodig is, voordat enige behandeling oorweeg kan word. Hierdie stelling wil ons graag ondersteun, maar vir ons as klinici is dit 'n logiese en nie 'n spesiale voorvereiste nie. Die suggestie dat so 'n ondersoek nie gedoen is nie en die spekulasie wat daarop volg, is onaanvaarbaar. Die suggestie en spekulasie is wel moontlik as 'n mens die geval op papier (die artikel) beoordeel, maar Dr Verstraete se vrese kan dadelik uit die weg geruim gewees het, indien hy by die geval betrokke was. Juis *as gevolg van* 'n behoorlik kliniese ondersoek en nie weens 'n gebrek daaraan nie, was dit op daardie stadium duidelik dat daar nie 'n aanduiding was vir die neem van 'n X-straal nie. Die letsel se aard en diepte is onder algemene narkose tydens die chirurgie behoorlik ondersoek en verder was daar geen aanduiding dat daar enige groeisel of obstruksie in die neusgange voorgekom het nie. Die resultate het bewys dat ons kliniese ondersoek ons die korrekte inligting verskaf het en dat ons die kliënt die koste van 'n onnodige X-straal gespaar het. Geen komplikasie het plaasgevind nie.
9. Die verwysing na die bestralings wat deur Thrall gedoen is, is interessant, maar dit gaan oor 'n ander diagnose as in ons artikel. Indien Dr Verstraete nie die diagnose aanvaar nie, is ons as klinici ongelukkig nie in die posisie om iets daaraan te doen nie. Ons het reeds die gebruik van die diagnose in punt 3 verdedig.

   Die feit dat ons met ons radioterapie geen komplikasies ondervind het nie, verhoog die waarde van die artikel. Sulke inligting kan vir ander klinici van waarde wees.
10. Die dispuut oor die terme "adamantinoma" en "ameloblast" bly na alles gesê is, slegs "interessant". Die term "ameloblast" wat in 1971 (volgens Moulton reeds in 1961) deur Gorlin voorgestel is, word deur Moulton as alternatiewe naam gebruik en wel as "ameloblastoma". Moulton[1] se publikasie het in 1979 verskyn en hy verskaf selfs 'n derde alternatiewe naam "enamelblastoma"! Dit is duidelik dat wetenskaplikes altyd oor die absoluut korrekte benamings sal stry. Ons meen dat dit 'n futiele oefening is om in 'n artikel soos hierdie te diep op hierdie sake in te gaan.

Om op te som lyk dit of Dr Verstraete veral probleme het met die aanhalings uit die publikasies, met spekulasies, asook die spesifieke diagnose. As hy egter die artikel konsekwent wou beoordeel, sal hy moet saamstem dat ten spyte van die gesprek wat gevoer is, die resultate van die geval uiters gunstig was. Die geval wou nie op ander behandeling, behalwe die radioterapie reageer nie, en vir ons as klinici is dit belangrik dat ons 'n behandeling gevind het wat genesing tot gevolg het. Die pasiënt geniet nou op 15 jaar, 3 jaar na die behandeling, nog goeie gesondheid en is 'n lewende voorbeeld van die sukses wat behaal is.

Retrospektief meen ons, het die artikel sy doel bereik en ons glo belangrike inligting is weergegee. Dit vorm die stimulus vir verdere publikasies.

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TO THE EDITOR — AAN DIE REDAKSIE

A SOURCE OF VETERINARY INFORMATION

In 1983 I conducted a pilot survey for Feline Leukaemia Virus (FeLV) infections and conditions often associated with such infections in the Western Cape and Johannesburg. This was done by sending a questionnaire to several private practitioners and a private veterinary laboratory which renders services to a large number of veterinary practices.

The findings *per se* of the survey were presented at the Congress of the SAVA held in Bloemfontein in September 1983 in a paper titled 'Feline Leukaemia/Lymphosarcoma and Feline AIDS in South Africa'.

Here, based upon the broad experience gained by the exercise, I wish to discuss a few points relating to the availability of information and the possibility of increasing veterinary knowledge.

In the first instance, the survey showed that useful information concerning FeLV infections can be extracted from existing records of private veterinary practices and laboratories. However, it yielded less information than expected. In part this was due to difficulty experienced by some veterinarians in extracting specific information relating to FeLV infections from their general case history records. I believe this problem largely could be offset by properly cross-referencing general records or by keeping a separate register for FeLV infections and associated conditions. A uniform system of record keeping would have the added advantage of ease of processing data from various practices. Clearly the more accurate the records the more reliable the information extracted. Thus in future surveys as far as possible predetermined evaluation criteria should be used. While I am not in a position to continue the work it is hoped that someone else might be and for which purpose I would gladly make the original questionnaire available.

Finally, it is suggested that the approach of tapping information available at private veterinary practices and laboratories could be useful in expanding knowledge of many other veterinary problems. In this context, it should be remembered that, unlike the situation in human medicine, in veterinary medicine there generally are no large hospitals run by the state, provincial or local authorities keeping extensive records. Therefore it is particularly important that we look to private veterinary practices and laboratories for information.

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BOOK REVIEW — BOEKRESENSIE

STRONGYLUS VULGARIS IN THE HORSE: ITS BIOLOGY AND VETERINARY IMPORTANCE

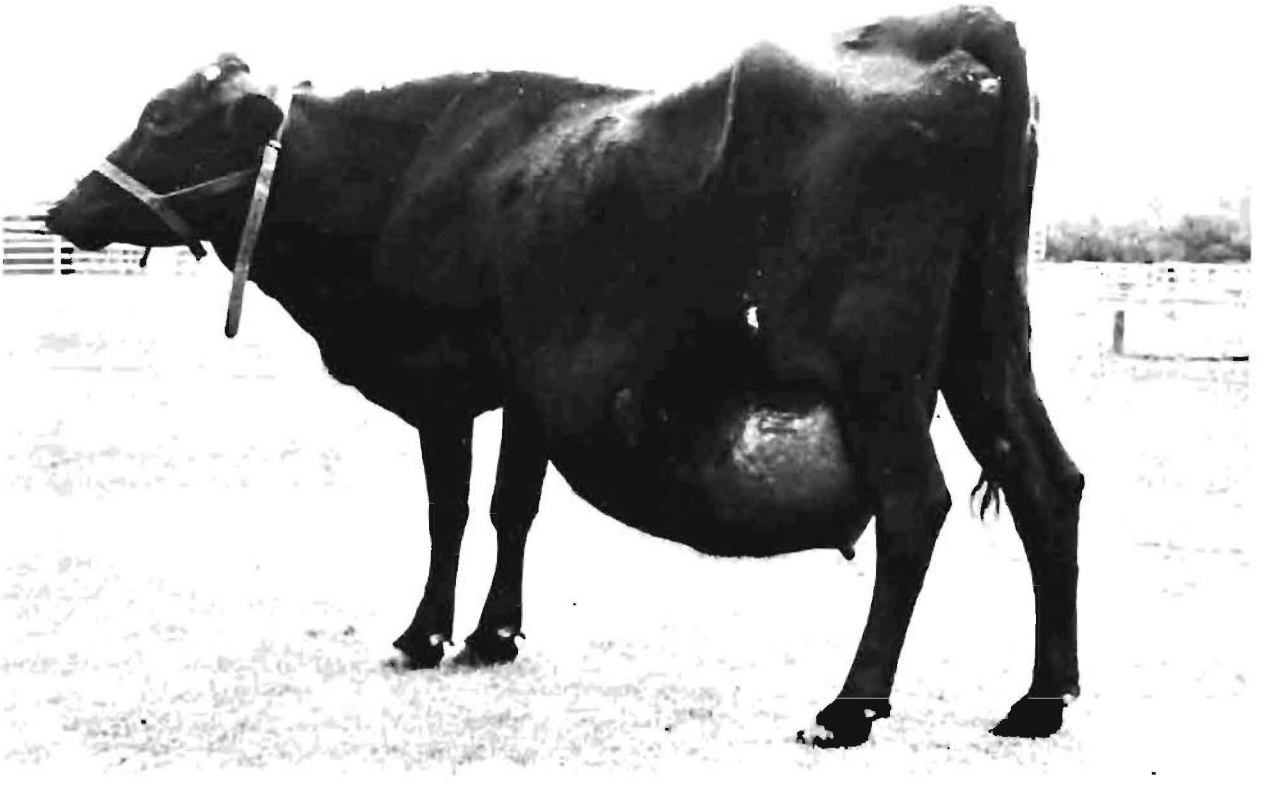
C.P. OGBOURNE and J.L. DUNCAN

2nd Edn. Miscellaneous Publication No. 9 of the Commonwealth Institute of Parasitology, 395 A Hatfield Rd, St. Albans, Herts. AL4 OXU, UK. 1985 pp v and 68, Figs 26, Tables 2, Refs 359, Price US $25,65 (ISBN 0 85198 547 5)

This publication comprehensively reviews current knowledge on *Strongylus vulgaris*, the highly pathogenic nematode parasite of equines. Aspects reviewed are distribution, the free-living phase of the life-cycle, pathogenesis and pathology, immunology, diagnosis, epidemiology, control and treatment.

All parasitologists, equine practitioners, veterinary students and stud managers will benefit from this excellent publication which is richly illustrated, systematic in its approach, practical and easy to follow.

F.S. Malan



HYDRALLANTOIS IN A BOVINE LEADING TO RUPTURE OF THE PREPUBIC TENDON AND ABDOMINAL MUSCULATURE

A gravid Afrikaner x Friesland cow was presented 5 days after the owner had noticed swelling of the abdomen. Hydrallantois was diagnosed. A trocar and cannula were inserted and 20 ℓ of a watery fluid were drained. By the next day the cow was in distress and a caesarian section was undertaken. Hydrallantois was confirmed and extensive adventitious placentation was observed. The prepubic tendon had ruptured, and there was extensive rupturing of the abdominal musculature. The cow was delivered of a 16 kg bull calf, which died virtually immediately. A post-mortem examination revealed neonatal atelectasis and pronounced pulmonary oedema.

Post-operative treatment of the cow consisted of large volume fluid therapy and antibiotic cover. The cow died 2 days later. Acute fibrinous peritonitis had developed and an aetiological diagnosis of hypovalaemic/septic shock was made.

We thank Prof. N.P.J. Kriek and Dr J.W. Nesbit for performing the post-mortem examinations.

Submitted by: B.L. Penzhorn and R.O. Gilbert, Department of Genesiology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, and J. van Heerden, Department of Medicine, Faculty of Veterinary Science, Medical University of Southern Africa, P.O. Medunsa, 0204 Medunsa.

HYDRALLANTOIS IN 'N KOEI, WAT LEI TOT SKEURING VAN DIE PREPUBIESE PEES EN BUIKSPIERE

'n Dragtige Afrikaner x Frieskoei is 5 dae nadat die eienaar opgemerk het dat haar buik begin opswel, aangebied. Hydrallantois is gediagnoseer en 20 ℓ waterige vloeistof is met behulp van 'n trokar en kannula afgetap. Teen die volgende dag het die koei in nood verkeer en is 'n keisersnit uitgevoer. Hydrallantois is bevestig en uitgebreide bykomstige plasentasie is waargeneem. Die prepubiese pees was geskeur, terwyl van die buikspiere uitmekaar geskeur was. 'n Bulkalf van 16 kg is verwyder, maar hy het feitlik onmiddellik gevrek. 'n Nadoodse ondersoek het neonatale atelektase en uitgesproke longedeem getoon.

Die na-operatiewe sorg van die koei het uit vloeistofterapie ('n groot volume, en antibiotiese dekking bestaan. Sy het 2 dae later gevrek. Akute fibrineuse peritonitis het ontstaan en 'n etiologiese diagnose van hipovolemiese/septiese skok is gemaak.

Ons bedank Prof. N.P.J. Kriek en Dr. J.W. Nesbit wat die nadoodse ondersoeke uitgevoer het.

Ingestuur deur: B.L. Penzhorn en R.O. Gilbert, Departement Geslagskunde, Fakulteit Veeartsenykunde, Universiteit van Pretoria, Posbus 12580, 0110 Onderstepoort; en J. van Heerden, Departement Geneeskunde, Fakulteit Veeartsenykunde, Mediese Universiteit van Suider Afrika, Pk. Medunsa, 0204 Medunsa.

ABSTRACTS

ABSTRACT: Reinecke, R.K., De Villiers, I.L., Lombard, Magdalena S. & Scialdo-Krecek, Rosina C., 1984. **Studies on *Haemonchus contortus*. XII. Effect of *Trichostrongylus axei* in Dorper lambs on natural pasture lightly infested with *H. contortus*.** *Onderstepoort Journal of Veterinary Research*, **51**, 81-88 (1984).

Weaned Dorper lambs on natural pasture were predosed with 40 000 infective larvae (L_3) of *Trichostrongylus axei*, irradiated (0,3 kGy) L_3 of *T. axei* or closantel at 10 mg/kg either in September or November 1978 and were compared with Merino yearlings predosed with 40 000 L_3 of *T. axei* in November 1977. The following summer (December-March) only 178,6 mm of rain fell and very few *H. contortus* were present on pastures. Artificial challenge with 20 000 L_3 of *Haemonchus contortus* with the local strain from the University of Pretoria's experimental farm was given 6-7 months after predosing with *T. axei*. When compared with the controls, significant reductions occurred only in Group 11 (*T. axei* irradiated at 0,3 kGy on Day +63) ($P = 0,025$); Group 2 (*T. axei* on Day 0) ($P = 0,003$) and Group 4 (*T. axei* and closantel on Day 0) ($P = 0,049$). We concluded that predosing with *T. axei* was unsuccessful in Dorpers and Merinos artificially challenged 6-7 months later with *H. contortus*.

ABSTRACT: Reinecke, R.K., De Villiers, I.L. & Joubert, Gerda, 1984. **Studies on *Haemonchus contortus*. XI. The effect of a bovine strain of *Trichostrongylus axei* in Merinos on natural pastures heavily infested with *H. contortus*.** *Onderstepoort Journal of Veterinary Research*, **51**, 71-79 (1984).

Sheep grazed on natural pastures heavily infested with infective larvae of *Haemonchus contortus*. Sixty-eight weaned Merinos were divided into 6 groups on Day 0 (23 November 1977), and on Day +14 (7 December) 79 Merinos were divided into 7 groups. There were 2 groups of undosed controls and other groups were either dosed with infective larvae of *Trichostrongylus axei* (bovine strain) only on Days 0 and +14, or in combination with *H. contortus*, or with subsequent doses of *H. contortus*, 28 days later. One group (Group 12) was dosed with *T. axei* and treated with a subcutaneous injection of di-iodonitrophenol (DNP) on Day +14. With the exception of 2 sheep, the sheep of the first 6 groups survived until slaughter in March and April 1978, while many sheep (43) of the latter 7 groups died or were killed *in extremis* from March-May. *T. axei* dosed on 23 November (Day 0) protected Group 2 by >50 % in >50 % of sheep. In the latter 7 groups the best results were achieved when DNP was combined with predosing with *T. axei*. The poor results were probably due either to delayed predosing with *T. axei* or a massive challenge in the wettest summer on record.

ABSTRACT: Giesecke, W.H., Spickett, A.M., Durand, Anette M., Van Staden, J.J. & Erasmus, J.A., 1984. **Electron microscopic observations on the luminal surface of teat cup liners of milking machines used under South African conditions.** *Onderstepoort Journal of Veterinary Research*, **51**, 47-70 (1984).

An investigation undertaken with the aid of scanning electron microscopy (SEM) on new and used teat cup liners revealed the generally poor quality of luminal surfaces. Even those of the brand-new distributor controls showed significant faults in the evenness and continuity of the liner surface. A hundred and 500 milkings apparently tend to aggravate faults like cracks, pores, grooves and pits, because of the general brittleness of some of the teat cup liners.

The poor quality of the liners investigated raises various questions about the method of distribution of teat cup liners and serious concern about the role they play in predisposing bovine udders to mastitogenic infections, spreading mastitis, affecting the production and quality of milk, increasing the cost of milk production and reducing profits of dairy farming.

ABSTRACT: Swanepoel, Martha L., 1984. **A study for the differentiation of *Actinobacillus seminis*, *A. actinomycetem-comitans*, *Histophilus ovis* and *Pasteurella haemolytica*.** *Onderstepoort Journal of Veterinary Research*, **51**, 41-46 (1984).

By using well-defined techniques under optimum conditions it is possible adequately to define the biochemical characteristics of typical *A. seminis* strains. *A. seminis* can be distinguished from *Histophilus ovis* on the latter's distinctive colony morphology, but it cannot be distinguished from *Actinobacillus actinomycetem-comitans*. These organisms, however, can be differentiated from *Pasteurella haemolytica* on serological grounds and the latter's greater pathogenicity for mice. It is appreciated, however, that intermediate forms occur which cannot as yet be satisfactorily allocated to any of the above-mentioned genera.

ABSTRACT: Boomker, J. & Kingsley, Shirley A., 1984. ***Paracooperia devossi* n. sp. (Nematoda: Trichostrongylidae) from the bushbuck, *Tragelaphus scriptus* (Pallas, 1766).** *Onderstepoort Journal of Veterinary Research*, **51**, 21-24 (1984).

A new species of *Paracooperia* Travassos, 1935 was found in the small intestines of 4 bushbuck, *Tragelaphus scriptus* (Pallas, 1766), and 3 greater kudu, *Tragelaphus strepsiceros* (Pallas, 1766), all culled in the Kruger National Park. The nematodes appear to be widespread in the Park, since the buchbuck originated from Skukuza in the central part of the Park, 2 of the 3 kudu from near Malelane in the south and the remaining kudu from Pafuri in the extreme north.

The worms are named *Paracooperia devossi* after Dr V. De Vos of the Kruger National Park and can be differentiated from the closely related *Paracooperia tragelaphi* Gibbons & Khalil, 1980 by the single indistinct ridge on 1 of the branches of the spicules. Furthermore, the spicules end as fairly large ovoid knobs, and vulvar flaps are present in the females.

THE BIRTH OF OUR JOURNAL

R.C. TUSTIN*

At a General Meeting of the South African Veterinary Medical Association held at the Union Buildings, Pretoria on 30 September 1926 and continued on the following day at the Government Laboratory, Onderstepoort, part of the agenda, as had become the custom at such meetings, was devoted to the presentation of papers or discussions on the following subjects: "Poisonous Plants" by Dr H.H. Curson, "Scab in Sheep and Goats in Basutoland" by Major F.A. Verney, "Animal Nutrition" by Dr W.J.B. Green, "Parabotulinus" by Dr E.M. Robinson, "Vaccines" by Mr J.C. Chalmers and "Sterility" by Dr J.B. Quinlan. At the end of the proceedings, the Vice President, Dr G. v.d. W. de Kock who was acting as chairman of the meeting in the absence of the President, Dr P.J. du Toit thanked the speakers for coming forward with such interesting papers of practical importance and expressed the hope that other members would follow their example, which gave food for such interesting discussion, and bring forward papers based on practical experience and field observation. It was then resolved by those present that the papers presented should be circulated to members of the Association in full and that as much as possible of the discussion be added.

It was this resolution which probably focussed attention on the possibility and desirability of actually producing and publishing a journal of our own, for it is in the minutes of the Annual General Meeting held on 15 April 1927 under the chairmanship of the President that Dr de Kock reported "that he and the Secretary (Mr A.C. Kirkpatrick) had taken it upon themselves to have the Papers (read at the last meeting) printed and circulated to all Members. This was in accordance with a resolution of the Association. They got an estimate of the cost of both printing and typing and eventually made arrangements to have them printed. He said he would like to go a step further, and that was the step suggested by Major Verney, 'that we run a Journal of our own' say with one publication a year, which would include Minutes, Papers, etc. and he wished to put forward the following proposal:

> 'That a small Committee be appointed consisting of say, the Secretary and another, to go into the question of suitability of publishing our own Journal, the sum that would be involved and how the subject matter would be arranged.'

"The Secretary . . . (stated that) the sum was about £45 for the printing of the papers (for circulation to all Members but) that while they had to pay £45 for 200 copies, they could get 1 000 copies for very little more." Mr Chalmers suggested that it might be possible to obtain a subsidy from the Government to publish an annual journal as the expenses involved might be too great for the Association to bear.

While it may indeed have been the above resolution that germinated the seed of the idea of printing and publishing our own Journal, the actual sowing of the seed may have been at the General Meeting held in Pretoria during September 1924 when a letter from Mr R. Payne was read to those present. Mr Payne complained about the "scanty and rather belated reports of the proceedings (of meetings) received by Members who were unable to attend meetings and suggested the engaging of a qualified reporter to take down a verbatim report of the proceedings and discussions, which could then be circulated with the papers read." This was thereafter done to some extent but Mr Payne was still dissatisfied with the state of affairs 3 years later as in another letter read to the meeting of 15 April 1927 while the desirability or otherwise of a journal was being debated, he again complained about the paucity of and delays in the circulation of reports and minutes of meetings. The outcome of the deliberations at the latter meeting was that it was agreed, following a suggestion of the President, "that a Committee consisting of Messrs G. v.d. W. de Kock, A. Goodall and the Secretary (A.C. Kirkpatrick) go into the question of the annual publication of a Journal containing Papers, Minutes, activities of Members, etc."

At a subsequent General Meeting held on 18 August 1927, after the President had explained what steps had been taken by Council in connection with the feasibility of publishing a journal, it was unaminously agreed by those present that the Association should have its own journal and that a Journal Committee consisting of Messers de Kock, Goodall and the Secretary with the addition of the President be authorised to proceed with the publication of the journal.

Thus it came about that Vol. 1 No. 1 appeared in 1927, Vol. 1 No. 2 in 1928, Vol. 1 No. 3 in 1929, Vol. 1 No. 4 in 1930, Vol. 2 No. 1 in 1931 and Vol. 2 No. 2 in 1932, and since 1933 a separate volume has been published in each subsequent year.

The aims and objects of the Association's journal and many of the sentiments expressed in the first editorial of Vol. 1 No. 1 still stand today 58 years later. This editorial reads as follows:

"The first number of the Journal of the South African Veterinary Medical Association has made its appearance. The amalgamation of the Natal, Cape Colony and Transvaal Veterinary Medical Associations, each with a small quota of members, took place in 1920, and one association was formed for the Union of South Africa. Through the united efforts of its members, this body has gained considerable importance, and it has undoubtedly contributed much to the consolidation of the veterinary profession in the Union. It has taken every opportunity to further the interest and status of the veterinarians, and it is hoped that the Bill championed

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by this Association, will within the next year or so become an Act of Parliament. The Association has, with a certain amount of success, interested itself in various appointments pertaining to the status of the profession. In this respect, it is bound to play a much bigger role, especially as soon as the profession enjoys full protection.

"For several years important papers have been read at the annual conferences of the Association, and it was felt that these papers presented valuable material for a journal. At present this Journal will be published annually, but it is hoped that in time the Association will be able to issue several numbers annually. The establishment of a periodical for the veterinary profession has been urged from various sources for many years with increasing insistence. Up to the present, the results of veterinary research and progress have been incorporated in departmental reports issued by the Union Government. The long delay in the publication of important results contained in these reports was a distinct drawback, especially to those members of the profession stationed in the country districts. The present Journal will overcome some of these difficulties and give increased facilities for the publication of results of scientific work. It will serve as a medium for information of the progress of research and promote the spread of scientific knowledge as the basis for rational and successful methods of prevention and treatment.

"The Journal will contain articles on the results of new work and experience in all departments of veterinary science, general views on subjects of special interest, reports of meetings of the Association, and notes and news of personal and general interest.

"The veterinary profession in the Union feels that the present Journal is to a large extent the outcome of the pioneer work in veterinary science commenced by the late Dr. Hutcheon and carried on by such ardent workers as Sir Arnold Theiler, Mr. Gray, Mr. Borthwick, and others. The Association realises how much it is indebted to these gentlemen who have rendered the profession in the Union a very great service in placing it on a sound footing. The brilliant research work conducted by Sir Arnold Theiler has opened up a very wide field. Great opportunities for research in veterinary science are offered and it is hoped that the result of this work will form the basis of many important articles for the Journal of the South African Veterinary Medical Association."

DR JOHN HUXLEY MASON 1899 – 1985

Dr John Huxley Mason – one of the best known and most respected members of the staff of the South African Institute for Medical Research and past-president of the South African Veterinary Association – passed away in the early hours of the morning of Monday 28th January, 1985 at the age of 85 years, a few days after a major operation for the repair of a large blood vessel.

Dr Mason was born in Glasgow on 3rd October, 1899. He was proud of being a Scotsman and of his Scottish heritage. After schooling in Hutcheon's Grammer School he took the veterinary course at the Glasgow Veterinary College of the University of Glasgow and was awarded his degree of MRCVS in 1920.

After three years in private practice as an assistant to various practitioners in London, Darlington and Wigan – about which he told many an intriguing story – he took a postgraduate course in bacteriology and was then in 1922 appointed Lecturer in Bacteriology in Glasgow Veterinary College. In 1924 he joined the staff of the Wellcome Research Laboratories at Beckenham in Kent as a Veterinary Research Scientist. Here he was one of the pioneers, with other wellknown bacteriologists Dalling, Glenny and Mollie Barr, in the study of the Clostridia. In these studies he made several important contributions to the understanding of the action of their toxins and of the immunity against them.

In 1927 he obtained the Fellowship of the Royal College of Veterinary Surgeons and in 1928 he was elected a Fellow of the Royal Society of Edinburgh.

In 1931 Dr Mason was appointed an Empire Marketing Board Research Fellow at the Onderstepoort Veterinary Research Institute where he made a deep and lasting impression. This appointment set the pattern for the rest of his life.

He continued with his studies of anaerobic organisms but extended his interests into other fields in each of which he made original contributions to our knowledge. With Dr Ray Alexander he studied heartwater and blue tongue in sheep and then they turned their attention to the rickettsial diseases of man – epidemic typhus fever, murine typhus and tick bite fever. Working in collaboration with the team at the S.A. Institute for Medical Research, it was shown that the rickettsiae causing these diseases grew prolifically in the tissues of the developing chicken embryo. The large scale production of typhus vaccine was based on this finding. Several million doses were issues to protect the population of the Transkei, the prisoners of war in Eastern Europe and the soldiers of the Union Defence Force.

In 1941, during World War 2, Dr Mason transferred from Onderstepoort to the S.A. Institute for Medical Research to take charge of the Anaerobic Bacteria Laboratories, a post for which he was uniquely qualified. These laboratories had been recently completed to meet the needs of the Union Defence Force. The Director of Onderstepoort, Dr P.J. du Toit was reluctant to allow his transfer but clearly it was in the interest of the country at that time and he finally agreed. Dr Mason not only directed the work of important laboratories but as a well qualified veterinarian he took charge of several hundred horses being used for the production of anti-gasgangrene serum, snake bite antivenom, antidiphtheria serum and a number of other therapeutic sera. He also took over the supervision of all the other laboratory animals and their accommodation. His keen interest in the welfare of these animals was immediately evident and they were better cared for than ever before. The Institute was able to meet all its responsibilities in the supply of vaccines and therapeutic sera to the Union Defence Force and to the Health Department and other health authorities not only of South Africa but of most of the countries of Africa south of the Sahara.

When Dr E. Grasset resigned from the Institute in 1945 to take up an appointment in Geneva, Dr Mason was appointed to succeed him as Superintendent of the Serum and Vaccine Division, an office he filled with great distinction. He continued with his research studies directed to the development and improvement of vaccines especially the vaccines against diphtheria, whooping cough and tetanus. Under his supervision the serum and vaccine laboratories were able to meet the needs when mass vaccination of the infant population of South Africa was undertaken in the late 1950's and early 1960's. As a result these diseases are now rarely seen in South Africa.

In 1960 he was appointed Deputy-Director of the Institute, a post he also filled with great distinction. He continued to direct the work of the Serum and Vaccine Division and to take a part in the wider sphere of the activities of the Institute as a whole.

In describing his work at Onderstepoort and at the Institute, he published as author and co-author over 110 scientific papers many making important original contributions to our knowledge. In addition to his own scientific writings Dr Mason was most helpful to other members of the staff in editing their papers, a help which many found invaluable.

He was President of the South African Veterinary Association for 4 years and was elected an Honorary Life Vice-President. He was a member of the Veterinary Research Club and several other scientific societies.

In September 1976 at the Annual Meeting in Bloemfontein, the President of the South African Veterinary Association Dr B.H. Pappin presented the Association's Gold Medal – its premier award – to John Huxley Mason for distinguished service to the Veterinary

profession.

He was dedicated to his work and most meticulous in all he did and expected similar dedication from all who worked with him and he was highly critical of slovenly work.

His personal relationship with members of the staff was excellent. Indeed all his staff regarded him with respect, admiration and affection, an appreciation fully earned by his never failing concern and consideration for their welfare. Many members of staff are eternally grateful for his help with their personal problems. I believe he regarded them as his children; certainly he looked after them as a father would look after his own children.

He will be sorely missed by his many friends, but at this time perhaps our greatest feeling should be one of gratitude for the privilege of the princely gift of his friendship and for his long and rewarding life – a life that was rewarding for the Institutes for which he worked, rewarding for the community in which he lived and rewarding for his adopted country South Africa.

J.H.S. Gear

BOOK REVIEW BOEKRESENSIE

PARASITOLOGY FOR VETERINARIANS

JAY R. GEORGI

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This book, like the previous edition, is divided into 3 parts. The first part provides brief descriptions of the parasitic insects, arachnids, trematodes, cestodes, nematodes and protozoa. Part 2 deals with the treatment and control of clinical parasitism in the different domestic animals and also contains a chapter on antiparasitic drugs, written by Vassillios J. Theodorides. Diagnostic parasitology is dealt with in Part 3, and this is subdivided into ante mortem, post mortem and histopathological diagnoses.

The book is definitely an improvement on the 3rd edition and is better organised. Several new photographs have been added to an already beautifully illustrated book, and the equally well illustrated chapter on histopathological diagnoses is a most welcome addition. Due to better space utilization, the 4th edition has 116 fewer pages despite the addition of a new chapter and photographs.

Some criticisms, however, are firstly, that several of the illustrations are not found near the description of the parasite, e.g. the larva of *Cuterebra* is illustrated on p. 13, while the parasite is described on p. 22 and the adult fly illustrated on p. 23. Secondly, the life cycles of the more important parasites are illustrated in the second part of the book, whereas they should accompany the descriptions and thirdly, little of the epidemiology of the acarids and nematodes is provided. There are mis-spelled scientific names e.g. *Schistosoma mattiei* instead of *S. mattheei*. Fourthly, the generic names *Taeniarhynchus*, *Hydatigena* and *Multiceps* for the adults of *Taenia saginata*, *Taenia taeniaeformis* and *Taenia multiceps* are unacceptable since they refer to the larval stages.

The book is clearly aimed at the North American market and the parasites that are important in the tropics and subtropics are poorly discussed. The cosmopolitan insects, acarids and helminths are dealt with in sufficient, but not superfluous, detail to enable undergraduate students to grasp the basics. However, at R84,55 it is not the kind of book one would prescribe.

The book should be available in libraries as a reference work and students at all levels, as well as diagnostic clinicians should find it a valuable aid. Pathologists should find the well-illustrated section on histopathology very useful.

J. Boomker