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TYDSKRIF VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING

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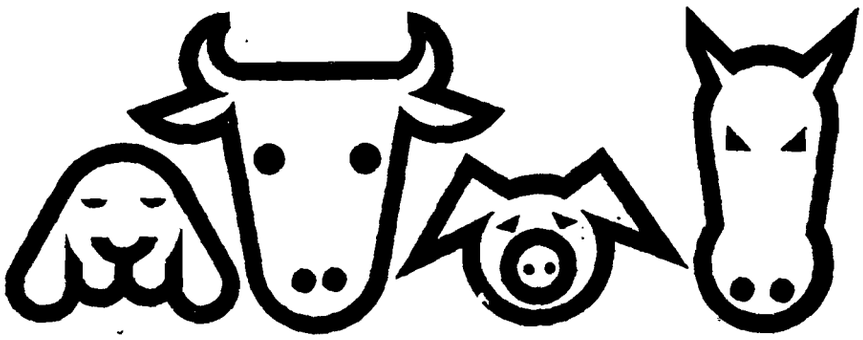

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ERASMUS J.A. and FAANHOF A. 1983. Blood selenium of sheep in some districts of the Northern Orange Free State. Journal of the South African Veterinary Association 54 (3): 187-188.

On page 188 of the article the normal levels of the selenium concentration in the blood should read 0,1 – 0,2 μg/ml (and not 1,0 – 2,0 μg/ml), and on a ration with a selenium content of about 0,33 μg/g the blood selenium concentration of cattle varied between 0,233 and 0,323 μg/ml (and not 0,833 and 0,323 μg/ml).

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EDITORIAL **VAN DIE REDAKSIE**

EXTENSION OF VETERINARY INVOLVEMENT IN MEAT HYGIENE CONTROL

In Notice 506 of 1983, appearing in the Government Gazette of 8 July 1983, the Chief Meat Hygiene Officer has taken steps which will have a profound effect on the role of the veterinarian in ensuring a safe, sound and wholesome meat supply and in promoting animal health in South Africa when they come into effect on the 1st of January 1984.

The Animal Slaughter, Meat and Animal Products Hygiene Act No. 87 of 1967 and its standing Regulations came into effect in October 1969. At that time there were about 1400 abattoirs in the Republic, many of them old and uneconomical and in which the basic principles of meat hygiene were or could not be implemented. In many, animals and meat were not subjected to inspection and it was therefore necessary to exempt all but the 17 larger abattoirs from those sections (25-29) of the Act which required a specific form of inspection. This allowed those local authorities who could do so to continue to inspect meat in terms of regulations framed under the old Public Health Act of 1919.

Now, some 14 years later, there are only 337 registered and approved red meat abattoirs in South Africa and about 99% of the meat emanating from these abattoirs is being subjected to some form of inspection. Of these abattoirs, 10 are owned and operated by the Abattoir Corporation, 200 by municipalities and 127 by private owners. Graded according to capacity and meat distribution, there are 26A, 19B, 33C, 82D and 177 Grade E (the smallest) abattoirs.

Because of this greatly improved situation the Chief Meat Hygiene Officer has now entirely withdrawn all exemption from those sections of the Act concerning inspection. The Act is now fully applicable to all 337 approved abattoirs and this means that all cattle, calves, sheep and goats, pigs and ostriches will have to be inspected before slaughter by veterinarians. In addition, secondary post-mortem inspection by veterinarians of detained carcases/organs for evaluation with a view to either approval, conditional approval or partial or total condemnation is required. It can be anticipated that in instances where personnel for routine primary inspection are not available, veterinarians may be approached to also undertake such routine inspections. Only where no veterinarian is available will an abattoir be given exemption from these inspection requirements.

For the purpose of acting as Veterinary Meat Inspectors under the Act, veterinarians who are approached by local authorities or private owners to undertake such inspections will have to be duly authorised. The Chief Meat Hygiene Officer, the Regional Meat Hygiene Officer or the Assistant Director (Veterinary Services) in the area* should, of course, be consulted in this regard.

*Western Cape (Bellville), Eastern Cape (Port Elizabeth), Natal (Pietermaritzburg), the Witwatersrand (Johannesburg), Pretoria or the Asst. Directors (V.S.) in Bloemfontein, Potchefstroom and Pietersburg.

Some abattoirs may require the full-time services of a Veterinary Meat Inspector but the majority will manage with part-time services or daily sessions and these will be the subject of contractual negotiations between the parties concerned.

The South African Veterinary Association has for many years advocated the official use of the part-time services of practising veterinarians in order to extend, promote and stabilize rural veterinary services. The advent of tuberculin testing and subsequently the brucellosis eradication scheme has done much to achieve these objectives. With the addition of meat hygiene work the matter has been taken a step further. Provided the members of the profession make their services available, the Chief Meat Hygiene Officer's decision will give greater breadth and depth to the scope of veterinary services and constitutes a major contribution to the promotion of human and animal health in South Africa. It goes without saying, however, that those veterinarians who become involved in meat hygiene work need to ensure that they are completely au fait with their duties and responsibilities in terms of the Act. They must be persons of integrity who are fully capable of making the correct diagnosis and evaluation of the fitness of the carcase for human consumption to be able to take full professional responsibility for the taking care of the interests of the consumer, the butcher or owner of the carcase, the producer of the food animal and the profession. To this end and provided there is an adequate demand, the Department of Veterinary Food Hygiene of the University of Pretoria's Faculty of Veterinary Science will be giving serious consideration to offering a short refresher course at an early date.

It is also incumbent upon veterinarians who are approached to take up duties as Veterinary Meat Inspectors to adopt an essentially reasonable and conservative attitude towards remuneration for their services when negotiating with local authorities or private owners of red meat abattoirs. The stabilising influence of a part-time appointment as Veterinary Meat Inspector on a newly established practice in a small town should not be overlooked. In addition, the opportunity to see the slaughter of the animals of one's meat producer clients and thus to be able to render a better advisory service to them in regard to the control of disease conditions diagnosed or confirmed at the abattoir, can be of inestimable value to both producer and veterinarian.

Veterinary public health has been defined by WHO as a component of public health activities devoted to the application of professional veterinary skills, knowledge and resources to the protection and improvement of human health. Meat hygiene has been one of the basic and long established components of veterinary public health. In an editorial appearing in this journal in 1979 (Vol 50 (3) : p 147) it was stated that there appeared to be little evidence of extension of the South African veterinarian's traditionally restricted sphere of activities

to ensure maximum utilisation of his training and capabilities. The Chief Meat Hygiene Officer is to be commended for changing this situation and introducing a new phase in meat hygiene services. By doing so he has completed a system which has as its goal the maximum protection of the interests of the consumer, meat trade and meat animal producer by maximum utilisation of available scientific veterinary knowledge and expertise. Provided the profession takes up the challenge this development will not only result in beneficial extension of veterinary services to all parts of South Africa but will also add greater meaning to the teaching of veterinary public health on the undergraduate level. It is perhaps by coincidence that the Chief Meat Hygiene Officer's Notice 506 of 1983 comes into effect on 1 January 1984 – the day that the newly established independent Department of Veterinary Public Health of the University of Pretoria's Faculty of Veterinary Science comes into being.

BOOK REVIEW BOEKRESENSIE

VETERINARY ANAESTHESIA

L.W. HALL and K.W. CLARK

8th edition. Bailliere and Tindall, London 1983 pp VII + 417 Figs 213 Tables 14 ISBN 0-7020-0961-X
Price ± R40,00

The earlier editions titled 'Wright's Veterinary Anaesthesia and Analgesia' are well known publications to student and practitioner. The effective use of these editions have been impaired by unnecessary subdivision, especially for those not well familiar with the book. In this revised edition it is divided into two principle categories. The first part deals with general principles in anaesthesia and part two discusses anaesthesia in the different species. A third part is concerned with specific anaesthetic topics such as accidents/emergencies and techniques for cardiac and intrathoracic surgery.

In the first part, Chapter 1, the subject of anaesthesia is discussed in its broad content. Definitions, the selection of an appropriate anaesthetic technique, pre-anaesthetic examinations, etc. are discussed. In the second chapter monitoring techniques and apparatus available to the anaesthetist are discussed. In the third chapter an introduction is given to general anaesthesia, the signs and stages of anaesthesia and the side effects of theatre contamination with traces of anaesthetic to personnel. Chapters 4-7 deals with the pharmacology of drugs used in premedication, induction and maintenance of anaesthesia. Drugs used currently like ketamine, etomidate and isoflurane are included. In Chapter 8 spontaneos and artifical ventilation are compared and the mechanism of side effects explained. In Chapter 9 apparatuses for inhalation anaesthesia are presented. The British and American classifications of inhalation apparatus are compared. Chapter 10 is devoted to the principles of local analgesia, the anatomy and physiology of nerves and the mode of action of local analgesics. The methods of producing local analgesia are discussed.

In the second part the anaesthetic aspect of each domestic animal is fully covered from sedation to maintenance of general anaesthesia, including specific local anaesthetic techniques appropriate for each species. A section on obstretical anaesthesia is included. The chapter on anaesthesia of the horse has been revised extensively and particular attention has been given to the pathophysiological effects of posture on pulmonary function. In the chapter on the bovine, appropriate attention has been given to local anaesthetic techniques such as intravenous regional analgesia, paravertebral and pudendal nerve blocks. The chapter on the pig deals with the problem of malignant hyperthermia, techniques for intubation and currently popular drugs used for induction such as 'Saffan', ketamine and metomidate. The anaesthesia of the dog and cat is covered comprehensively. A new chapter on anaesthesia of birds, laboratory animals and wild animals has been included.

The third part deals with emergencies in the cardiovascular and respiratory system. The last chapter deals with anaesthetic techniques for thoracic and cardiac surgery.

This 8th edition has fulfilled the requirement of a standard reference on the principles of anaesthesia and the application in all the domestic animals.

G.F. Stegmann

FINANCIAL AWARENESS FOR VETERINARY PRACTITIONERS

J.W.E. ADAMS*

ABSTRACT: Adams J.W.E. **Financial awareness for veterinary practitioners.** *Journal of the South African Veterinary Association* (1983) **54** No. 4, 219-222 (En). Durban Veterinary Clinic, 12 Currie Road, 4001 Durban, Republic of South Africa.

Simple methods are explained which enable private practitioners to organise and understand month-end financial information and consequently to react thereto.

Key words: Financial, Veterinary practitioner.

INTRODUCTION

In today's climate of high inflation, high interest rates, unemployment, and generally unfavourable economic conditions, the practising veterinarian usually finds himself poorly trained to make decisions in his own best (least worst) interest.

The financial information that is deemed necessary in this practice at each month-end is described. This information enables one to determine one's financial position and then to make informed decisions which flow from that position.

The steps that will be described are all practical and not time consuming, and should fall easily within the capabilities of all practitioners. Nothing is included or suggested that is not carried out by the author at each month-end. Those brave souls tempted to follow the routine suggested here, undeterred by what they might discover, are hereby warned. If "e" equals the effort required to maintain this or any new possibly improved routine, then "e^3" is the minimum effort needed to introduce the change.

MONTH-END TOTALS

The month-end totals are listed on a typed form as indicated in Fig. 1. These figures must be available on the day the books are closed at the month-end.

1. Number of Clients (or Invoices): This is the most important piece of information as far as practice growth is concerned. This, as well as turnover, and the amount paid per client (invoice) are used to graph the practice's performance monthly, and from year to year. These graphs are considered in detail below.
2. Turnover: This is the amount of money available to meet expenses if the practice is on a cash basis. If credit is permitted, or encouraged (heaven forbid) then turnover defines the amount of money that might become available at some indeterminate future data. Businessmen offering credit are called "benign financiers" competing with banks and credit card organisations but not enjoying any financial gain.

3. Expenses

3.1 *Rent:* This is the cost of occupying the practice premises. It is either the rental paid to a landlord or a sum of all property expenses, e.g. bond, rates, insurance. Where the premises is owned by the practice, a market related "rental" must be budgetted for.

*Durban Veterinary Clinic, 12 Currie Road, 4001 Durban.

3.2 *Drugs:* To calculate drug costs properly, one needs a record of purchases, sales, practice use, and an opening and closing stocktake. We settle for checking the drug invoices and literally keeping an eye on stocks. The purchases should vary with turnover. If they do not, find out why not.

3.3 *Salaries:* Note the column for bonusses which are paid when turnover warrants it.

3.4 *Telephone:* International and trunk calls not made on practice business are charged to the doctor's personal drawings.

3.6 *Petty Cash:* The total is viewed in relation to monthly average and to turnover. Periodically vouchers (signed) are scrutinised.

3.8 *Motor Expenses:* In our practice motor vehicles are owned by the veterinarians personally. All expenses are charge through the practice. Equal fuel allowance are paid from petty cash weekly and maintenance differences are equalised by salary, adjustment.

For example:

	Dr A		Dr B		Total
Motor Expenses	R100		R200		R300
Adjustments	+ 50		− 50		÷ 2
	R150	=	R150	=	R150

Salary adjustment is thus an additional R50 for Dr A while Dr B has R50 subtracted.

3.10 *Professional Salaries:* Knowing turnover and having totalled expenses one is able to calculate the funds available to the partners. It is recommended that as far as possible this should be a constant amount each having in mind the level of turnover. A fixed monthly income also aids budgetting on the home front. To calculate annual income:

Annual Turnover
less Practice Expenses
Income available to partners (÷ partners)
less Income on capital account (invesment income)
Income as practising veterinarian
less Income Tax
Disposable Income

e.g. R100 000 Turnover
less R 60 000 Practice Expenses
R 40 000 (÷ 2 partners)
= R 20 000
less R 1 000 (capital account 10 000 @ 10%)
R 19 000
less R 5 000 (Income Tax)
R 14 000

0038-2809/83/04/0219-0222

	Debtors	(at beginning of month)
plus	Turnover	(current month)
	xxxxxxxx	
less	Banking	(current month)
	Debtors	(at the end of the month)

e.g.	2 000	(debtors at beginning of month)
plus	10 000	(turnover)
	12 000	
less	9 500	(banking)
	2 500	(debtors at end of month)

NB: Banking: The figure used is actual banking less bad debts recovered and included in deposits.

Debtors: Any debts moved into the "bad" file must be subtracted from the derived figure.

The calculated debtor's figure should agree with the debtor's figure given by the bookkeeper. Any disagreement should be accounted for by checking the accuracy of all elements in the calculation as well as auditing the debtor's list.

GRAPHS

While it is informative and interesting to graph many aspects of practice the following monthly figures are considered essential.

1. Turnover
2. Number of invoices issued (or number of clients seen)
3. Average charge per invoice/client
   (Turnover ÷ invoices/clients)

It is convenient to use 3 different colours representing the above, solid lines for the current year and dotted lines for the previous year.

This display tells much more than may be immediately apparent.

If turnover is up how has it risen? More clients, more rands per client, or both?

The percentage increase in turnover from one year to the next should not be less than the increase in consumer price index (inflation) to be making progress.

The increase in rands and cents per client is the indicator of fee adjustment. To keep pace with inflation this amount should not be less than the increase in inflation.

True growth is an increase in the number of clients. An ideal situation would be an increase in turnover exceeding the inflation rate, brought about by fee increases equal to the inflation rate, and more clients seen this year than last.

INCOME: EXPENSES

Increased expenses can quickly offset advances made in income. If costs are accurately recorded they can be expressed as a percentage of income. Expenses are volatile and are not necessarily consistent but if they are approximately 50 % of turnover well and good. Should running costs exceed 60 %, leaving less than 40 cents per rand charged for distribution to professional staff then the practice profitability needs investigating.

CONCLUSION

The foregoing recommendations may seem onerous and tedious but any veterinary business ignores them at its peril.

The steps described can be completed in 1½ hours per month. One hour for salary and bonus calculations including cheque signing at the end of the month. A further 30 minutes is needed for graphs and reviewing the bank reconciliation, usually between the 5th and 10th of the month.

More formal statements of account, e.g. trial balances, statement of income and expenditure, and the balance sheet are ideal instruments for practice financial control. However, until they are produced by an in-house computer they remain unfortunately too removed in time to assist in month end deliberations.

Nothing has been said here about budgeting or forecasting. This has been left for a future occasion in the belief that as in navigation, to find out where you are going, first determine where you are.

HORSE LYMPHOCYTE CELL SYNCHRONIZATION : IMPROVED TECHNIQUE FOR CHROMOSOME ANALYSIS

U.MÄRKI and D.R. OSTERHOFF*

ABSTRACT: Märki U.; Osterhoff D.R.**Horse lymphocyte cell synchronization: Improved technique for chromosome analysis.** *Journal of the South African Veterinary Association* (1983) **54** No. 4, 223-224 (En). Department of Zootechnology, Faculty of Veterinary Science, University of Pretoria, P O Box 12580, 0110 Onderstepoort, Republic of South Africa.

A method using methotrexate for horse lymphocyte cell synchronization and thymidine for incorporation into DNA replication is described. This method provides a powerful technique for the study of chromosomal abnormalities and detailed analysis of chromosomal replication patterns. The determination of horse karyotypes with many similar chromosomes needs a special method which reveals the numerous and informative stages of the cell cycle.

Horse lymphocyte cell cultures treated with colcemid (20 min) and harvested 6 hours after the release of the 17 hour-block with methotrexate show the best results for exact identification and analysis of horse chromosomes.

Key words: Horse, leukocyte, lymphocyte, cell synchronization, methotrexate, chromosomal abnormalities.

INTRODUCTION

During recent years many cell synchronization techniques have been developed using thymidine[1 4 5], amethopterin[1 10] or bromodeoxyuridine (BRdU)[5] to obtain R- or G-band prophasic and prometaphasic chromosomes. Methotrexate (4-Amino-10-methylfolic acid) (MTX) is a potent inhibitor of the enzyme dihydrofolate reductase[8 9] and the addition of this 4-amino analogue of folic acid to cultures of exponentially growing mammalian cells inhibits the synthesis of thymidilic acid[7]. This thymidineless state can be stopped by the release of MTX and enrichment of the media with thymidine or a thymidine analogue, for example BRdU. Chromosomal aberrations in domestic animals seem to be responsible for subfertility and embryonic death[6] and are also to some extent implicated as a reason for infertility and subfertility in stallions and mares. The use of MTX as a synchronizing agent in horse lymphocyte cell synchronization provides a great help in the karyotype analysis of horses, because a great number of metaphases with very long chromosomes can be obtained. The diagnosis of any chromosomal abnormalities and their relationship to infertility and/or subfertility in horses requires a distinct identification of the chromosomes.

MATERIALS AND METHODS

Pokeweed-stimulated horse peripheral lymphocytes were cultured at 38°C in Medium 199 (Gibco Europe) supplemented with 20 % foetal calf serum for 72 h. Two different procedures were followed:

Non-synchronized cultures

Before the cultures were harvested they were treated with 0,2 ml colcemid (10 mg/ml) for the last 60 min of culture time. Hypotonic treatment by 0,075 mol KCl, fixation and preparation of slides was done according to the conventional cytogenetic technique used for chromosomal analysis.

Synchronization of the lymphocytes

After 72 h the cells were partially synchronized by 10 µmol MTX. At the end of the 17 h MTX block, the cells were released by 2 washes with Medium 199 supplemented with foetal calf serum. The cells were finally resuspended and reincubated in complete media supplemented with thymidine (0,3µg/ml) for 5, 6, 7 or 8 h respectively. Cells were harvested at hourly intervals after the release of the block, following the exposure to 0,2 ml colcemid (10 mg/ml) for either 20 or 60 min. The cells were harvested as described above.

The G-banding staining was performed as follows:

The one day-old air-dried preparations were incubated at 65°C for 60 min and afterwards for 15-20 in trypsin (0,03 g trypsin in Dulbecco's phosphate buffered saline (PBS) without calcium and magnesium). After dipping twice into phosphate buffer (pH 6,8), the slides were stained in 2 % Giemsa solution in phosphate buffer (pH 7,2) for 15 min and then mounted with Entellan (Merck, Germany).

RESULTS AND DISCUSSION

The results are presented in Table 1.

In comparing number and quality of the metaphases between non-synchronized and synchronized cultures it became evident that:

1. In synchronized cultures, the best results with colcemid treatment were reached at 20 min, instead of 60 min.
2. In nonsynchronized cultures, the quality of the few metaphases seen was not satisfactory after a colcemid treatment of 20 min, but was somewhat better using a longer treatment.

Table 1: HORSE LYMPHOCYTE CULTURES, SYNCHRONIZED AND NON-SYNCHRONIZED

Colcemid treatment min	Nonsynchronous	Hours after MTX release in BUdR-encirched medium			
		5	6	7	8
20	–	+	++++	+++	+
60	+	+	++	+	+

–, inconsistent results; +, very few metaphases; ++, good results; +++, very good results; ++++, excellent results

In synchronized cultures, changes in number and quality of the metaphases were closely related to the length of time the cells were cultured after the release of the MTX block (Fig. 1). The results were much better

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6 h after the release of the block in comparison to 5 h afterwards. The best and most abundant metaphases were found at 6-7 h after the block release.

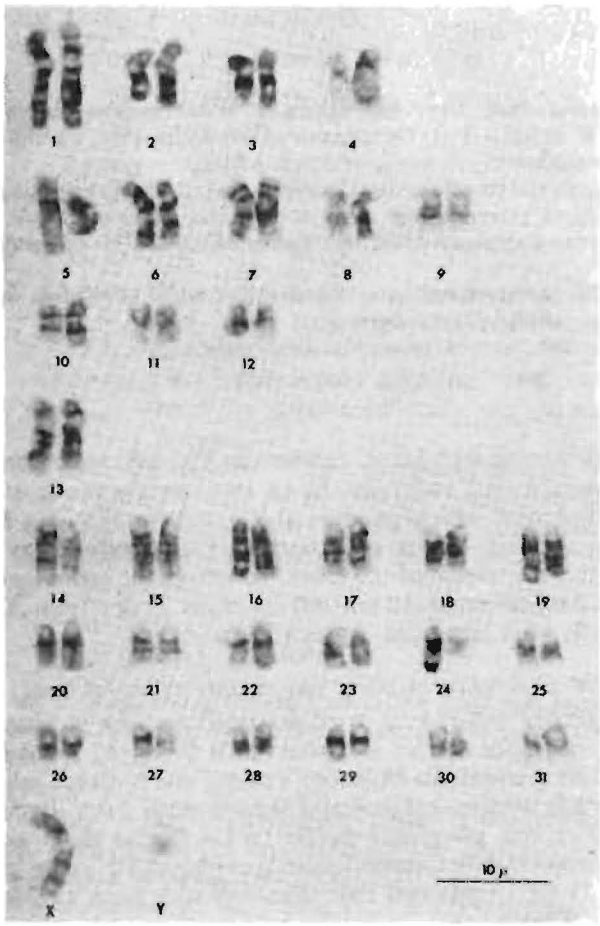

Fig. 1: G-band karyotype of a normal stallion with 31 identical or homologous pairs of chromosomes known as autosomes (chromosome Nos 1-31) and with 1 divergent pair of chromosomes (chromosome X and Y), after the lymphocyte cell synchronization with a 17 h MTX treatment and a 6 h reincubation with thymidine.

Camargo et al.[1,2] found the mitotic peak of human lymphocytes was reached approximately 5-6 h after the removal of the MTX block. At this time (15 min after colcemid treatment), a mitotic index of 9-14 % was observed. This can be regarded as a very high value for the mitotic index for phytohemagglutinin (PHA) stimulated lymphocytes, considering that only 50-80 % of the lymphocytes respond to PHA[3].

In the present study mitotic indices were not calculated because mitoses observed only give a numerical answer but do not indicate the quality of the metaphases. In the horse in particular, with its large number of chromosomes, long chromosomes are required to show up the different banding patterns.

Many different metaphase stages of the S-phase of the cell cycle were observed, probably due to blocking of the horse lymphocytes at different times of the cell cycle. The combination MTX and thymidine incorporation for cell synchronization followed by colcemid treatment resulted in a powerful technique for the examination of chromosomal abnormalities and detailed analysis of chromosomal replication patterns. An exact analysis of high mitotic index, increased frequency of prophasic and prometaphasic mitoses and high percentage of informative replicational stages in the S-phase is needed for the establishment of the horse karyotype with its great number of similar chromosomes.

In further investigations the timing of the cell synchronization will be refined with a more exact analysis of the different stages of the S-phase by means of flourescent banding techniques.

ACKNOWLEDGMENTS

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FUNCTIONAL ENDOCRINE MODIFICATION OF THE THYROID FOLLOWING OVARIECTOMY IN THE CANINE

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ABSTRACT: Van der Walt J.A.; Van der Walt L.A.; Le Roux P.H. **Functional endocrine modification of the thyroid following ovariectomy in the canine.** *Journal of the South African Veterinary Association* (1983) **54** No. 4, 225-229 (En). Box 16779, 0116 Pretoria North, Republic of South Africa.

The study involves bilateral ovariectomy in bitches followed by the monitoring of oestradiol, thyroxine and total triiodothyronine levels in order to study control mechanism alterations. Levels of oestradiol dropped significantly following surgery to reach basal concentrations after 2 weeks. Serum total thyroxine, free thyroxine and total triiodothyronine decreased following ovariectomy to stabilize in a steady state within 3 weeks of surgery. Levels of thyroid-binding globulin initially increased as a result of oestrogen ablation, thereafter levels decreased steadily. Modification of the hypothalamo-thyroxine-releasing hormone receptor site and changes in androgens and thyroid-binding globulins are discussed as possible causes for the changes in circulating thyroid hormone concentrations.

Key words: Thyroid functions, eunuchoid syndrome, ovariectomy.

INTRODUCTION

Bilateral ovariectomy in bitches is a common veterinary procedure but may lead to the development of an eunuchoid syndrome as a result of hypooestrogenism[17]. The clinical signs of the eunuchoid syndrome closely mimic those symptoms associated with hypothyroidism. As a result, the treatment of the eunuchoid syndrome may take the form of thyroid replacement, often in conjunction with oestrogen therapy. A method of preventing the development of this syndrome is by the autotransplantation of ovarian tissue to the gastric serosa[16 17], thereby ensuring liver metabolism of steroids prior to their release into the general circulation.

Thyroid hormones are recognised as the regulators of metabolism in most tissue in adult homeotherms. Since the calorigenic activity of triiodothyronine (T_3) is 3 – 5 times greater than that of thyroxine (T_4), T_3 is the major hormone regulating energy metabolism[24]. The conversion of T_4 to T_3 through monodeiodination in peripheral tissue accounts for at least 80 % of the circulating T_3 in humans[24] as well as in sheep[10] and rats[25]. In man, less than 0,05 % of the total serum T_4 is present in the free form[24]. Since the intracellular hormone is in equilibrium with the free hormones, the latter would represent a more accurate indicator of the hormone dependent metabolic state.

Oestrogens remain the most common cause of thyroxine-binding globulin (TBG) increase and have shown to cause fluctuations in TBG with topical application[15]. Anabolic steroids and androgens[4] produce converse effects to oestrogens[19]. Large doses of glucocorticoids are also associated with a decrease in TBG concentrations[22]. Adrenal steroids suppress thyroid-stimulating hormone (TSH) secretion through a direct effect upon the pituitary and also inhibit responsiveness of the pituitary to thyroid-releasing hormone (TRH)[23].

Pharmacological doses of oestrogen also inhibit TSH secretion by mechanisms that may be of a direct nature or via the hypothalamus[13]. Tests have been developed for the evaluation of the integrity of feedback mechanisms regulating thyroid gland functions[21]. When applied in combination with other tests, they provide a means of differentiating between thyroprivic, pituitary and hypothalamic thyroid failure. Unfortunately the basis of these tests rests on the concurrent measurement of TSH[21]. Since the thyrotropins are species specific glycoproteins it has not proved possible to utilize interspecies antibodies for the measurement of canine TSH. For this reason the differentiating tests have proven of minimal value in animal studies.

In the canine, hypothyroidism is reflected by an array of clinical signs such as mass increase, dullness, lethargy, hypocholesterolaemia, artherosclerosis and hyperlipidaemia. Skin changes occurring in dogs with hypothyroidism have been well documented[19]. The assessment of thyromegaly in canines through the use of TSH respone tests have proven to be of some value[6 14], while hypothyroidism has been suggested as a possible cause for invertebrate disc disease[12]. An interesting observation from the latter report is that significantly more spayed females were affected by the syndrome.

The eunuchoid syndrome in bitches refers to the clinical signs which result from removal of the sexual organs. A large proportion of ovariectomized bitches develop this syndrome which is thought to be precipitated by a subclinical deficiency of female sex steroids. Clinical signs which characterize the syndrome include urinary incontinence, obesity, alopecia, pruritis, seborrhoea and vulvar dermatitis[16]. Spayed bitches maintained on daily doses of ethinyl oestradiol (5-10 mg/kg) become more alert and active, but will revert to the pretreatment state if medication is withdrawn[16].

In an attempt to determine the possible relationship of these conditions, a group of experimental bitches were ovariectomized and their concentrations of total thyroxine (TT_4), free thyroxine (fT_4), total triiodothyronine (TT_3) and oestradiol (E_2) were monitored at weekly intervals. Concurrent estimations of TBG was accomplished through an assay for triiodothyronine uptake (T_3U).

MATERIALS AND METHODS

Subjects

The subjects used in this study comprised 12 female German Shephard dogs (7-31 months of age) supplied by the South African Defence Force Dog Training Centre. The bitches were selected at random from those females which were to be excluded from the Army's own breeding programme. The bitches were to be ex-

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cluded for a variety of reasons, the main one being nervous disposition. None of the animals had previously been bred and none had shown oestrus irregularities.

Sample collection

The day prior to surgery 10 ml blood was collected into plain evacuated glass containers from the cephalic vein (Sample 0). The following day the bitches were ovariectomized. Starting on Day 6 following surgery, the dogs were bled at weekly intervals for 5 consecutive weeks (Samples 1-5). After being allowed to clot, the blood was centrifuged, the serum was drawn off and aliquoted for storage at −20 °C until assay. The samples were accumulated over the trial period and then processed as a single group in order to obviate interassay variation.

Assay Methodology

E_2 was assayed by a radioligand method as previously described[16]. TT_3 and TT_4 were assayed by specific radioimmunoassay methods[16,17], while T_3U was assayed by a standard solid-phase method utilizing a specific free hormone antibody titred to yield a specific TBG value. The assay for fT_4 was accomplished utilizing the method of Amersham International. This assay uses extremely low concentrations of antibody and a high specific activity label. It has proven highly specific and precise, and delivers results correlating to the standard equilibrium dialysis method with minimal equilibrium imbalance.

Statistical analysis

Radioimmunoassay data were analysed by fitting a weighted regression plot to a probit-log transformation by computer. Statistical analysis of data was performed on a Hewlett-Packard 250 computer using appropriate programmes for one-sample statistics. The samples collected each week were evaluated to determine the mean ($\bar{x}$), standard deviation (SD) and the coefficient of variation. Statistical significance was calculated using the student's t-test.

RESULTS

Oestradiol

In this study the preoperative concentrations of E_2 ranged from 132-310 pmol/ℓ ($\bar{x}$ = 198, SD = 24) (Fig. 1). The third week following surgery the values had dropped to range between 88 and 110 pmol/ℓ. This represents a 50 % decrease in the level of circulating E_2. This concentration of E_2 accounts for that hormone produced from the biosynthetic pathways of the adrenal cortex. The most dramatic and highly significant ($p < 0{,}001$) drop occurred during the first week following surgery. The decline observed after the first week followed what appeared to be a half-life removal pattern. Once equilibrium had been reached, the post-operative serum concentrations differed significantly from those preoperative ($p < 0{,}0005$).

Total triiodothyronine

The serum concentrations of TT_3 revealed a decline from Sample 0 to reach an equilibrium approximately 17 % below the original starting level (Fig. 2). The decline of TT_3 is statistically highly significant ($p < 0{,}0005$). The most dramatic decline occurred between Week 2 and 3 ($p < 0{,}0001$), while there was no significant difference between preoperative levels and

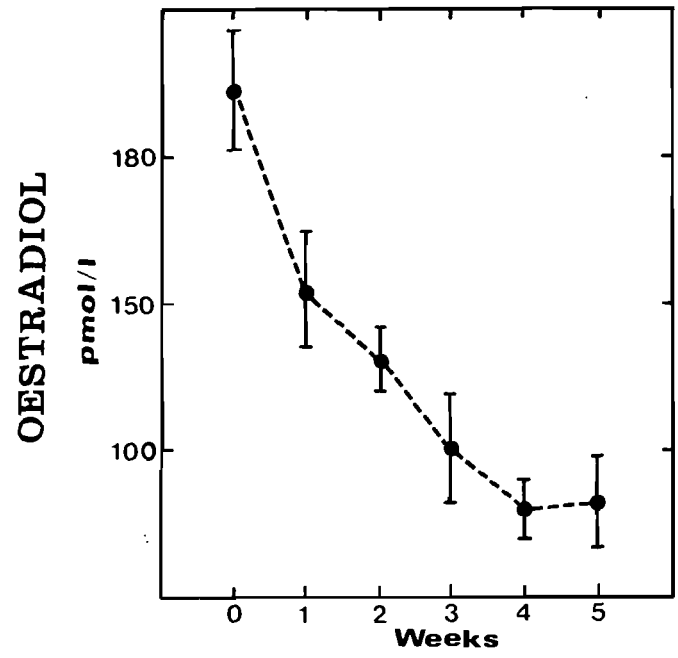

Fig. 1: Mean (± SD) peripheral E_2 concentrations in bitches prior to and following ovariectomy.

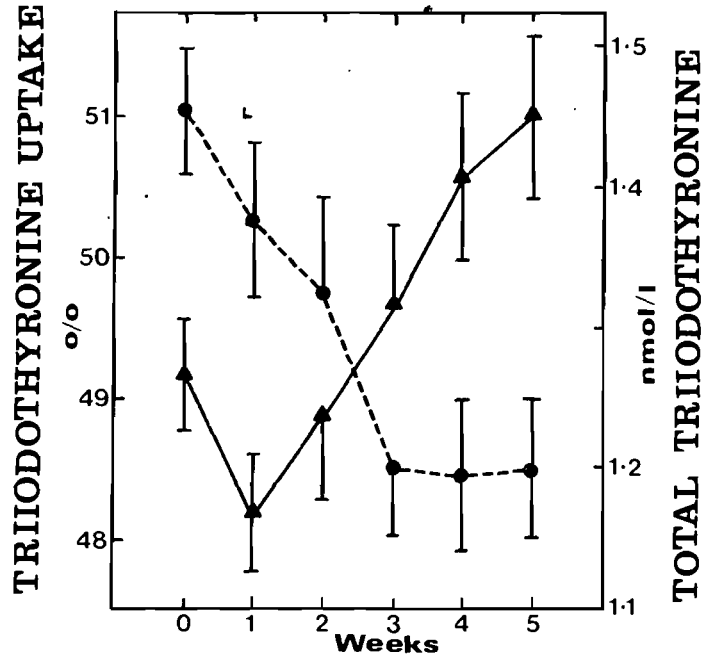

Fig. 2: Mean (± SD) peripheral TT_3 concentrations (● – – – – ●) and T_3U (▲——▲) in bitches prior to and following ovariectomy.

those obtained at one week following surgery. The preoperative range of TT_3 was 1,24-2,1 nmol/ℓ ($\bar{x}$ = 1,46 ± 0,24 nmol/ℓ).

Triiodothyronine uptake

The range of the preoperative samples was established in the experiment to fall between 46,7 and 51 % (Fig. 2). Preoperative assay of T_3U delivered a mean of 49,2 ± 1,3 %. A decline in T_3U was descernible at Week 1 ($p < 0{,}05$) followed by a gradual rise through the monitoring period to reach a mean peak level of 51,1 %. The difference between the mean preoperative sample and that at Week 5 is highly significant

($p < 0{,}0005$). These results would indicate that binding protein levels rose sharply following ovariectomy with a subsequent gradual decline in the binding capacity.

Total thyroxine

Preoperative TT_4 concentrations ranged from 19,6-48,9 nmol/ℓ ($\bar{x} = 31{,}4 \pm 4{,}8$ nmol/ℓ) (Fig. 3). TT_4 then declined by approximately 17·% in the first week following the ovariectomy, although this difference was not of statistical significance ($p < 0{,}01$). Thereafter the levels of TT_4 plateaued to Week 2, declining once again to a nadir of 30 % of the original concentration at Week 3 through Week 5. The difference between the concentration preoperatively and that reached at equilibrium is highly significant ($p < 0{,}0025$).

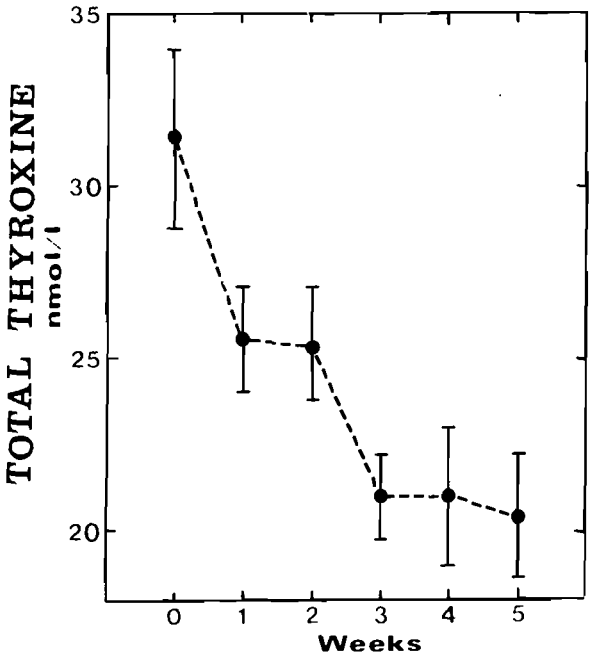

Fig. 3: Mean (± SD) peripheral TT_4 concentrations in bitches prior to and following ovariectomy.

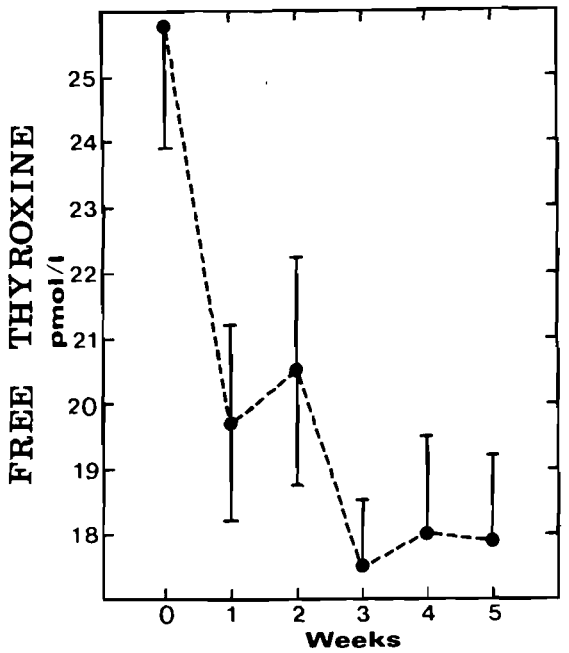

Fig. 4: Mean (± SD) peripheral concentration of fT_4 in bitches prior to and following ovariectomy.

Free Thyroxine

The range of fT_4 prior to ovariectomy in the experimental bitches varied from 15,1-44,5 pmol/ℓ ($\bar{x} = 25{,}7 \pm 8$ pmol/ℓ) (Fig. 4). The concentrations of fT_4 dropped by some 23 % in the first week following surgery ($p < 0{,}0025$). Thereafter the values rose slightly to Week 2 and subsequently declined to reach equilibrium at Week 3 following ovariectomy. The total decline in fT_4 pre- and postoperative was 22 % ($p < 0{,}0025$).

DISCUSSION

Thyroid hormone deficiency leads to a wide variety of physiological and clinical disturbances involving virtually every organ system. The insidious onset of hypothyroidism and progression of the disease makes recognition and diagnosis difficult. Following thyroidectomy or hypophysectomy, 3-4 weeks are required for the development of symptoms[18]. A similar interval is required for the appearance of histologically-identifiable myxoedema[11]. Marked symptoms do not appear for 2-3 months. These delays reflect the presence of substantial stores of T_4 in extracellular fluid and tissues.

Peripheral circulating levels of E_2 in the trial bitches indicated that none were in oestrus at the time of the study. The mean E_2 concentration prior to ovariectomy was 198 pmol/ℓ and is in accordance with values obtained in the same laboratory in different studies on a variety of animal species[2,17]. Previously, a study in dogs had demonstrated the ovarian production of E_2 of bitches in oestrus to reach a peripheral concentration as high as 700 pmol/ℓ with concurrent proof of ovulation demonstrated by elevated progesterone values[16]. Levels of E_2 in the present study dropped significantly following ovariectomy but failed to reach adrenal basal values until 2 weeks post surgery. The most dramatic decrease in E_2 concentration occurred between the time of ovariectomy and the first sampling a week following surgery. E_2 displays a typical and classical ablative half-life clearance pattern. Free E_2 has been shown to possess a half-life clearance rate of approximately 20 min. Since the majority of E_2 (some 98,7 %) is bound to cytosol receptors, the attainment of a true steady equilibrium following ovariectomy will be delayed. In the current study, basal E_2 concentrations of between 70 and 110 pmol/ℓ were estalished 2 to 3 weeks postoperative. The slope of the decrease from Week 1 is consistent through Week 3, indicating a gradual displacement of the bound E_2 and the attainment of equilibrium. Measurement of E_2 a year following ovariectomy in bitches[16] has resulted in typical concentrations of this magnitude.

Subsequent to ovariectomy, the number of available E_2 binding sites on the carrier protein will rise as the specific hormone decreases. In effect, this will release many sites for non-specific binding. The particular E_2 binding protein (steroid hormone binding globulin, SHBG) thereafter probably follows a similar trend to E_2 since its concentration is controlled and modulated by the circulating levels of E_2. The clearance rate of the protein is of far longer duration than that of the steroid and one may thus expect a more protracted influence by the SHBG rather than the E_2.

The most dramatic secondary changes resulting from ovariectomy occurred in the thyroid function tests. All the thyroid function parameters assayed, with each animal serving as its own control, showed significant modification following surgery. The concentrations of

TT_3 showed a significant decrease, plateauing to a mean level of 83 % of the original value at Week 3 following ovariectomy. In the case of this hormone, the most significant drop occurred between Week 2 and 3. The explanation for this phenomenon is unclear. It would appear that a steady state is reached from Week 3 onward. Initial studies[16] had indicated little significant change in the concentration of TT_3 following ovariectomy when this parameter was assayed one year after surgery. The steady state reached from Week 3 is consistent in all animals, but is still within the limits of the normal range for the canine. A positive correlation has been reported between TT_3 concentration and body mass, with values increasing by overeating and decreasing with mass loss[5]. Rapid dimunition of TT_3 may also be produced by total caloric or carbohydrate deprivation[20]. This decrease may reach the same magnitude as the decrease registered in the present study, although it appears to be of a more rapid onset and may occur within 24 hours. Since food deprivation was not applied in the present study and the body mass of the animals remained constant, the changes in TT_3 cannot be attributable to diet. A more logical explanation is that the change in TT_3 is due to changes in TBG concentration. The magnitude of TT_3 change is, however, smaller than potential changes in T_4 since the affinity of T_3 for TBG is lower, relative to T_4.

The most dramatic thyroid hormone changes were those of the T_4 concentrations. One week following ovariectomy, the levels of TT_4 had decreased by 17 % while those of fT_4 showed a 23 % drop. Upon stabilization at 3 weeks following surgery, the concentrations of T_4 had decreased markedly by one third of the original values, yet still retained an identical free to bound ratio of 0,08 %. Both entities showed a stabilization occurring between Week 1 and Week 2, indicative of a rebound increase occurring at this time. When one, however, considers the TBG concentration at this stage, this phenomenon may be explained on the basis of increased TBG binding sites. Thereafter the TBG concentrations decrease, probably as a result of diminished oestrogen stimulation.

Serum TT_4 is low in conditions associated with a low TBG concentration[26]. In the absence of primary abnormalities in hormone secretion or in its regulation, changes in the fT_4 level should be continually adjusted by appropriate activation or inhibition of hormone secretion or inactivation. It is unlikely that the sensor controlling hormonal supply is set to detect an optimal intracellular hormone concentration, but may respond to a specific metabolic action of the hormone. Such an effect is, however, probably closely related to the intracellular hormone concentration and hence it is best quantitated by measuring the free hormone concentration in serum. Changes in total hormone levels can also arise as a result of changes or modification to the number or affinity of the available binding sites. This study would indicate that the binding ability is due primarily to the change in the concentration of the hormone-binding protein, since TBG decreases following ovariectomy. Whether the decrease in the TBG concentration is directly as a result of oestrogen deprivation or secondary to metabolic androgen domination subsequent to depletion of oestrogen, is not clear.

An important aspect in the control of cellular activity is through the modulation of the number of cell membrane receptor sites. A decreased number of receptor sites for a hormone would increase the hormone concentration required for full cellular response. In the present model, oestrogens, and thyroid hormones exert their effects on TSH responsiveness to TRH by modulating the number of affinity of TRH receptor sites[8]. This could then lead to a diminished thyroid hormone state through the re-setting of the sensor mechanism at a lower rate. Under these conditions it may be expected that measurements of the various thyroid hormone parameters would reflect changes in T_3 and T_4 concentrations which parallel the changes in total TBG concentrations and in the unsaturated binding sites of TBG. In agreement with other cyclic AMP-mediated systems[3,8], the primary step in the action of the hormone is receptor binding. Altered receptor synthesis or degradation, masking or unmasking of receptors through conformational changes, receptor release or translocation to an intracellular site[7] could account for the alterations of pituitary TRH receptor levels. Since treatment with oestrogens has been shown to increase mitotic activity while hypothyroidism increased the population of thyrotrophs, receptor changes may also be due to changes in the pituitary cell population[8].

The present study lends evidence for changes in thyroid hormone concentrations as an ultimate result of ovariectomy, which may eventually manifest clinically as hypothyroidism as a result of hypooestrogenism. Both TT_3 and TT_4 decrease following ovariectomy to stabilize in a new steady state within 3 weeks of surgery. Levels of TBG as indirectly assessed by the T_3U test increase initially, probably as a result of non-specific binding site increase following the sudden ablation of ovarian oestrogens. Hereafter, the levels of TBG decrease steadily. These phenomena may be explained through oestrogen deprivation leading to a decreased number of conformation of hypophyseal TRH receptor sites. TSH responsiveness will now be affected leading to a decrease in the levels of thyroid hormone. A contributory factor to this mechanism may be the role played by the suddenly dominant androgens which, in the absence of oestrogen, will further decrease TBG concentration, thereby increasing the severity of the condition.

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BOEKRESENSIE — BOOK REVIEW

VETERINÄRMEDIZINISCHE PARASITOLOGIE

J. BOCH & H. SUPPERER

3de Uitgawe. Verlag Paul Parey, Berlin & Hamburg. 1983 pp iv + 533 Fig. 192 Prys DM 98
(ISBN 3-489-66116-8)

Hierdie reeds bekende handleiding is met die nuwe uitgawe bykans heeltemal op datum gebring en in 'n paar opsigte verbeter. Metodiek, beheer en behandeling is heelwat opgeknap en tot en met 1982 is die literatuur bygewerk. Die outeurs het die hulp van verdere medewerkers ingeroep om sekere van hierdie afdelings te verbeter en baie moeite is duidelik gedoen om wyd na te slaan ten einde die jongste inligting te bekom.

'n Paar voorbeelde dien gemeld te word: 1. Die voorbereiding van monsters vir versending is vir die eerste keer in die boek ingesluit. Dit beantwoord noodsaaklike vrae soos die neem, fiksering al dan nie, manier van verpakking en maksimum tydsduur voor monsters die laboratorium bereik. 2. Wat betref diagnose is die McMaster metode (wat in 1939 beskryf, en lank reeds wêreldwyd algemeen vir wurmeiertellings in die mis van herkouers gebruik word) uiteindelik opgeneem. Dit is egter jammer dat met betrekking tot die interpretasie van die namatode eiertelling in die mis (e.p.g.), slegs gesê word dat dit baie versigtig interpreteer moet word, sonder om enige verdere besonderhede te verskaf. 3. 'n Afdeling oor die konsentrering van mikrofilaria en kliniese laboratorium ondersoeke (bv. pepsinogeen bepaling) word ook nou aangebied. 4. Die metodiek van nadoodse ondersoek is ook vir die eerste keer ingesluit. 5. Die herwinning van ova uit onder andere grond en gras kan as uiters handige inligting beskou word.

'n Paar lastige drukfoute is opgemerk, bv. *Ostertagia ostertagia* (i.p.v. *ostertagi*) (op 2 plekke), *Fasziola hepatica* (i.p.v. *Fasciola*) en Le Rouse (i.p.v. Le Roux).

Die boek verskaf goeie basiese inligting wat vir die vakrigtings protosoologie, helmintologie en entomologie handig saamgevoeg is. Vir die Suid-Afrikaanse praktisyn en student wat 'n boek vir lokale praktyk benodig, is daar egter ernstige tekortkominge. Die epidemiologie is streng van toepassing op Duitse en Europese toestande en weens groot basiese verskille is dit nie slegs 'n saak om die "omgekeerde seisoene" (teenoor die suidelike halfrond) in berekening te bring nie. Inligting wat lokaal benodig word, is eenvoudig nie ingesluit in die boek nie. Die entomologie van herkouers word bv. in 38 bladsye afgehandel en bosluise soos *Amblyomma*, *Hyalomma*, *Boophilus* en *Rhipicephalus appendiculatus* word slegs met betrekking tot siektes genoem. Verder is daar slegs 'n kort afdeling oor weerstandbiedendheid van nematode teen wurmmiddels (inligting wat vir ons in Suid-Afrika onontbeerlik word). Die afdeling oor parasiete van wild, hoewel handig om saam gegroepeer te hê, is vir lokale omstandighede van weinig waarde, omdat parasiete alhier so baie vanaf dié in Europa verskil.

Opsommend is ek van mening dat die boek vir lokale navorsers van groot nut kan wees. Dit word egter nie vir praktisyns en studente aanbeveel nie, behalwe as hulle 'n spesiale belangstelling in parasitologie het.

J.A. van Wyk

XIIIth WORLD CONGRESS ON DISEASES OF CATTLE TO BE HELD IN DURBAN, SOUTH AFRICA, 17-21 SEPTEMBER 1984

SECOND ANNOUNCEMENT AND CALL FOR PAPERS

This promises to be a Congress no veterinarian involved in cattle practice and research can afford to miss. All colleagues also interested in presenting papers are cordially invited to submit abstracts of their intended presentations. These should be no longer than 300 words, typewritten in English and submitted to reach the Chairman of the Scientific Programme Committee at the address given below before 31 January 1984. An abbreviated list of proposed topics is:

1. Diseases of ruminants, domestic and wild, and their global importance (emphasis on foot and mouth disease, bluetongue, Rift Valley fever).
2. Herd health programmes (emphasis on computer programmes, herd reproduction and mastitis control, immunisation).
3. New concepts in the control of internal and external parasites (biological, chemical, immunological) and of tick-borne diseases.
4. Bovine nutrition including inadequate nutrition in causing disease.
5. Toxicology – the role of plant toxins, pesticides, insecticides and mycotoxins.
6. Bovine surgery aspects of anaesthesia and advances in genital surgery.
7. Reproduction (emphasis on dry cow management, post partum problems cystic ovarian degeneration).
8. Embryo transfer, recovery and preservation.
9. Game ranching in conjunction with bovines.

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The Small Animal Symposium is organized by the South African Veterinary Association's Clinicians Group. Its theme is continued education and will incorporate a Surgery Course on Soft Tissue Surgery (urogenital and gastro-intestinal tracts, and head and neck surgery) presented by Drs Richard Greene and Tom Greiner, and a Medical Course on Canine and Feline Endocrinology (disorders of the pituitary, thyroid, adrenal and pancreas) presented by Dr Mark Peterson. These 3 veterinarians are from U.S.A.

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The South African Veterinary Association looks forward to meeting you in Durban in 1984!

INSTRUMENTATION AND TECHNIQUE OF REMOVAL OF PERMANENT TEETH IN THE DOG

F.J.M. VERSTRAETE*

ABSTRACT: Verstraete F.J.M. **Instrumentation and technique of removal of permanent teeth in the dog.** *Journal of the South African Veterinary Association* (1983) **54** No. 4, 231-238 (En). Department of Surgery, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

A practical method was developed to remove the different permanent teeth of the dog as atraumatically as possible and with a minimal risk of complications. Emphasis is placed on the importance of completely loosening the root from its periodontal attachment and subsequently lifting the tooth out by means of a root elevator, instead of forceful extraction with an extraction forceps. Specific considerations for each tooth are given.

Key words: Dental extraction, dentistry, dog, surgery, tooth.

INTRODUCTION

The extraction of permanent teeth unfortunately still plays a very important role in animal dentistry. Conditions such as transverse fractures with pulp exposure, haematogenous pulpitis and caries can often be treated conservatively but the necessary instrumentation is not often within the scope of a normal veterinary practice[5]. If this is not available, extraction is usually the only acceptable alternative.

Severe periodontitis is the most important indication for extraction. Degeneration of the periodontal membrane as a result of infection is an irreversible process. If one does not remove affected teeth, unnecessary pain will be caused, together with an increased hazard of toxaemia and bacteriaemia[5,12].

Longitudinal fractures, which cannot be treated conservatively, are a less frequent indication for extraction. The involvement of an alveolus in the fracture line of a fractured maxilla or mandible is another indication for an extraction[12].

Extraction of teeth has always been considered as one of the basic surgical skills a veterinarian needs to know. Most classical surgical textbooks, however, give only little attention to it and dentistry in general[13]. It is thus not surprising that the standard of this procedure is often quite low when compared to human dentistry.

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The techniques and instrumentation of human dental extraction are, however, not entirely applicable to canine dentistry. In human dentistry, special extraction forceps are designed for each kind of tooth[2,14]. Most of these forceps are useless and even hazardous if used in the dog. Sharply curved forceps, such as the so-called lower molar forceps and forceps with sharp triangular tips are particularly prone to cause root fracture. Extraction forceps specifically designed for dogs unfortunately are not commercially available.

A practical method has been developed in the Dental Clinic, Department of Surgery, Faculty of Veterinary Science, University of Pretoria to extract the different permanent teeth in the dog as atraumatically as possible and with a minimal risk of fracturing the roots or causing oronasal fistulae. The most suitable instruments from human dentistry were selected (Fig. 2, 3 & 5).

It is unacceptable to leave fractured roots of an extracted tooth behind[12]. Persistent infection, osteitis of the mandible, sinus formation and chronic rhinitis are common sequelae of this malpractice. This complication of dental extraction can, however, not always be avoided, and therefore a method of removing root tips will also be described.

GENERAL PRINCIPLES

Dentistry in the dog should be done under general anaesthesia, preferably inhalational anaesthesia using a cuffed endotracheal tube. The general principles of

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surgery also apply to dental extraction[12]. It is, however, practically impossible to work in a sterile fashion in the oral cavity. Nevertheless one should try to work in as clean a manner as possible. Before extraction of teeth, dental calculus and subgingival debris should be removed by means of ultrasonic and manual scaling and curettage. Subsequent flushing with a mild antiseptic solution is useful[8].

The next step is to loosen the tooth from the surrounding gingiva, that is, to sever the epithelial attachment. In case of gingivitis and periodontitis, the epithelial attachment is already partially or totally destroyed. The gingiva can be detached by introducing a small hand-held scalpel blade (Aesculap No. 15) or a gingivectomy knife between the tooth and the gingiva around the entire circumference of the tooth (Fig. 1 – A).

Multirooted teeth should now be sectioned according to their root configuration (vide infra).

The most important step is to loosen the root of the tooth from the periodontal ligament (Fig. 1 – B). For this a No. 73 and No. 74 Miller Apexo root elevator (Hu-Friedy)* are used (Fig. 2).

The most convenient elevator is gently inserted between the root and the alveolar bone, beginning at the buccal side and progressing more deeply and rostrally. The other elevator is then used to loosen the caudal part. The same process is repeated on the lingual side of the root, until the entire root is loosened from its periodontal attachment. In most cases the tooth can then be lifted out of the alveolus by means of a root elevator. It is not advisable to use the adjacent alveolar bony ridge or tooth as a fulcrum for levering the tooth out. The roots of the teeth of the dog are too long, compared to the diameter of the crown-root interphase, to allow this method.

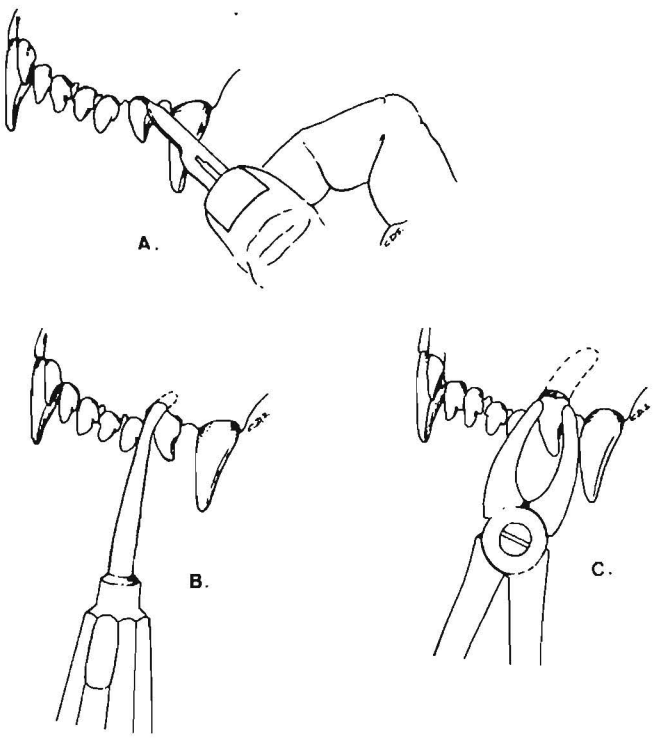

Fig. 1: Extraction of an upper 3rd incisor according to the general principles (see explanation in text).

*Hu-Friedy dental instruments are imported in the Republic of South Africa by E.R. Bernard (Pty) Ltd, Johannesburg.

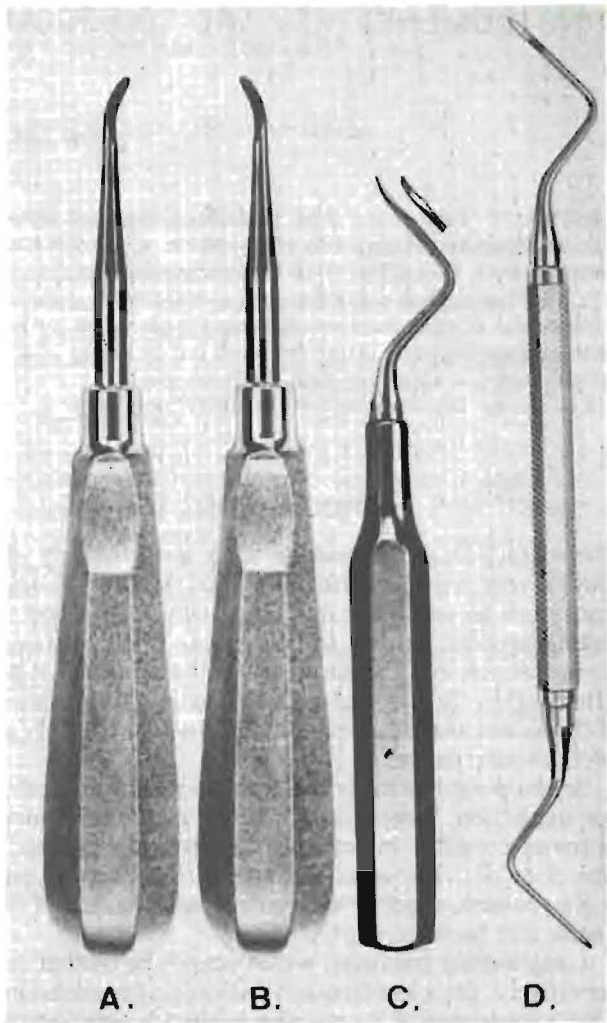

Fig. 2: Root Elevators and Root Tip Elevators
A. Miller Apexo No. 73 Root Elevator
B. Miller Apexo No. 74 Root Elevator
C. Heidbrink No. 4 Root Tip Elevator
D. Heidbrink No. 13/14 Root Tip Elevator

Severing the periodontal attachment completely can be difficult and tedious especially if a healthy tooth is to be extracted. In order to minimize the risk of a root fracture one should always try to loosen the root completely by means of root elevators. If small remnants of periodontal membrane make it difficult to lift the tooth out, an extraction forceps may be used for final loosening and extraction of the tooth. The forceps' grip should be on the proximal part of the root and not on the crown. The shape of the cross-section of that part of the root therefore determines which extraction forceps is to be used. All roots except the mandibular incisors which are mesiodistally flattened, have a round to oval cross-section. The diameter varies from 1-12 mm, depending on the tooth and the size of the dog. We have found that a so-called upper anterior forceps or a slightly curved lower anterior forceps such as the Cryer No. 150 and the Milwaukee No. 3 (Hu-Friedy) are most suitable (Fig. 3 – A, B). These forceps have a slightly conical grip and fit most teeth within 2-10 mm diameter.

Occasionally smaller forceps such as the No. 150S (Hu-Friedy) are used. For extraction of a canine and the buccal roots of the upper 4th premolar in large dogs, a Mead No. 2 forceps (Hu-Friedy) is used (Fig. 3 – D).

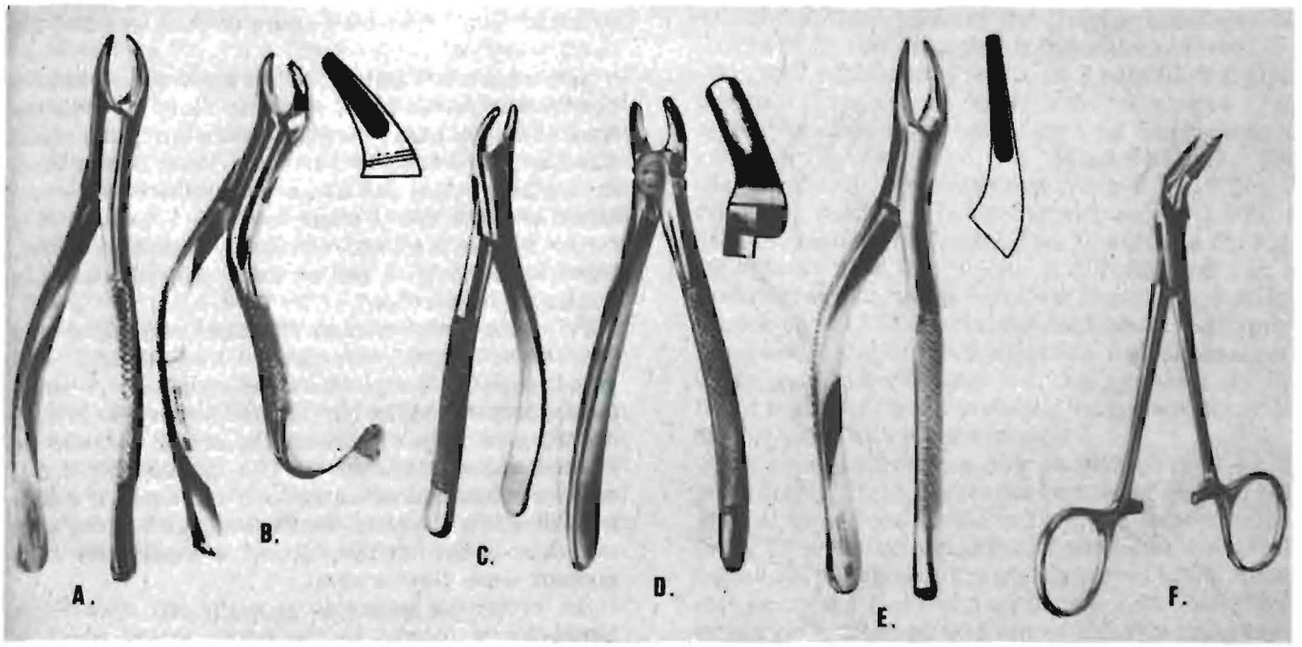

Fig. 3: Extraction Forceps and Root Forceps
A. Cryer No. 150 Extraction Forceps
B. Milwaukee No. 3 Extraction Forceps
C. Cryer No. 150 S Extraction Forceps
D. Mead No. 2 Extraction Forceps
E. Tomes No. 69 Root Forceps
F. Steiglitz (45°) Root Forceps

This is a forceps designed for upper molars in man, but its shape is similar to an upper anterior forceps, except that it is of a heavier pattern and the beaks leave a gap of 3 mm when closed. Under no circumstances should forceps with sharp triangular tips, such as most of the human upper and lower molar forceps, be used, because of the high risk of root fracture. In human oral surgery sharply curved forceps are often used for mandibular teeth[2,14]. These forceps are not convenient if used in the dog. One is often inclined to move them up and down, with the possibility of causing a transverse fracture of the tooth to be extracted.

Once the extraction forceps is securely positioned on the proximal part of the root, one tries to rotate the tooth gently through a very limited angle, clockwise and counter-clockwise, whilst applying very little traction. No attempt should be made to rotate the tooth through an angle of more than 20°. If loosening of the root by means of the root elevators was adequate, it should now be possible to lift out the tooth without undue effort (Fig. 1 – C). If resistance is encountered, it indicates that an important part of the periodontal ligament is still intact. In this case one should not proceed with the forceps extraction, but loosen the root once again by means of root elevators. An alveolotomy can be done if the buccal alveolar ridge prohibits deeper insertion of the root elevator (vide infra).

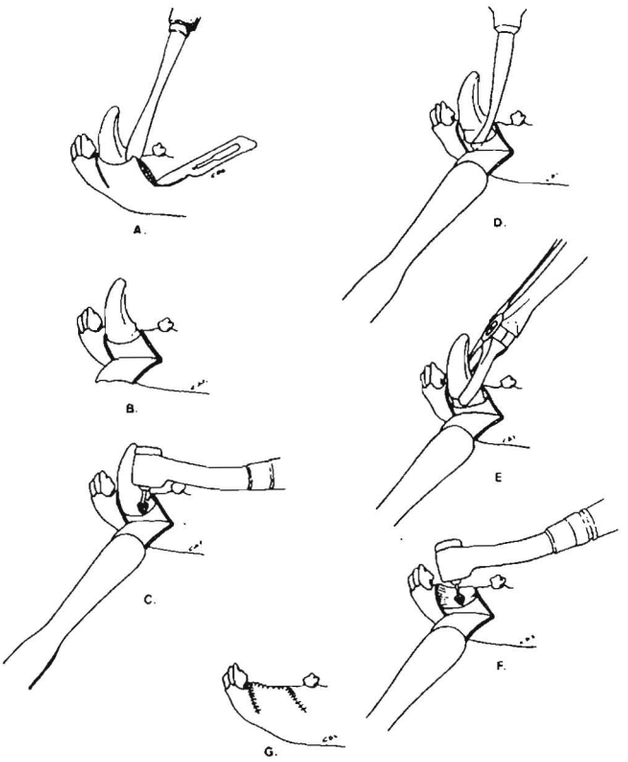

Fig. 4: Extraction of a lower canine (See explanation in text).

After extraction, the root should be inspected. If the apex or an even longer part of the root has broken off, an effort is made to remove it. Larger root tips can be loosened and lifted out by means of the root elevators. Smaller fragments can be loosened by means of a root tip elevator. Currently we use a Heidbrink No. 4 root tip elevator and a Heidbrink No. 13/14 (Hu-Friedy) for very small root tips (Fig. 2). These instruments are used in the same way as the root elevators. Root tip elevators should, however, be used with extreme care on the upper jaw, because they are very sharp and are likely to penetrate the alveolar bone and enter the nasal cavity. Once the root tip has been loosened, it can be lifted out of the alveolus by means of a Tomes No. 69 or a Steiglitz (45°) root forceps (Hu-Friedy) (Fig. 3). The latter is used for very small fragments. If advanced dental equipment is available, a root tip can also be drilled away with a fast cutting dental burr on a high speed handpiece (K. Zetner, Faculty of Veterinary Science, University of Vienna, personal communication). Care should, however, be taken not to damage the alveolar bone excessively.

The root should also be inspected for the presence of remnants of alveolar bone attached to it. This is especially important when extracting an upper canine and also to some extent of an upper 3rd incisor or 4th premolar. The presence of a bony fragment indicates that a piece of alveolar bone has been avulsed. In practice this is only possible on the lingual side of the roots of the abovementioned teeth, because there is a bone plate of only 0,5-1 mm in thickness between these roots and the nasal cavity. If this complication occurs, bleeding will be profuse and must be stopped. This is accomplished by putting a gauze swab in the alveolus for 5 minutes. It can then be removed and replaced by surgical bone wax (Ethicon). Care should be taken not to introduce an excessive amount of bone wax into the nasal cavity.

Severe haemorrhage is not often a problem. It usually occurs after extraction of a healthy tooth or when the nasal cavity has been penetrated. In the latter case, nasal bleeding is evident on the same side. In the former case, the capillary oozing can effectively be controlled by sustained pressure with a gauze swab for 5 minutes. It is rarely necessary to use oxidized cellulose (Surgicel, Ethicon), although it can be safely employed even in conditions of infection[4].

Following the extraction, the empty alveolus should be flushed and inspected. Any debris and granulomatous tissue should be removed. A small curette such as a Miller No. 10 bone curette (Hu-Friedy) can be used (Fig. 5). Ideally the alveolus should be flushed with sterile saline[12,13]. It is questionable if this offers a substantial advantage over tap water. If a dental drill unit is available, the flushing can be done with the three-in-one syringe, giving a moderately high pressure water flow or spray.

An extraction wound is generally left open[5,12]. A blood-clot is formed in the empty socket which is gradually replaced by granulation tissue and covered by epithelium[14]. Wound healing is usually uneventful and rapid in the oral cavity[13]. Most teeth are extracted

Fig. 5: Additional Instruments
A. Miller No. 10 Curette
B. Mead No. 3 Periosteal Elevator
C. Seldin Retractor

because of an infectious process and suturing of the gingiva over the extraction wound is therefore contra-indicated, as this prohibits natural drainage of infected debris left behind. If after extraction, 2 large gingival flaps are present, they can be approximated by 1 or 2 interrupted sutures[5]. This will enhance the healing process. No attempt should be made to apply tension on these sutures. Gingiva is friable tissue and sutures will easily cut through it if postoperative swelling occurs. The suture materials of choice in the oral cavity are polyglycolic acid (Dexon, Davis & Geck), polyglactin 910 (Vicryl, Ethicon) and polydioxanone (PDS, Ethicon), because they are absorbable, resistant to infection and cause minimal tissue reaction[15].

Postoperatively hexitidine spray (Oraldine Spray, Warner) is applied to the oral cavity. In cases of multiple extractions this is prescribed to be applied 4 times daily for the following 3 days. Food should be withheld until the next day. Antibiotics are not routinely given but can be indicated in exceptional cases[16].

SPECIAL CONSIDERATIONS

Incisors

The incisors have single but relatively long roots. Extraction according to the general principles is possible (Fig. 1) and usually easy in cases of periodontitis where there is a very prominent alveolar bone resorption. An extraction forceps is not usually needed and the tooth can be lifted out with the root elevator. Care should be taken not to damage the interdental bone plate, which is very thin mesially.

The upper 3rd incisor has a relatively long, curved root with a triangular cross-section and comes very close to the nasal cavity. These features make a very careful but complete loosening of the tooth by means of the root elevators necessary. Rotational movements are almost impossible.

Canines

A canine is generally difficult to extract because of its extremely long and curved root. In instances of periodontitis there is usually enough alveolar bone resorption on the buccal side to allow for the root elevators to loosen the entire root surface. In a healthy tooth this is not the case and some of the alveolar bone must be removed[13,14]. This procedure is called alveolotomy[2].

On the buccal side of the tooth, 2 parallel or slightly divergent incisions are made into the gingiva (Fig. 4 – A). An electrotome can be used for this purpose to minimize bleeding into the operation field. The mucoperiosteal flap is elevated using a Mead No. 3 periosteal elevator (Hu-Friedy) and reflected with a Seldin retractor (Hu-Friedy) (Fig. 5), exposing the buccal alveolar bone (Fig. 4 – B). If a dental drill unit is available, an osteoplasty burr (Star Dental Diamond Instruments No. F63) can be mounted on the high speed handpiece. Using constant irrigation, the proximal part of the buccal alveolar bone is drilled away (Fig. 4 – C). If this equipment is not available, the bone plate can be nibbled away with a bone rongeur.

The root elevators can now be inserted (Fig. 4 – D) more deeply. If the whole root cannot yet be loosened, more alveolar bone should be removed. It is important to try to sever the periodontal ligament at the apicolingual side of the root. The slightly curved Miller Apexo root elevators (Hu-Friedy) are therefore adequate. Final extraction is achieved with one of the abovementioned forceps (Fig. 4 – E). Because of the oval cross-section of the root, rotational movements are only possible through a very limited angle.

After removal of an upper canine it is very important to check if a communication with the nasal cavity exists evidenced by ipsilateral nose-bleeding. This can be caused by avulsion of a part of the alveolar wall during extraction or by periapical bone destruction due to a chronic inflammatory process. This can lead to a persistent oronasal fistula.

After an uncomplicated extraction surgical bone wax (Ethicon) can be installed in the empty socket to stop capillary oozing and to prevent food from entering. The wound can then be left open or the edges approximated with 1 or 2 simple interrupted sutures. Following an alveolotomy, the gingival edges are more easily approximated and sutured. Care should be taken that no debris is trapped at the base of the flap[13].

If communication with the nasal cavity occurs, more care should be taken to close the wound. After debridement of the alveolus surgical bone wax (Ethicon) is installed. Should an alveolotomy be performed, it is

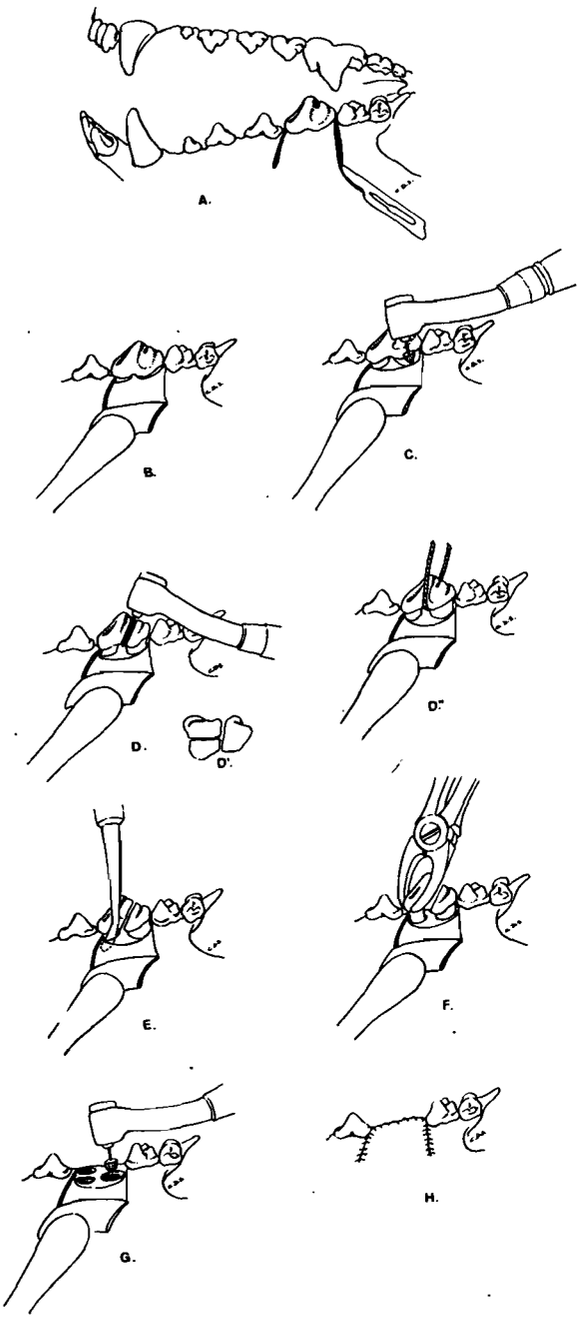

Fig. 6: Extraction of an upper 4th premolar (dog is lying on its back – see explanation in text).

usually possible to close the gingiva over the wound after removing any remaining sharp bone edges (Fig. 4–E-F). If this procedure has not been done, an alveoloplasty can be performed[2,13,14]. Similar to the alveolotomy, 2 parallel incisions are made in the buccal gingiva and a mucoperiosteal flap elevated and reflected. The elevated part of the alveolar ridge is removed with an osteoplasty burr, a bone rongeur or a small file. If a similar ridge is present on the palatal side, it can be removed in the same manner. Afterwards it is usually possible to suture the gingival flaps together.

1st Premolars and mandibular 3rd molar

These small teeth have a relatively short, conical root and are easily removed using the general principles described above.

Two-rooted teeth

These include the upper 2nd and 3rd premolar, the lower 2nd, 3rd and 4th premolar, and the lower 1st and 2nd molar. If, because of periodontitis, important alveolar bone resorption has taken place, the smaller premolars can sometimes be lifted out intact after thorough loosening of the periodontal attachment. In most cases, however, it is necessary to cut the tooth into two parts, thus creating the equivalent of two single-rooted teeth. After loosening the gingiva from the crown and retracting it somewhat, the furcation of the root is visible. If a dental drill unit is available, the tooth can very easily be sectioned using a fast cutting cylindrical diamond burr[13] (Fig. 6–D) (Star Dental Diamond Instruments No. 556-7) on the high speed handpiece or a diamond disk on the straight handpiece[6]. Otherwise a Gigli wire saw (Fig. 6–D''), a tooth splitting forceps, a tooth splitting chisel or a small hacksaw blade can be used[19,14]. The former is certainly the safest.

A large bore hypodermic needle can be inserted under the crown at the furcation of the root and the wire saw introduced through the needle[19]. While the surgeon saws the wire through the crown, an assistant has to fix the jaw and take care that no soft tissue is caught by the wire saw. After the tooth has been sectioned, the 2 parts can be separately extracted using the general principles described above. It is not advisable to use the one half of the tooth as a fulcrum for levering the other out. The tooth roots of a dog are too long to allow this method to be used safely.

The mandibular 1st molar is a very large tooth. In order to achieve a better exposure of the furcation of the root and to be able to insert the root elevators more easily, an alveolotomy can be performed. It is advisable to approximate the wound edges after the extraction. Removal of the elevated alveolar ridge will facilitate this.

Three-rooted teeth

These teeth include the upper 4th premolar and 1st and 2nd molars and are the most difficult to remove, because of their size and/or the poor visibility in the caudal part of the oral cavity.

To achieve maximum visibility, the animal should be put on its back, with the mouth held open by means of an oral speculum. The cuffed endotracheal tube should be fixed to the mandibula. A gauze tampon should be installed in the oropharynx to prevent aspiration of blood and irrigation fluid.

The upper 2nd molar sometimes has fused roots, resulting in one, short, cone-shaped root. In this case it is fairly easy to loosen it and lift it out with a root elevator. If the three roots are present, they are short but quite curved, making this tooth difficult to extract. Because the tooth is so small sectioning by means of a drill or a Gigli wire saw is very difficult. It is more practical to try to remove the tooth intact. Should one of the roots fracture it is removed using a root tip elevator.

The upper 4th premolar and 1st molar are very large teeth, with two long buccal and one shorter palatal root. If no periodontitis is present, it is advisable to remove some of the buccal alveolar bone after a buccal gingival flap has been made[13] (Fig. 6–A-C).

The upper 4th premolar is first sectioned between the 2 buccal roots (Fig. 6–D-D''), and then in between the

rostral buccal root and the palatal root (Fig. 6 – D') in the same way as described for two-rooted teeth. The upper 1st molar is first sectioned between the palatal root and the 2 buccal roots, and then between the 2 buccal roots. Afterwards the 3 fragments can be removed separately, according to the general principles outlined above (Fig. 6 – E-F). Care should be taken not to penetrate the maxillary recess or even the infraorbital canal. If there is a periapical inflammatory process present, a communication with the maxillary recess is very likely.

After the extraction sharp bony edges can be removed (Fig. 6 – G), the alveoli inspected and flushed, and the gingiva approximated or closed (Fig. 6 – H). If the maxillary recess has been entered or haemorrhage is a problem, surgical bone wax (Ethicon) can be used. If the reason for extraction was a periapical abscess causing a sinus tract, it is advisable to avoid the use of bone wax and to leave the extraction wound open in order to promote drainage[1,7,9,14].

DISCUSSION

The use of a root elevator to loosen the tooth prior to final extraction with a forceps is an accepted method in canine oral surgery[1,5]. The emphasis, however, is still on forceful extraction[17]. Mumaw & Miller on the contrary have shown that practically no extraction force is necessary if the periodontal ligament is completely severed[11]. In human dentistry extraction is more acceptable because of the relative shortness of the roots. Even two-rooted teeth can usually be extracted using pendulum movements[2,14]. Kaplan & Jeffcoat[10] have described the extraction of a supernumerary tooth in a dog by rocking the tooth. According to Tholen[12] pushing the tooth buccally and lingually can be used to expand the alveolar bony plates in the dog. It is questionable if this method can safely be used in the dog, because of the length of the dental roots and the hardness of the bone, especially on the mandibula.

Root elevators are often used in human oral surgery as levers, using the adjacent tooth as a fulcrum to lever the tooth out of the socket[2,14]. Tholen[12] has described the use of this method in the dog, even for extraction of two-rooted teeth. The length of the roots necessitates such a deep insertion of the root elevator, that no levering action is actually possible without causing considerable damage to the alveolar bone.

The use of a gingival flap and an alveolotomy to obtain better access to the roots are techniques that are well accepted in human oral surgery[2,14]. In veterinary oral surgery these techniques have not found wide acceptance[1,5]. Tholen[13] has emphasized the usefulness of them for difficult extractions in the dog. According to Frost[7], after an alveolotomy the upper 4th premolar can easily be extracted in one piece. Most authors agree that two- and especially three-rooted teeth should be sectioned before extraction is attempted[1,5,6,9,13]. A small hacksaw blade or a Gigli wire saw are perfectly adequate[1,9]. A circular diamond disk on a straight handpiece[5,6] or a cylindrical burr[7] on a high-speed contra-angle handpiece[13] can more easily be used if a dental drill unit is available. If a large wound is created, closure of the surgical site after thorough debridement of the socket and removal of sharp bony edges will enhance the healing process[13]. Drainage should be ensured if a septic process is present[1,7,9,14].

By adopting certain techniques of human oral surgery it is possible to raise the standard of dental extraction in the dog considerably. The marked anatomical differences between the two species should, however, always be borne in mind.

ACKNOWLEDGEMENTS

Thanks are due to Dr I.E. Gordon, Prof. S.W. Petrick and Mr J.T. Soley for editorial assistance, to Mrs C.D. Janse van Vuren and Mrs H. Smit for the illustrations and to Mrs E. Faber for typing the manuscript.

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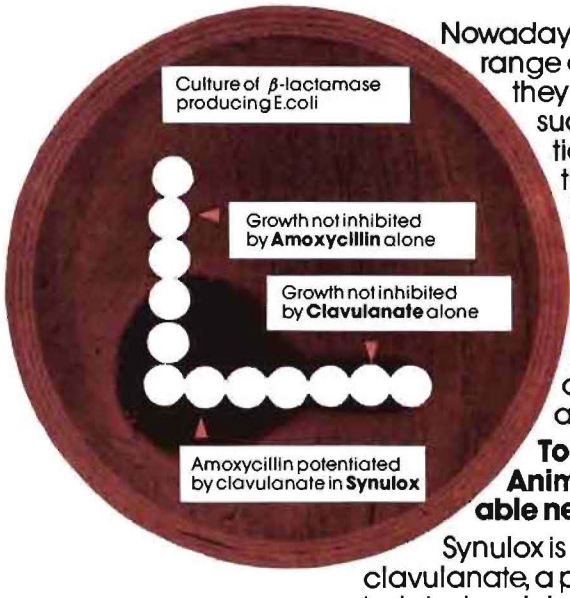

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BOOK REVIEW **BOEKRESENSIE**

ANIMAL HEALTH
HEALTH, DISEASE AND WELFARE OF FARMLIVESTOCK

DAVID SAINSBURY

Granada Publishing Limited. London, Toronto, Sydney and New York 1983, 232, Figs. 64, Tabs 21, Publ. Price R22,75.

The contents of the book are divided into 7 chapters the first of which deals with environmental and husbandry influences on livestock health. It gives basic but sound information on husbandry and housing in disease prevention. The second and third chapters give basic information on the anatomy and physiology of the animal body, a broad description of the organisms responsible for disease and some basic concepts of immunity and disease prevention.

The last 4 chapters deal with some of the disease conditions which occur in cattle, pigs, sheep and poultry.

The level of the information in the book is directed at the farmer or stockman and as such should make a good contribution towards herd health. Naturally a lot of the information given is more applicable to European conditions.

S. van Amstel

BOOK REVIEW **BOEKRESENSIE**

NOTES ON CANINE INTERNAL MEDICINE

P.G.G. DARKE

John Wright & Sons, Bristol, 1983 pp x + 260 Figs 14 ISBN 0 7236 0676 5 Price not mentioned

The book is a brief synopsis of the differential diagnosis, clinical signs, investigations and management of common clinical syndromes of the respiratory, cardiovascular, gastro-intestinal, endocrine and urogenital systems and of the pancreas, liver and spleen of the dog. The differential diagnosis and diagnostic approach to syndromes such as polydypsia, anaemia, thinness, weight loss and recurrent pyrexia are also presented. A small section on autoimmune disorders and acute poisoning is also included.

The text has been written in note form and the brevity in some sections, for example on cardiac dysrhythmias, autoimmune conditions, the investigation of anaemia and acute poisoning may be confusing or misleading. The book should, however, not be regarded as a finite text on medical disorders of the chest and abdomen but merely as an aid in the logical solving of the more common medical problems.

The book has obviously not been written with subtropical conditions in mind and local readers should bear in mind that locally potentially important conditions such as babesiosis, ehrlichiosis and *Spirocerca lupi* infestations are often not included as differential diagnoses.

There are a few printing errors present and it is a little bit disturbing to find more than one on the same page. More spacious presentation of data under some of the subheadings would have facilitated reading thereof.

Notes on Canine Internal Medicine is, however, the type of booklet that I personally would like to see being used by students during their clinical years (in and out of clinics). It could well be an important mechanism in stimulating discussion and further reading by the interested student. The book with its concise text should likewise be a valuable addition to the busy clinician's armament in the solving of medical mysteries.

J. van Heerden

OXYTETRACYCLINE PLASMA CONCENTRATIONS IN SHEEP AFTER THE ADMINISTRATION OF A POLYVINYLPYRROLIDONE FORMULATION

A. IMMELMAN and J.J. VAN RENSBURG*

ABSTRACT: Immelman A.; Van Rensburg J.J. **Oxytetracycline plasma concentrations in sheep after the administration of polyvinylpyrrolidone formulation.** *Journal of the South African Veterinary Association* (1983) **54** No. 4, 241-242 (En). Department of Physiology, Pharmacology and Toxicology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

A polyvinylpyrrolidone formulation of oxytetracycline was injected intramuscularly into 5 sheep at 2 different dosages of 10 mg and 20 mg/kg body mass respectively. Plasma levels were determined. The higher dosage not only attained a higher peak value, but the concentration at 12 hours was 0,5 μg/ml. At the lower dosage rate a level of 0,63 μg/ml was recorded 32 hours after administration when that of the higher dosage rate was 1,5 μg/ml.

Key words: Oxytetracycline, plasma levels, sheep.

INTRODUCTION

In South Africa the tetracycline group of antibiotics is used to control infectious diseases such as heartwater in ruminants[6] and anaplasmosis in cattle[7], as well as other infections caused by tetracycline susceptible organisms. The tetracycline most often used is oxytetracycline and then in various formulations using different vehicles. Propylene glycol (PG) is the most common vehicle utilized in commercially available products. In a few instances polyvinylpyrrolidone (PVP) is used. The latest addition to these formulations is a long-acting formulation incorporating an organic solvent, 2-pyrrolidone which acts by controlling drug precipitation at the injection site[2].

In a comparative study of the lesions caused after an intramuscular injection of the 2 formulations it was shown that the PVP formulation causes less tissue damage than the propylene glycol formulation[4].

It is recorded that very similar plasma concentrations of oxytetracycline were attained in dogs after administration of a long-acting formulation and a PVP formulation when the active material was given at the same dosage level[5].

The purpose of this study is to compare the plasma levels of oxytetracycline after the intramuscular administration of a PVP formulation at 2 different dosage rates in sheep.

MATERIALS AND METHODS

The experimental animals used were 5 Merino x Mutton Merino wethers. Their average mass was 44,7 kg (36,8-52,7 kg) and they were fed lucerne hay, water being freely available. During the trials the animals were kept indoors in individual pens.

The oxytetracycline formulation used was a 10 % solution with polyvinylpyrrolidone as vehicle (Engemycin 10 %, Wellcome). The accuracy of the information supplied on the label was accepted and the dosage calculated accordingly.

In the first trial the drug was administered at a dosage of 10 mg/kg body mass. Double this dose (20 mg/kg) was given in the second experiment which was carried out one week later using the same animals to minimize the possible effect of individual variations that do occur. On both occasions, the injections were given by deep intramuscular injection into the M. gluteus using an 18 gauge needle. The full dose was deposited at one injection site. After the administration the sheep were observed for signs of pain, swelling and discomfort.

Blood specimens were collected from the V. jugularis using heparin as anticoagulant, 0,5, 1, 2, 4, 6, 7,5, 24 and 32 h after each administration. In the second trial further samples were taken in addition at 48, 56, 72, 80 and 96 hours. These samples were centrifuged, the plasma collected and stored at 4°C until they were assayed within 48 h of collection. A fluorometric method with a senstivity of 0,1 μg/ml was employed for the oxytetracycline assay[3].

RESULTS

No pain, swelling or any discomfort was observed in the sheep after administration of the drug in either of the trials.

The mean plasma concentrations of oxytetracycline attained in the 5 experimental animals after the different dosages are given in Fig. 1.

In the first trial (10 mg/kg) the plasma concentration was 1,05 μg/ml when the first specimens were collected 30 min after administration. The peak concentration of 4,02 μg/ml was reached 4 h after the onset of this study. The 1 μg/ml level was maintained for 24 h. When the last specimens were collected after 32 h the plasma level of oxytetracycline was 0,63 μg/ml.

The higher dosage in the second study resulted in a much higher plasma concentration of oxytetracycline. Within 30 minutes the concentration was 2,6 μg/ml and 2 h after administration the peak level of 6,04 μg/ml was reached. After 48 h the concentration had fallen to 0,89 μg/ml and the 0,5 μg/ml level was reached 72 h after administration.

DISCUSSION

The use of a PVP formulation in dogs can lead to side effects as a result of histamine release. The higher dosage (20 mg/kg) is therefore not recommended for dogs[5]. In this study on sheep, however, no adverse reactions were recorded at the higher dosage level.

The higher dosage not only resulted in a higher peak

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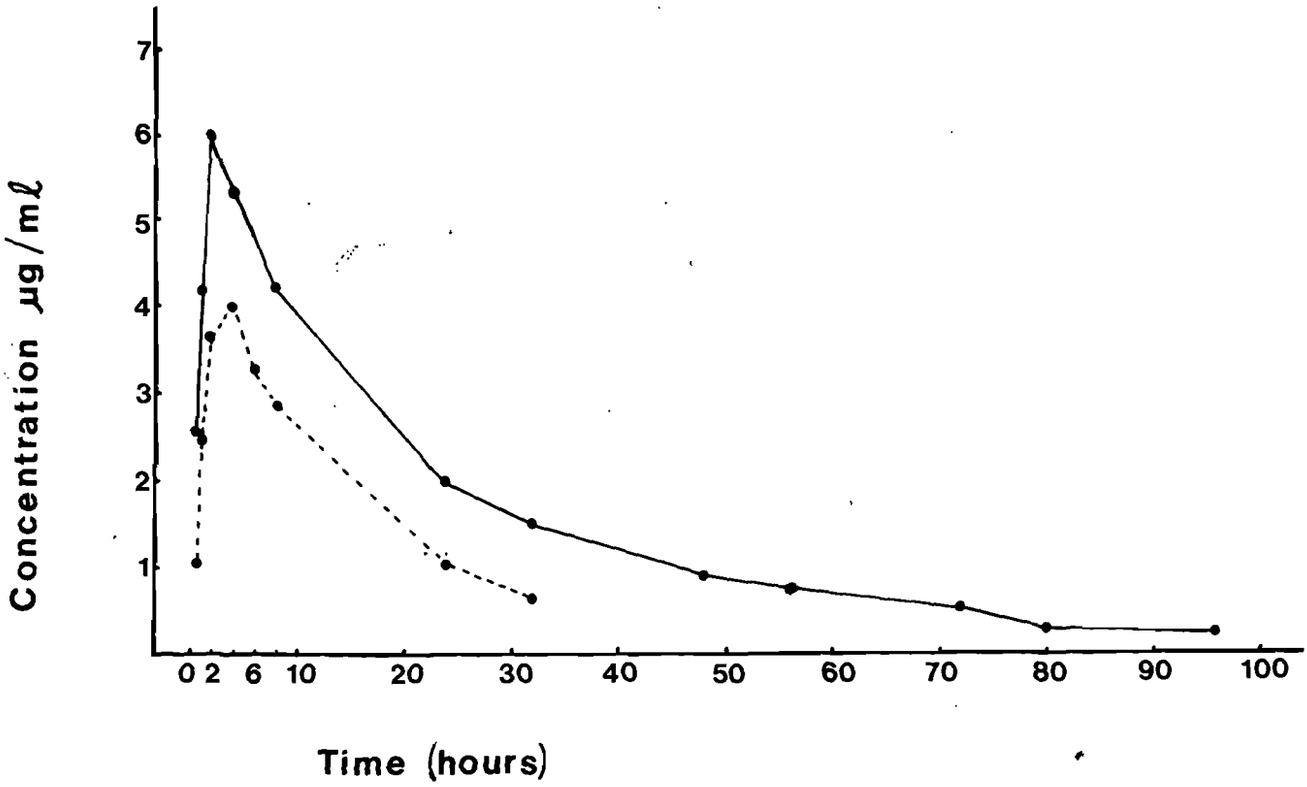

Fig. 1. The mean oxytetracycline plasma concentrations in 5 sheep after the intramuscular administration of 10 mg/kg (- - - - -) and 20 mg/kg body mass (———) respectively.

plasma concentration of oxytetracycline, but higher levels were also maintained over a longer period. It is generally accepted that 0,5-1 µg/ml is the minimum inhibitory concentration required for most common bacterial infections[1]. This would mean that the higher dosage maintained effective levels for up to 72 h. Using the standard 10 mg/kg body mass dose, effective levels were maintained up to the point when the experiment was terminated (32 h).

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We want to express our appreciation towards the pharmaceutical company, Wellcome, for supplying the drug and for financial assistance.

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THE EFFECT OF LINCOMYCIN-NEOMYCIN TREATMENT ON EXPERIMENTAL ANAEROBIC BACTERIAL BOVINE MASTITIS

J.H. DU PREEZ*, A.S. GREEFF** and UTE KRAFT***

ABSTRACT: Du Preez J.H.; Greef A.S.; Ute Kraft. **The effect of lincomycin-neomycin treatment on experimental anaerobic bacterial bovine mastitis.** *Journal of the South African Veterinary Association* (1983) **54** No. 4, 243-245 (En). Department of Pathology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

Three healthy lactating quarters of a Friesland cow were each experimentally infected with a pure culture of a strain of either *Bacteroides fragilis*, *Eubacterium lentum* or a *Peptostreptococcus* sp. respectively. The onset and progression to clinical mastitis was monitored 12 hourly by examination for clinical signs of inflammation, bacterial culture, somatic cell counts and with a strip cup. All infected quarters developed clinical mastitis within 24 hours. The 2 quarters infected with *B. fragilis* and *E. lentum* respectively were treated 4 times consecutively at 12 hour intervals, commencing at 24 h by intramammary instillation of 10 ml of a mixture containing 200 mg lincomycin hydrochloride, 200 mg neomycin sulphate and 5 mg methylprednisolone (Lincocin Forte, Upjohn). Both quarters became clinically normal and no bacteria could be detected in the secretions 12 hours after the first treatment. At 36 hours the strip cup became negative, and the somatic cell count dropped to $< 500 \times 10^3$ at 72 hours after the initial treatment. The quarter infected with a *Peptostreptococcus* sp. was unable to overcome the infection by natural means when intramammary treatment was delayed for the first 36 hours after the onset of clinical mastitis. Subsequent treatment of this quarter gave results similar to those treated earlier.

Key words: Experimental bovine mastitis, anaerobic bacteria, lincomycin-neomycin treatment.

INTRODUCTION

Several reports have incriminated a variety of non-sporulating anaerobic bacterial species in the aetiology of bovine mastitis[3,6,12,13,14].

Isolation rates for these organisms from cases of clinical mastitis varies from 7,4 % (treatment before sampling not excluded)[3] to 58,8 % (treatment excluded)[6]. Greeff et al.[5] and Du Preez et al.[3] have shown that clinical mastitis could be induced in lactating quarters by experimental infusion with pure cultures of several anaerobic bacterial species. Histopathological evidence indicates that these organisms are capable of acting as primary pathogens under experimental conditions[4].

Their significant presence in cases of clinical mastitis poses the question of their susceptibility to antimicrobial treatment. Although the majority of Gram positive strains are susceptible to penicillin-based antibiotics, the more frequently isolated Gram negative rods are resistant[3,6]. Anaerobic bacteria are also mostly refractive to aminoglycoside antibiotics and tetracyclines[15].

Lincomycin has been widely reported for its activity against a broad range of non-clostridial anaerobic bacteria including Gram negative rods[10,11,15]. It is also highly active against mastitogenic strains of *Staphylococcus aureus* and *Streptococcus* spp.[2]. With the exception of chloramphenicol-containing preparations, lincomycin in combination with neomycin appears to be the only antibiotic presently available in South Africa as an intramammary preparation with activity against a wide range of a sporogenous anaerobes[8].

Our experience in the use of lincomycin combined with neomycin for the treatment of mastitis experimentally induced by *Bacteroides fragilis*, *Eubacterium lentum* and a *Peptostreptococcus* sp. is reported here.

MATERIALS AND METHODS

Mastitis Induction

A lactating Friesland cow with quarters judged healthy according to criteria of the International Dairy Federation[9] was used for the induction of mastitis. One ml of either *Bacteroides fragilis*, *Eubacterium lentum* or a *Peptostreptococcus* sp., grown to a density of ± 2-6 x 10^6/ml colony forming units in peptone yeast extract glucose broth, was infused separately via the teat canal into the gland cistern of each of 3 quarters under strictly anaerobic conditions[3]. The onset and development of mastitis was monitored 12 hourly for 5 days, and thereafter on Days 7, 10 and 15 by standard clinical and cytobacteriological procedures[9].

Bacteriology

The 3 strains of anaerobic bacteria employed for the induction of mastitis had all been isolated from cases of clinical mastitis. Some of their relevant characteristics are listed in Table 1. The methods for sampling, cultivation and identification of anaerobic and other bacteria as well as the determination of antimicrobial sensitivity and β-lactamase production has been described[1,3,7]. Sampling always took place immediately before the application of therapy.

Table 1: RELEVANT CHARACTERISTICS OF ANAEROBIC BACTERIA USED FOR MASTITIS INDUCTION

Bacterial Isolate	Sensitivity to Antibacterial Agents					Production of β-lactamase
	PEN	CHL	LIN	NEO	MET	
B. fragilis	–	+	+	–	+	+
E. lentum	+	+	+	+	+	ND
Peptostreptococcus sp.	+	+	+	+	+	ND

PEN = Penicillin G
CHL = Chloramphenicol
MET = Metronidazole
\+ = Sensitive
LIN = Lincomycin
NEO = Neomycin
– = Resistant
ND = Not determined

Antimicrobial therapy

Treatment consisted of the intramammary instillation of the contents of a 10 ml syringe of "Lincocin Forte", Upjohn (containing 200 mg lincomycin hydrochloride

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Table 2: THE EFFECT OF 'LINCOCIN FORTE' TREATMENT ON THE CLINICAL AND CYTO-BACTERIOLOGICAL PARAMETERS OF MASTITIS INDUCED BY ANAEROBIC BACTERIA

Time After Mastitis Induction	Quarter	Bacteria Present		Clinical Signs	Strip cup	Somatic cell count x 10^3	Therapy
		Anaerobes	Other				
0 h	LF	None	None	Normal	–	105	None
	RF	None	None	Normal	–	430	None
	LH	None	None	Normal	–	210	None
	RH	None	None	Normal	–	105	None
12 h	LF	None (Control)	None	Normal	–	210	None
	RF	*B. fragilis*	None	Normal	–	>5000	None
	LH	*E. lentum*	None	Milk yellow	–	>5000	None
	RH	*Peptostreptococcus* sp.	None	Normal	–	>5000	None
24 h	LF	None (Control)	None	Normal	–	105	None
	RF	*B. fragilis*	None	Pain	++	>5000	LNM
	LH	*E. Lentum*	None	Pain	++	>5000	LNM
	RH	*Peptostreptococcus* sp.	None	Pain	++	>5000	None
36 h	LF	None (Control)	None	Normal	–	65	None
	RF	None	None	Normal	+	>5000	LNM
	LH	None	None	Normal	+	>5000	LNM
	RF	*Peptostreptococcus* sp.	None	Pain, swolen	++	>5000	None
48 h	LF	None (Control)	None	Normal	–	105	None
	RF	None	None	Normal	+	>5000	LNM
	LH	None	None	Normal	+	>5000	LNM
	RH	*Peptostreptococcus* sp.	None	Pain, swolen	++	>5000	None
60 h	LF	None (Control)	None	Normal	–	105	None
	RF	None	None	Normal	–	1950	LNM
	LF	None	None	Normal	–	2245	LNM
	RH	*Peptostreptococcus* sp.	None	Pain, swolen	++	2455	LNM
72 h	LF	None (Control)	None	Normal	–	65	None
	RF	None	None	Normal	–	2860	None
	LH	None	None	Normal	–	2600	None
	RH	None	None	Little pain	+	3770	LNM
84 h	LF	None (Control)	None	Normal	–	65	None
	RF	None	None	Normal	–	125	None
	LH	None	None	Normal	–	1755	None
	RH	None	None	Normal	+	2715	LNM
96 h	LF	None (Control)	None	Normal	–	65	None
	RF	None	None	Normal	–	430	None
	LH	None	None	Normal	–	430	None
	RH	None	None	Normal	+	2860	LNM
108 h	LF	None (Control)	None	Normal	–	65	None
	RF	None	None	Normal	–	430	None
	LH	None	None	Normal	–	105	None
	RH	None	None	Normal	–	1755	None
120 h	LF	None (Control)	None	Normal	–	105	None
	RF	None	None	Normal	–	65	None
	LH	None	None	Normal	–	105	None
	RH	None	None	Normal	–	1255	None
Day 7	LF	None (Control)	None	Normal	–	105	None
	RF	None	None	Normal	–	105	None
	LF	None	None	Normal	–	65	None
	RF	None	None	Normal	–	430	None
Day 10	LF	None (Control)	None	Normal	–	65	None
	RF	None	None	Normal	–	105	None
	LH	None	None	Normal	–	65	None
	RH	None	None	Normal	–	430	None
Day 15	LF	None (Control)	None	Normal	–	105	None
	RF	None	None	Normal	–	105	None
	LH	None	None	Normal	–	65	None
	RH	None	None	Normal	–	430	None

LF = Left front LH = Left hind ++ = Strong positive – = Negative
RF = Right front RH = Right hind + = Positive (Flakes) LNM = lincomycin-neomycin-methylprednisolone

monohydrate, 200 mg neomycin sulphate and 5 mg methylprednisolone), as required.

Treatment of those quarters infected with *B. fragilis* and *E. lentum* respectively commenced at 24 h and was repeated 4 times consecutively at 12 h intervals.

RESULTS

The relevant data obtained under experimental conditions of mastitis induction and subsequent therapy with "Lincocin Forte" is reported in Table 2. From this it is evident that clinical mastitis resulted in each quarter within 24 h after infusion with anaerobic bacteria. Somatic cell counts in milk from the 3 infected quarters however exceeded 5 x 10^6 cells per ml at 12 h.

At 36 h, 12 hours after the first dose of "Lincocin-Forte", the quarters infected with *B. fragilis* and *E. lentum* became bacteriologically negative and clinical signs disappeared. There was no change in these parameters during the 15 day monitoring period. Thirty-six hours after initial treatment the strip cup became negative but the somatic cell count remained high until it dropped to < 500 x 10^3 cells/ml, 72 hours after commencement of treatment. It subsequently stabilised around 100 x 10^3 cells/ml.

Although the quarter infected with a *Peptostreptococcus* sp. also showed signs of overt clinical mastitis at 24 h, treatment was withheld for a further 36 hours in an attempt to establish whether the infection could be overcome by natural host defence mechanisms. Since this could not be accomplished within 36 hours, treatment with 'Lincocin Forte' was instituted at 60 h according to the same schedule as that applied to the other 2 quarters. Twelve hours after the first treatment no bacteria could be isolated and the clinical signs improved (little pain, not swollen), the quarter became normal after 24 hours. The strip cup became negative after 48 hours and 72 hours after commencement of therapy, the somatic cell count had dropped to < 500 x 10^3 cells/ml.

Throughout the monitoring period the uninfected quarter remained normal.

DISCUSSION

Effective control over the infections established by the different anaerobic bacterial species was accomplished. Therapy resulted in the rapid disappearance of the bacteria from the secretions of all the infected quarters within 12 hours after the first 10 ml dose of "Lincocin Forte". This may be particularly significant in the case of the quarter infected with a *Peptostreptococcus* sp. This quarter was left untreated for 36 hours after the establishment of clinical mastitis. This allowed more time for the colonization and establishment of the pathogen since no signs of effective host resistance to the infection could be detected. Subsequent treatment of this quarter resulted in normalization of all the parameters monitored and these results were comparable to the quarters treated sooner. These results thus provide a basis for evaluating "Lincocin Forte" for its efficacy in eliminating pathogenic anaerobic infections of the bovine udder under field conditions. The polymicrobial nature of udder infections however often involves multiple anaerobic as well as facultative species together[6]. Many anaerobic species, noteably *B. fragilis*, are resistant to commonly employed antibiotics. These factors therefore may very well influence the efficacy of a single drug being used in the field.

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FITTING THE VET FOR TODAY'S VITAL TASKS

Leslie Cottington, agricultural writer and broadcaster for the BBC

Famed as they are for their love of animals, even the British might be surprised to hear that the academic standards required of those entering their country's veterinary schools are now higher than those for any other university undergraduate course, including medicine for humans.

The veterinary surgeon – or vet – is seen in a multitude of different roles. Most families first meet him when he treats the dog or cat, acts as manicurist for the budgie, or assists the goldfish in its watery environment. But that is only part of a vet's job. Much of his time is spent combating diseases and treating casualties among farm animals.

Vets also try to prevent disease ever breaking out on the nation's farms. By doing so, they ensure that prime breeding stock purchased by overseas buyers can be safeguarded against the most insidious ailments. Some vets are concerned with the specialised area of fish farming. And horses, whether the child's first pony or the bluest of blue-blooded thoroughbreds, fall within the veterinary surgeon's province.

Yet other vets provide facilities for diagnosing disease problems and thus give a back-up service to practising veterinary surgeons in the front line of disease control.

Many more are involved with the various pharmaceutical companies undertaking research into new drugs. In short, a wide range of career opportunities exists within the veterinary profession, and all need a sound training base.

Fierce Competition

University training of future veterinary surgeons is undertaken at 6 British veterinary colleges – 4 in England and 2 in Scotland. Each year, about 350 new graduates enter the list of registered vets, a list that runs to 10 500 names.

Competition for entry is fierce. Entry requirements vary, but all demand good passes in a range of subjects taken at the Ordinary level of the General Certificate of Education examination or its Scottish equivalent – examinations which pupils usually sit at the age of 16. In addition, at least 3 prescribed science subjects have to be passed at Advanced level examinations taken 2 years later. The prescribed sciences are usually chemistry, physics and biology, and often candidates must get top grades in 2 of these and a good average in the third.

The courses normally followed by successful entrants last 5 years. The standard today is far removed from that of the era portrayed by the vet-turned-novelist James Herriot, who is now in his late sixties, and is that demanded of a highly trained scientist. A study of anatomy will show the budding vet the relationship of the organs within the body; their microscopic structure and arrangement with that of the tissues is dealt with in histology.

Physiology relates the normal functions of the body to their structure and this is combined with a course in biochemistry, which explains how various reactions occur within the body's cells. The action of drugs is dealt with in pharmacology, and the normal development of the animal before birth is studied in embryology.

In time, the student is instructed in the recognition of diseased organs and the various viruses, bacteria, parasites and toxic agents that do the damage. Animal husbandry comes last, including the relationship between the domestic animal and its environment. And, of course the practical subjects of surgical procedure – gynaecology, obstetrics and anaesthesia in surgery – are also included.

The Final Hurdle

At various stages during the course, university examinations have to be taken and passed. These culminate in the final degree examination. The degrees awarded vary between the 6 veterinary schools and are differently titled. The full cost of the course is about £6 280, excluding any student loans or grants.

Tony Andrews, who lectures at the Royal Veterinay College is London, says: "We are not getting as many overseas students as we would like, and that is a great shame because many of our courses are aimed at people from overseas. Those who have been are more than satisfied. Governments of countries as far apart as Nigeria and Malaysia have made that clear by regularly adding names to our course lists."

Wide Choice

No specialisation is possible in the undergraduate course, but subsequent studies are tailored to meet the many needs of veterinary medicine today. At present all post-graduate courses are being costed at the full rate of £6 600.

The choice is wide. For example, Bristol offers a course on meat science, and Liverpool runs an MSc in avian medicine and applied parasitology. Edinburgh University's Royal (Dick) School provides a diploma/MSc course in tropical veterinary medicine, animal health, tropical animal production and health, diagnostic veterinary pathology, neuro-physiology, tropical veterinary science and veterinary public health.

There are many more post-graduate courses, but just as important is the fact that training for doctorate degrees, especially for the Doctor of Philosophy, is available from all 6 colleges in all branches of the veterinary discipline. Such training is considered by many to be the best in the world and helps to keep Britain in the forefront of international research.

Schemes exist which provide financial assistance to enable a limited number of students to come to Britain. Details of them are available from local British Council Offices or, where there is no British Council office, from the British Embassy or High Commission.

Veterinary medicine in Britain has come a long way since the first veterinary college was founded in London in 1791. Curiously, the founder was a Frenchman. If that seemed odd, other happenings in the veterinary world at the time were no less unusual. While today's vet will inject calcium into the dairy cow suffering from milk fever, the accepted practice then was to inflate the cow's udder using a bicycle pump. Such bizarre and unscientific treatments are happily a thing of the past.

PIGEON HERPESVIRUS CONFIRMED IN SOUTH AFRICA

B. POLLARD* and ENSLIE J. MARAIS**

ABSTRACT: Pollard B.; Marais J. **Pigeon herpesvirus confirmed in South Africa.** *Journal of the South African Veterinary Association* (1983) **54** No. 4, 247-248 (En). Poultry Section, Veterinary Research Institute P.O. Box 12502, 0110 Onderstepoort, Republic of South Africa.

The history, geographical distribution, clinical signs, pathology and virology of pigeon herpesvirus infection and pigeon herpesvirus encephalomyelitis are briefly reviewed. A case of pigeon herpesvirus infection was diagnosed on clinical, macro- and histopathological appearance and confirmed by isolation of the virus in embryonated eggs, its growth in tissue culture, and by electron microscopy.

Key words: herpesvirus, pigeon, encephalomyelitis.

INTRODUCTION

To the best of our knowledge, the earliest reference to an inclusion body disease in pigeons was recorded by Smadel et al.[17] in the United States of America in 1943, where it was initially diagnosed as ornithosis (psittacosis). Later investigations by Smadel et al.[16] in 1945 showed that an unknown virus causing intranuclear inclusions and necrosis of parenchymatous organs was a separate entity and immunologically distinct from the ornithosis agent and that they frequently occurred together.

In Denmark, Marthedal & Jylling[13] describe motality in pigeons associated with a diptheroid necrotising oesophagitis, inflammation of parenchymatous organs and pneumonia with the presence of intranuclear inclusion bodies at the same sites.

In Scotland, Cornwell et al.[5] observed a syndrome characterised by conjunctivitis, rhinitis, dyspnoea, general malaise and weakness with foetal hepatic necrosis associated with intranuclear inclusions. The causative agent was identified as a herpesvirus.

Similar syndromes have been described in the United States of America[12], Czechoslovakia[18], Australia[3 4 19], New Zealand[22] and Belgium[25].

The clinical signs and lesions described are highly variable but typically the disease affects birds less than 6 months of age[13] and may occur as chronic sporadic losses[22] or as acute enzootic outbreaks[5]. Clinical signs may include anorexia, general malaise, weakness, conjunctivitis, rhinitis, nasal discharge and necrotic stomato-laryngopharyngitis with the development of yellowish exudate in the oral cavity[4 5 13 22]. Post mortem findings include hepatomegaly with focal necrotic, sometimes haemorrhagic hepatitis and pancreatitis associated with basophilic and/or eosinophilic intranuclear inclusion bodies. Other findings are interstitial nephritis, lienitis, tracheitis and interstitial pneumonia as well as inflammation with exudate formation in the oesophagus and crop[4 6 7 11 24].

Inoculation of specimens onto the chorio-allantoic membrane (CAM) of embryonated eggs results in plaques from 1-2 mm in diameter on the CAM and skin of the embryo with extensive necrosis of various body organs. Embryonic death occurs within 4-9 days[4 8 11], but intranuclear inclusion bodies, most abundant on the second day post inoculation, decrease in number until the fifth day when they are almost absent[4].

In tissue culture the virus is cytopathogenic in chick embryo fibroblasts, duck embryo liver cells and chick kidney cells as well as baby hamster kidney cell cultures. Cytopathic effects in chick embryo liver cell cultures consist of rounding of cells with rapid detachment of the monolayer, producing a clear central zone[9 23 24].

In addition to the syndrome described above, a nervous disease of pigeons attributable to a herpesvirus has been described. At this stage its relationship to the aforementioned disease is still unclear and thus it is also discussed here. The syndrome is characterised by ataxia, torticollis, head circling, muscle tremors, inability to fly or walk normally, greenish diarrhoea and progressive paralysis of legs and wings[1 14 15].

First described in the U.S.A. in 1953 by Dougherty & Saunders[10], it was referred to as pigeon meningoencephalitis. The disease was self-limiting, lasting only 3 months, and had low mortality but no definitive infectious agent could be isolated. Thompson et al.[22] in describing herpesvirus infection of pigeons noted a mild demyelination of the cerebellum with degeneration of Purkinje cells, but did not comment on its significance. In Iraq, a contagious paralysis associated with meningoencephalitis[15] and viral encephalomyelitis syndrome giving a similar clinical, pathological and laboratory picture[2 20 21] have been described. These syndromes differed significantly from that described by Dougherty & Saunders[10] with respect to the very high mortality, contagiousness and persistence over a period of a year[15]. The causative virus was identified tentatively as a herpesvirus[15 20 21], which although similar to the pigeon herpesvirus, differed at least with respect to its pathogenicity in pigeons.

CASE HISTORY AND CLINICAL FINDINGS

A single pigeon was submitted from a flock of 60 of which 4 had already died over a 4-6 week period. All the pigeons that had died had been purchased and introduced into the flock from another source. The birds showed general malaise, anorexia, mild rhinitis, râles and the accumulation of a yellowish exudate in the oral cavity and pharyngeal area. The time from first observing the bird as depressed until death was about 4-6 days.

PATHOLOGY

The necropsy revealed a mild inflammation of the oral cavity, pharynx, oesophagus and crop with the ac-

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cumulation of whitish exudate in the pharynx and crop. A smear of crop contents was negative for *Trichomonas gallinae*. The liver was enlarged and pale with white foci 0,2 mm in diameter distributed throughout.

Histopathology of the liver showed a focal necrotic hepatitis with the presence of basophilic and eosinophilic intranuclear inclusions. The crop showed epithelial degeneration with vacuolisation and cellular disintegration. Cellular debris, macrophages and heterophils formed a layer above the lesions. In some areas the necrosis extended throughout the entire depth of the epithelium to form small ulcers. Intranuclear inclusions were present in epithelial cells in and around the lesions.

VIROLOGY

Inoculation of ground pigeon liver suspension onto the chorioallantoic membrane of 9 day-old embryonated eggs produced small pock-like lesions on the CAM. A subsequent second passage of ground CAM resulted in Embryonic death 5-6 days post inoculation. The CAM showed pock-like plaques, 1 mm in diameter with thickening of the CAM between plaques.

The isolate was cytopathogenic in chicken embryo liver cell cultures causing degeneration and destruction of the monolayer within 3 days. The isolate also grew in VERO* cell cultures causing rounding up of cells, detachment within 48 h and degeneration of monolayers within 4 days.

Electron microscopy of crop epithelium and liver revealed viral particles with icosahedral symmetry, some of which were enveloped. Unenveloped particles measured 100 nm and enveloped particles 140 nm in diameter.

DISCUSSION

The history, clinical symptoms, pathology and virology of this disease outbreak are consistent with that of pigeon herpesvirus infection (PHV). Although PHV has been diagnosed on histopathology and an agent causing pocks on the CAM of embryonated eggs was isolated as long ago as 1973 (S.B. Buys and L. Coetzee 1982 Veterinary Research Institute, Onderstepoort, personal communication) and 7 suspected cases have been diagnosed in this laboratory on the evidence of hepatic intranuclear inclusion over the period 1980-1982 (B. Pollard, J. Marais and P.D. Barnes 1982, Veterinary Research Institute, Onderstepoort, unpublished work) this is, to the best of our knowledge, the first time PHV has been reported and confirmed in South Africa.

Further characterisation of both the PHV and the pigeon herpes encephalomyelitis virus as well as serological and further tissue culture research are required in order to examine their relationship to one another, and to provide information on distribution and frequency of occurrence.

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*African green monkey kidney cells.

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PERSISTENT ANTHELMINTIC EFFECT OF IVERMECTIN IN CATTLE

G.E. SWAN and R.G. HARVEY*

ABSTRACT: Swan G.E.; Harvey R.G. **Persistent anthelmintic effect of ivermectin in cattle.** *Journal of the South African Veterinary Association* (1983) **54** No. 4, 249-250 (En). MSD Research Centre, Private Bag 3, 1685 Halfway House, Republic of South Africa.

The persistent anthelmintic effect of ivermectin given subcutaneously at 200 mcg/kg was evaluated against induced infections of *Haemonchus placei*, *Ostertagia ostertagi*, *Cooperia* spp. (*C. pectinata* and *C. punctata*), *Bunostomum phlebotomum* and *Oesophagostomum radiatum* in cattle. Forty-four Friesian bull calves raised under worm-free conditions were restrictively randomized to one untreated control group and 3 ivermectin treated groups of equal size according to mass. Animals in the different treated groups were treated either 9, 7 or 5 d before infestation, which was induced in all animals on the same day. The results are presented as percentage reduction and Non Parametric claims. Nine days after treatment the effect of ivermectin was virtually undiminished against *O. ostertagi* and *B. phlebotomum* and 7 d after treatment against *Cooperia* spp. Counts of all worms were reduced by 99 % or more following the treatment given 5 d before infection. According to the Non Parametric Method, "A" claims (i.e. 80 % effective in 80 % of the treated animals) were achieved against all 5 worms up to 7 d after treatment and against *O. ostertagi* and *B. phlebotomum* up to 9 d after treatment.

Key words: Persistent anthelmintic effect, ivermectin, *Haemonchus placei*, *Ostertagia ostertagi*, *Cooperia pectinata*, *Cooperia punctata*, *Bunostomum phlebotomum*.

INTRODUCTION

The broad spectrum antiparasitic effects of ivermectin have been documented in many publications[4,5]. It has been noted that the effect of ivermectin against ectoparasites persists for at least some days after the medication is given[1,6]. Subsequently, Bremner et al.[3] and Barth[2] reported that the persistent activity is also effective against nematodes. This paper describes the results of a trial in South Africa confirming this persistent anthelmintic activity.

MATERIALS AND METHODS

Forty-four Friesian bull calves raised under worm-free conditions were ranked according to weight and 11 replicates formed. Within each replicate the animals were then randomly allocated to one of 4 treatment groups. Infestations were induced in all on the same day by administration of infective larvae. *Bunostomum phlebotomum* larvae were applied dermally. A mixture of *Haemonchus placei*, *Ostertagia ostertagi*, *Cooperia* spp. (*C. pectinata* and *C. punctata*) and *Oesophagostomum radiatum* was given orally on filter paper discs in gelatin capsules.

Group 1 was unmedicated. The animals in Group 2 were given 200 mcg/kg (the recommended level) of ivermectin (Ivomec, Merck & Co., Inc.) by subcutaneous injection 9 d before infestation with the larvae while those in Groups 3 and 4 were given the same medication 7 d and 5 d resp. before the larvae were administered.

The animals were slaughtered and processed for worm recovery 26 – 30 d after the nematode infestation had been induced.

The worm burdens of the different treatment groups were ranked separately for each parasite and the efficacy determined by the modified Non Parametric Method (NPM)[7].

RESULTS

Percentage reduction and NPM efficacy claims were as follows:

	200 mcg/kg of ivermectin number of days before infection			
	Unmedicated*	5 days	7 days	9 days
H. placei	1122.7 ± 389.1	99.6(A)**	84.9(A)	37.2(X)
O. ostertagi	916.4 ± 386.7	100. (A)	99.8(A)	95.8(A)
Cooperia spp.	1622.7 ± 444.7	99.9(A)	99.6(A)	87.8(B)
O. radiatum	996.8 ± 348.2	99.9(A)	95.1(A)	73.3(C)
B. phlebotomum	319.1 ± 171.8	100. (A)	95.5(A)	97.0(A)

*Arithmetic mean of worm counts ± standard deviation.
**NPM efficacy claim given in parenthesis.
A = More than 80 % effective in more than 80 % of treated cattle.
B = More than 60 % effective in more than 60 % of treated cattle.
C = More than 50 % effective in more than 50 % of treated cattle.
X = Ineffective.

According to the NPM, "A" claims (i.e. 80 % effective in 80 % of the treated animals) were obtained against all worms up to 7 d after treatment, as well as against *O. ostertagi* and *B. phlebotomum* at 9 d after treatment.

DISCUSSION

It is evident that in this trial the effect of the ivermectin was virtually undiminished 9 days after administration against infections of *O. ostertagi* and *B. phlebotomum*, and 7 days after treatment against infections of *Cooperia* spp. Counts of all worms were reduced by 99 % or more following the treatment given 5 days before infestation.

As has been mentioned by Barth[2], such persistent activity could be exploited by using treated cattle to reduce populations of infective larvae on pasture. This should have effects much more far-reaching than is usually expected from treatment of an established infestation.

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BOOK REVIEW

BOEKRESENSIE

KEEP YOUR PIGEONS FLYING

LEON F. WHITNEY

2nd ed. Faber and Faber, London. 1983. pp. 252, Figs. 15, ISBN 0-571-11541-1. Price: R10,40

Although the author specifically states that this book is intended for racing pigeon fanciers and not for scientists, it can be a valuable asset on the veterinarian's bookshelf. As the title indicates, the main object of the book is to keep the pigeons flying by proper health care, disease prevention and optimal nutrition.

A wide variety of viral, bacterial, fungal and parasitic diseases and ailments of pigeons are dealt with. Other chapters include elementary physiology, nutrition, pigeon surgery, drugs and their uses and public health hazards caused by pigeons.

The book was first published in 1961. The second edition appeared in 1968. The copy received for reviewing is the 1983 paperback format of the second edition. During the 15 years new drugs and especially more effective anthelmintics and insecticides have become available. In this respect the book is outdated.

J.F.W. Grosskopf

THE EFFICACY OF IVERMECTIN* AGAINST HELMINTH AND ARTHROPOD PARASITES OF IMPALA

I.G. HORAK**, J. BOOMKER***, SHIRLEY A. KINGSLEY*** and V. DE VOS****

ABSTRACT: Horak I.G.; Boomker J.; Kingsley Shirley A.; De Vos V. **The efficacy of ivermectin against helminth and arthropod parasites of impala.** *Journal of the South African Veterinary Association* (1983) **54** No. 4, 251-253 (En). Tick Research Unit, Rhodes University, 6140 Grahamstown, Republic of South Africa.

The efficacy of ivermectin, injected subcutaneously at a dosage rate of 200 mcg/kg live mass, was determined against nematodes, ixodid ticks and lice infestations acquired by free-living impala, *Aepyceros melampus*, in the Kruger National Park. Although the parasite burdens of the untreated control animals varied considerably, ivermectin appeared to be highly effective against 7 nematode species and effective against 3 others. Of the 4 tick species recovered, only *Boophilus decoloratus* appeared to have been affected. In the case of the lice infestations, ivermectin was highly effective against 3 species of *Linognathus*, but ineffective against the 2 *Damalinia* species present.

Key words: Ivermectin, lice, tick, nematode infestation, impala.

INTRODUCTION

The efficacy of ivermectin against parasitic nematodes in cattle and sheep has previously been demonstrated[1,2,10]. It is also effective against single-host ticks[5] and some multi-host ticks[6] on cattle and against sucking lice[5,8]. The present paper records its efficacy against helminth and arthropod parasites of impala, *Aepyceros melampus*, a wild ruminant species.

MATERIALS AND METHODS

Fourteen free-living impala, of various ages and both sexes, were caught without the use of chemicals during May 1982 in the vicinity of Skukuza, in the south of the Kruger National Park. These animals were confined at Skukuza in an enclosure (70 x 30 m), in which they had free access to water and were fed dry hay cut from the veld. There was also a little grass on which they could nibble, in the enclosure.

Six animals were treated with ivermectin administered subcutaneously at a dosage rate of 200 mcg/kg live mass on the day after their capture and 2 untreated animals were slaughtered at the same time. The treated animals were marked with paint on the rump and ran with the untreated controls until they had all been slaughtered.

Six to eight days after treatment, 3 treated impala and 3 untreated controls were slaughtered. Fourteen and 15 days after treatment the remaining two treated impala and two controls were slaughtered. One of the treated animals and one of the controls, destined to be slaughtered at the latter occasion, had been caught by a leopard during the intervening period.

Helminths and arthropods were recovered from these animals as previously described[4]. The lungs were, however, not processed for worm recovery.

Table 1: THE ANTHELMINTIC EFFICACY OF IVERMECTIN IN IMPALA

Impala No.	Age	Sex	Treatment	Day slaughtered	Numbers of nematodes recovered														
					Longistrongylus sabie		*Haemonchus krugeri*		*Trichostrongylus*		*Cooperia hungi*		*Cooperioides hamiltoni*	*Impalaia tuberculata*		*Strongyloides* sp.	*Gaigeria pachyscelis*	*Oesophagostomum columbianum*	
									thomasi	*colubriformis*									
					4th	Adult	4th	Adult	Adult	Adult	4th	Adult	Adult	4th	Adult	Adult	Adult	4th	Adult
1	29 months	M	Control	0	0	27	0	6	0	0	0	32	27	0	0	0	26	0	0
2	17 months	M	Control	0	0	96	0	53	179	1457	0	205	531	0	252	400	25	25	0
3*	Adult	F	Control	6	620	106	90	0	353	0	0	1680	453	0	101	0	0	0	3
4*°	29 months	F	Control	6	0	3	0	0	111	681	0	278	278	0	26	515	1	0	1
5*	Adult	F	Control	7	0	1	0	25	84	53	25	229	157	25	0	625	0	50	0
6	29 months	F	Ivermectin	8	1	0	0	0	0	0	0	1	51	0	0	73	0	0	0
7	17 months	F	Ivermectin	8	0	0	0	0	0	0	0	50	105	0	0	1	0	0	0
8*	17 months	M	Ivermectin	8	0	1	0	1	0	0	0	128	100	0	0	57	0	0	0
9°°	17 months	M	Control	14	1	9	0	25	463	2329	1	553	431	0	79	156	4	0	25
10°°°	Adult	M	Control	15	100	552	25	25	1029	5147	228	734	1329	575	462	200	1	0	75
11	17 months	M	Ivermectin	15	0	0	0	0	0	75	0	125	1	0	0	50	0	0	0
12	17 months	F	Ivermectin	15	0	0	0	0	0	25	0	0	52	0	0	25	0	0	0

*Infested with adult paramphistomes 4th = Fourth stage larvae
°*Trichostrongylus falculatus* 47 Adults
°°*Trichostrongylus falculatus* 474 Adults, *Cooperia neitzi* 145 Adults
°°°*Cooperioides hepaticae* 269 Adults

*Ivomec: MSD (Pty) Ltd.
**Tick Research Unit, Rhodes University, 6140 Grahamstown.
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****National Parks Board, Skukuza.

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RESULTS

The nematode burdens of the impala are summarized in Table 1.

With the exception of *Cooperia hungi*, *Cooperoides hamiltoni* and *Strongyloides* sp., against which efficacy seemed variable, ivermectin was highly effective against the various nematode species present in the impala.

The ixodid tick burdens of the impala are summarized in Table 2.

The tick burdens of the control and treated animals generally decreased the longer the animals were kept in the enclosure prior to slaughter. With the exception of the treated animals slaughtered 15 days after treatment, on which there may have been some activity against adult *Boophilus decoloratus*, ivermectin appeared to have had no effect on the adult or immature stages of the other ticks.

The lice burdens of the impala are summarized in Table 3.

Ivermectin was highly effective against the sucking lice *Linognathus aepycerus*, *Linognathus nevilli* and *Linognathus* sp., but had no apparent effect on the biting lice *Damalinia aepycerus* and *Damalinia elongata*.

DISCUSSION

Thirteen species of nematodes were recovered from the impala and, as can be expected with naturally acquired infestations, the worm burdens varied considerably. Although ivermectin was generally highly effective against most nematodes present its efficacy against *Cooperia hungi* and the related *Cooperioides hamiltoni* was variable. A similar phenomenon has also been noted with *Cooperia curticei* in sheep[10]. The efficacy against *Strongyloides* sp. also appeared variable. The effect of ivermectin against this genus has apparently not previously been determined in ruminants.

Table 2: THE ACARICIDAL EFFICACY OF IVERMECTIN ON IMPALA

Impala No.	Treatment	Day slaughtered	Numbers of ticks recovered														
			Amblyomma hebraeum			*Boophilus decoloratus*				*Rhipicephalus appendiculatus/ zambeziensis*				*Rhipicephalus evertsi evertsi*			
			Larvae	Nymphae	♂	Larvae	Nymphae	♂	♀	Larvae	Nymphae	♂	♀	Larvae	Nymphae	♂	♀
1	Control	0	464	192	0	1776	1568	256	250	448	0	36	12	240	0	0	2
2	Control	0	1216	386	0	3744	1904	528	146	432	0	14	2	304	96	2	0
3	Control	6	58	18	0	320	678	288	124	8	0	24	8	12	20	0	0
4	Control	6	50	46	2	360	798	244	130	0	0	10	10	120	8	0	0
5	Control	7	32	34	0	396	1026	266	132	12	0	16	6	56	106	0	0
6	Ivermectin	8	94	54	0	464	864	234	248	14	0	6	8	24	8	0	0
7	Ivermectin	8	546	54	0	376	384	130	60	10	0	0	2	56	0	0	0
8	Ivermectin	8	144	0	0	624	1280	216	80	8	0	2	2	64	96	0	0
9	Control	14	26	16	0	0	354	116	98	16	0	4	0	2	8	0	0
10	Control	15	10	16	0	30	600	298	128	12	6	2	0	0	6	18	0
11	Ivermectin	15	12	2	0	36	196	6	12	4	6	4	2	4	0	0	0
12	Ivermectin	15	58	4	0	214	210	42	28	0	0	2	2	0	4	0	0

Table 3: THE INSECTICIDAL EFFICACY OF IVERMECTIN ON IMPALA

Impala No.	Treatment	Day slaughtered	Numbers of lice recovered									
			Damalinia aepycerus		*Damalinia elongata*		*Linognathus aepycerus*		*Linognathus nevilli*		*Linognathus* sp.	
			Nymphae	Adults	Nymphae	Adults	Nymphae	Adults	Nymphae	Adults	Nymphae	Adults
1	Control	0	0	0	0	16	0	0	0	0	0	0
2	Control	0	880	512	160	96	64	96	0	0	64	96
3	Control	6	18	4	4	6	14	20	36	12	0	4
4	Control	6	4	4	16	12	32	36	0	4	0	12
5	Control	7	0	6	4	20	0	2	2	2	0	0
6	Ivermectin	8	6	2	4	4	0	0	0	0	0	0
7	Ivermectin	8	8	32	1160	650	0	0	0	0	0	0
8	Ivermectin	8	32	0	464	184	0	0	0	0	0	0
9	Control	14	10	2	0	2	60	62	0	0	0	2
10	Control	15	96	0	16	12	140	198	54	46	52	216
11	Ivermectin	15	12	0	6	2	0	0	0	0	0	0
12	Ivermectin	15	76	38	28	26	0	0	0	0	0	2

The impala harboured 4 species of ixodid ticks. As they were probably not exposed to further infestation in the pen, their tick burdens decreased fairly rapidly as the ticks engorged and dropped off. This makes interpretation of the results difficult, but with the possible exception of *B. decoloratus*, ivermectin was not effective against the tick species present. As *B. decoloratus* is a one-host tick, the apparent efficacy against the adult ticks may partially have been due to an effect against the immature stages resulting in delayed maturation. Improved control of *B. decoloratus* and possibly the other ticks, too, can probably be obtained by regular short interval treatment of animals[6,7].

The efficacy of ivermectin against *Linognathus* spp. and its inefficacy against *Damalinia* spp. on cattle has previously been reported[8]. The present experiment shows a similar pattern for these lice genera on impala.

Although free-living wild ruminants are seldom treated for parasites such treatment is advisable upon capture and translocation[3]. Ivermectin with its low toxicity[9] and high efficacy against most parasitic nematodes, sucking lice and the parasitic larvae of several fly species[5] would appear to be the drug of choice on such occasions.

ACKNOWLEDGEMENTS

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BOOK REVIEW — BOEKRESENSIE

HANDBOOK OF VETERINARY NEUROLOGIC DIAGNOSIS

JOHN E. OLIVER & MICHAEL D. LORENZ

W.B. Saunders Company, Philadelphia. 1983 pp IX and 371, Figs. 179, Tables 109, Price R47,50.
(ISBN 0-7216-6967-0)

Professors Oliver and Lorenz are to be congratulated on a well-written text providing a logical approach to the solving of neurological disorders in animals. The dog has been used as the model in their book with brief reference to appropriate species differences.

The text is divided into two major sections. The first dwells on the essential fundamentals of neurologic diagnosis such as the history, physical examination of the nervous system, localization of lesions in the nervous system and ancillary diagnostic aids. A chapter dealing briefly with the principles of medical treatment has also been included.

The second part of the text presents the most common clinical neurological problems presented to the clinician such as seizures, blindness, coma and paresis of a limb. The anatomic diagnosis is firstly reviewed, followed by a diagnostic and treatment plan. A brief discussion on the major differential diagnostic conditions is also presented.

A very useful and stimulating self-assessment section concludes every chapter throughout the book.

The text is amply illustrated with numerous photographs, sketches, diagrams and tables.

This book is a must for veterinary clinicians and students with an interest in veterinary neorology. I have no hesitation in strongly recommending it as a valuable aid in the diagnosis of neurological disorders.

J. van Heerden

BOVINE PARAFILARIASIS AT THE CATO RIDGE ABATTOIR: SEX PREVALENCE AND DISTRICTS OF ORIGIN

D.B. WEAVER*, H.G. WALLACE* and P.M. KRETZMANN*

ABSTRACT: Weaver D.B.; Wallace H.G.; Kretzmann P.M. **Bovine parafilariasis at the Cato Ridge abattoir: Sex prevalence and districts of origin.** *Journal of the South African Veterinary Association* (1983) **54** No. 4, 254 (En). Cato Ridge Abattoir, P.O. Box 206, 3680 Cato Ridge, Natal, Republic of South Africa..

Records indicate that *Parafilaria bovicola* -infested cattle slaughtered at the Cato Ridge abattoir originate from certain areas not previously recorded. Futhermore, moderate to severe infestations are diagnosed significantly more frequently in the entire male bovine than in female animals or oxen (castrated males). The data under review cover the period March 1981-August 1982, a period of 18 months.

Keywords: *Parafilaria bovicola,* sex prevalence, cattle helminths.

INTRODUCTION

Carmichael & Koster[1] reported that the distribution of parafilariasis in South Africa is fairly widespread, involving the Bushveld, the Lowveld, the Northern and South Western aspects of the Transvaal, the Eastern Orange Free State, Northern Natal and Zululand.

DISTRIBUTION

Our records indicate that cattle slaughtered at the Cato Ridge Abattoir are derived from Natal, Kwa Zulu, East Griqualand, the Eastern Cape and the Eastern aspects of the Orange Free State.

During the 18 months under review, the origin of carcases found to be infested with *Parafilaria bovicola* was recorded at the Cato Ridge Abattoir. The disease was identified in cattle derived from the following districts: Ubombo, Hlabisa, Ngotshe, Lower Umfolozi, Eshowe, Lower Tugela, Umzinto, Umzimkulu, Underberg, Ixopo, Impendhle, Richmond, Pietermaritzburg, Camperdown, New Hanover, Umvoti, Lions River, Mooi River, Estcourt, Weenen, Kliprivier, Dundee, Vryheid, Babanango, Kokstad, Swartberg, Cedarville, Mount Currie and Kwa Zulu, Harrismith and Senekal in the Orange Free State and Elliot and Queenstown in the Eastern Province.

Feedlots are the centralized depots for livestock and approximately 60 % of the cattle slaughtered at Cato Ridge Abattoir originate from them. With the increasing movement of cattle between farms and to feedlots it is believed that parafilariasis may be becoming far more widespread and possibly even enzootic in much of Natal and other areas from which stock is drawn. It is at present impossible in many cases to determine whether the disease is acquired or contracted in each of the districts described or whether the condition has been imported.

SEX PREVALENCE

Wallace et al.[2] describes in detail the method of inspection and handling of carcases at the Cato Ridge Abattoir. From the statistics kept of all the incidents of parafilariasis diagnosed during secondary inspection by the Veterinary and Control Meat Inspectors of the Division of Veterinary Services, the evidence collated indicates that in the moderate to severe cases of the disease, there is a 4,6 times as great a prevalence of parafilariasis in bulls than in cows or 2,6 times that found in oxen (Table 1).

Table 1: PREVALENCE OF PARAFILARIASIS IN BULLS, COWS AND OXEN

	Kill		Carcases with parafilariasis	
	Total	Percentage	Number	Percentage
Bulls	15 300	5,2	332	2,17
Cows	102 567	34,7	484	0,47
(Heifers) Oxen	177 733	60,1	1 453	0,82
Total	295 600	100,0	2 269	0,77

Statistical analysis using a X^2 test of significance showed that the difference in prevalance between bulls, females and oxen was highly significant ($X^2_{(2\ DF)} = 519$, $P < ,001$). Both the comparisons between bulls and the remainder as well as the comparison of oxen versus females were statistically significant as well ($X^2 = 417$ and 113 respectively, $P < ,001$).

CONCLUSION

The recorded places of origin of cattle found to have parafilariasis on slaughter at the Cato Ridge Abattoir indicates that further studies into the distribution of the insect vectors should be considered. Lastly, the greater prevalence of parafilariasis in bulls leaves a further avenue open for research.

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ATTEMPTED PREVENTION AND TREATMENT OF *GEIGERIA FILIFOLIA* MATTF. POISONING (VERMEERSIEKTE) IN SHEEP

J.P.J. JOUBERT*

ABSTRACT: Joubert J.P.J. **Attempted prevention and treatment of *Geigeria filifolia* Mattf. poisoning (vermeersiekte) in sheep.** *Journal of the South African Veterinary Association* (1983) **54** No. 4, 255-258 (En). Section of Toxicology, Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa.

A pilot trial with 2 sheep per group demonstrated that no protection is provided by cysteine, sulphur or sodium thiosulphate, when administered simultaneously with daily doses of *Geigeria filifolia* plant material. Treatment with piracetam** of 4 sheep showing symptoms of vermeersiekte did not improve their recovery rate. Serum enzyme activity tests for creatine phosphokinase, glutamic oxaloacetic transaminase and gamma glutamyl transpeptidase were of no diagnostic use.

Key words: Prevention, treatment, *Geigeria filifolia*, Vermeersiekte, plant poisoning, sheep.

INTRODUCTION

Poisoning following the ingestion of *Geigeria* spp. plants (vermeerbos) gives rise to vermeersiekte, which, literally translated, means vomiting disease. This poisoning syndrome largely effects sheep and to a lesser extent goats and cattle. It occurs in the North Western Cape and on the highveld of the Orange Free State and Transvaal. The area with the highest incidence is the Ghaap plateau northwest of Kimberley, where as many as 50 000 sheep died during 1954[1]. Another large outbreak occurred during 1981 (R.W. Muir 1981, State Veterinarian, Kimberley, personal communication).

The clinical signs of vermeersiekte may be one or a combination of some of the following: Vomition of ruminal contents which stains the nostrils and lips a greenish tinge, stiffness or painful muscles resulting in trembling of skeletal muscles and frequent recumbency, paralysis which may be an advanced form of stiffness, and bloat[1,7,8].

Sheep may graze vermeerbos for about 3 weeks before showing clinical signs of vermeersiekte. If the sick animals are removed from the vermeerbos grazing, they usually recover, unless regurgitated ingesta causes foreign body pneumonia[1,7].

At autopsy, signs of secondary pneumonia can often be seen. Histopathological examination has revealed the presence of vacuolation and hyalinization of oesophageal and skeletal muscle fibres[4]. Smit, according to Grosskopf[1], demonstrated degeneration, perivascular oedema and focal areas of necrobiosis in thalami of 13 vermeersiekte sheep.

The poisonous substances in these plants are sesquiterpene lactones and many have been isolated[1,2,6,9,10]. Chemists have proposed that they bind covalently by means of an alkylation reaction with their exocyclic methylene groups to sulfhidryl groups of cysteine residues of enzymes and other proteins[2]. This mechanism of action prompted the experimental use of cysteine to treat sesquiterpene lactone poisoning in sheep. An LD_{92} of hymenoxon (a sesquiterpene lactone) was injected intraperitoneally into sheep. This was followed 10 minutes later by the intravenous (i.v.) administration of cysteine. Sheep which received 69-138 mg/kg body mass of cysteine showed a significant increase in survival rate, compared with those which had received lower dosages and the untreated control animals[6].

Meanwhile, farmers and veterinarians have reported that piracetam (Nootropil, UCB Pharmaceuticals) injected i.v. into sheep suffering from vermeersiekte enhanced their recovery rate. Piracetam is 2 oxo pyrrolidine acetamide, which can be regarded as a cyclic derivative of gamma amino butyric acid (GABA), an inhibitory impulse transmitter substance in the brain. It improves the glucose turnover and increases the synthesis of adenosine triphosphate (ATP) in neurones, resulting in an increase in available energy for neurones[5].

Vermeersiekte is most difficult to control by the application of judicious grazing management on the Ghaap plateau with its limestone soil, as the substratum consists of a hard calcrete formation with very little top soil. The perennial grasses and shrubs are limited to a few areas with deep top soil, while pioneer plants, such as *Geigeria filifolia* Mattf., inhabit the remainder of the plateau. It must therefore be accepted that vermeerbos will always be a potential danger in this area[1].

MATERIALS AND METHODS

Experiment 1

Ten Merino wethers of about 1 year of age and having body masses varying between 31 and 42 kg were used.

G. filifolia plants in the flowering stage were collected at Koopmansfontein on the Ghaap plateau. These samples were identified at the Botanical Research Institute in Pretoria, dried, milled and stored at room temperature.

Biochemical grade L-cysteine from Merck Chemicals, commercial sodium thiosulphate sulphur and piracetam were dosed simultaneously with the plant material, while piracetam was administered only after signs of vermeersiekte had commenced. The dosing regimen is set out in Table 1. Sheep which died, were autopsied.

Experiment 2

The sheep employed in this experiment were 5 Merino ewes and one wether, all of them 2-toothed. Their body masses varied between 37 and 50,5 kg.

G. filifolia material prepared for the first experiment was made use of. All the sheep were dosed per stomach tube with 5 g/kg *G. filifolia* once a day, excepting over weekends. Blood sampling and mass measurements were repeated once a week, until signs of vomition or

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**Nootropil, UCB Pharmaceuticals.

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stiffness were evident. From that stage, blood samples were taken every second day. These specimens were analysed to determine levels of creatine phosphokinase (CK), glutamic oxalo acetic transaminase (GOT) and gamma glutamyl transpeptidase[3] (8-GT) in the serum.

The sheep were randomly divided into 2 groups, so that there were 2 in the control group and 4 in the treatment group. The latter group was treated with piracetam at 3 g per sheep i.v. from the 3rd day on which they showed clinical signs of vermeersiekte. Greenish stained mouth and nostrils and trembling of skeletal muscles were taken as the first signs. The first treatment coincided with the last dose of *G. filifolia* material. Sheep which died were autopsied.

RESULTS

Experiment 1

All the sheep developed vermeersiekte after about 15 doses of *G. filifolia*. One of the piracetam sheep developed secondary pneumonia and it, as well as 2 other sheep, was euthanased. Vermeersiekte was diagnosed in all 3 cases at post mortem examination. The results of Experiment 1 are presented in Table 2.

Table 1: DOSING REGIMEN OF SHEEP WITH: *G. FILIFOLIA*, CYSTEINE, SULPHUR, SODIUM THIOSULPHATE AND PIRACETAM

Sheep group: No: / Treatment	Control 2	Cysteine 2	Sodium thio-sulphate 2	Sulphur 2	Pira-cetam 2
Geigeria material daily 5 g/kg per os	X	X	X	X	X
Cysteine 100 mg/kg i.v. once daily		X			
Sodium thiosulphate daily 10-20 g per os per sheep			X		
Sulphur daily 5-6 g per os per sheep				X	
Piracetam 4 g per sheep i.v. per day for 2-3 days*					X

*Treatment commenced after symptoms of vermeersiekte were evident

Table 2: RESULTS OF TREATMENT OF VERMEERSIEKTE-SHEEP, WITH: CYSTEINE, SULPHUR, SODIUM THIOSULPHATE AND PIRACETAM

Sheep No treatment	Time elapsed until vermeer-siekte occurred (days)	*G. filifolia* No. of doses	Clinical sings	Fate: Recovery (days) after with-drawal of *G. filifolia*
1 Control	21	15	Stiffness and tremors of muscles	Recovered 4
2 Control	21	15	Nostrils stained green and tremors of skeletal muscles	Recovered 3
3 Sulphur	18	15	Stiffness and tremors of skeletal muscles	Condition deteriorated: Euthanased on Day 21
4 Sulphur	18	15	Stiffness and tremors of skeletal muscles	Condition deteriorated: Euthanased on Day 21
5 Sodium thiosulphate	10	7	Nostrils stained green, anorexia	Recovered 4
6 Sodium thiosulphate	22	16	Nostrils and lips stained green, vomited	Recovered 2
7 Cysteine	22	15	Nostrils stained green	Recovered 2
8 Cysteine	21	15	Stiffness and tremors of skeletal muscles	Recovered 3
9 Piracetam	18	15	Nostrils stained green, secondary pneumonia, treated Days 18-20	Condition deteriorated: Euthanased on Day 21
10 Piracetam	21	15	Nostrils stained green, tremors of skeletal muscles, treated Days 22 and 23	Recovered 2

Table 3: THE TREATMENT OF *G. FILIFOLIUS* POISONING (VERMEERSIEKTE) IN SHEEP WITH PIRACETAM

Sheep				*G. fillifolia* 5 g/kg		Clinical appearance		Piracetam twice a day for 2 days at 75 mg/kg i.v.: effect
Mass (kg)		No.	Group	Period dosed (days)	Doses No.	Days sick (No.)	Symptoms	
Initial	Terminal							
50,5	46	1	Control	39	31	9	Nostrils stained green, vomited, unease, trembling of muscles, frequently recumbent Recovery phase: Unable to swallow chewed food	—
41	32	2	Control	19	15	16	Nostrils stained green, vomited, gnashed its teeth, groaned with expiration, coughed, anorexia, trembling of muscles, Recovered slowly	—
45,5	39	3	Treated	24	18	11	Vomited, gnashed its teeth, muscles trembled, frequently recumbent, anorexia. Recovery phase: Unable to swallow chewed food	Habitus improved, vomition decreased for a few hours after treatment. Recovered completely 6 days after treatment
37	34	4	Treated	29	21	5	Vomited profusely, gnashed its teeth, coughed, groaned with expiration. Died after 5 days	Received no treatment before death
42	40	5	Treated	26	20	9	Skeletal muscles trembled severely when forced to stand, mostly recumbent, anorexia	No perceptible improvement after treatment. Recovered completely 4 days after treatment
42	37	6	Treated	30	22	7	Vomited, gnashed its teeth, anorexia, muscles trembled, back arched	Improved visibly for a few hours after each treatment. Recovered completely 2 days after treatment

Table 4: SHEEP SERUM VALUES OF CPK, ϑ-GT AND GOT IN VERMEERSIEKTE

	CPK Values IU/ℓ						ϑ-GT Values IU/ℓ						GOT Values IU/ℓ						Remarks
Sheep (No.)	1	2	3	4	5	6	1	2	3	4	5	6	1	2	3	4	5	6	
Day 0	68	58	74	74	83	55	18	14	19	13	22	22	43	38	53	39	45	49	Normal values
8	110*	79	77	96	181*	85	17	14	19	14	22	21	44	40	48	40	44	45	
14	71	96	87	114*	89	97	18	13	19	13	19	17	47	49	49	50	40	44	
21	67	114*	67	140*	81	83	15	12	19	16	19	19	50	47	61	59	49	51	
23		166*	148*	76	71	78		13	17	12	18	23		39	59	54	52	61	
25	72	95	81	82	117*	67	15	9	16	17	13	21	56	35	70	88	57	46	
28	81	81	126*		93	67	14	8	16		15	12	50	27	67		64	43	
30	93	91	90		78	92	16	10	14		16	17	61	27	55		62	43	
32	85	70	78				20	17	17				66	32	49				
35	107*						14						62						
37	66						14						73						
39	123*						11						69						
42	58						10						73						
44	57						11						86						

*CPK values indicating slight bruises due to handling

Experiment 2

All the sheep developed vermeersiekte. One sheep in the treated group died before piracetam could be administered. Results of the treatment with piracetam are summarized in Table 3. The treatment transiently improved the condition of vermeersiekte, but vomition recurred after a few hours. Furthermore, treated sheep did not recover remarkably sooner than the untreated controls.

One of the controls required as many as 28 doses of *G. filifolia* before clinical signs of vermeersiekte appeared. Serum levels of CPK, GOT and δ-GT did not increase in correlation with the development of these clinical signs. Serum enzyme test results are shown in Table 4.

DISCUSSION

None of the sheep treated with cysteine, sulphur or sodium thiosulphate showed increased resistance to the effects of *G. filifolia*. Although only 2 sheep were used per group, the results strongly indicate that these substances have no preventative or therapeutic value for vermeersiekte. In addition, it is important to note that sulphur and sodium thiosulphate were used at almost their maximum safe levels[7].

Treatment with piracetam resulted in an improvement of short duration in affected sheep, but did not reduce the recovery period sufficiently to warrant its use. Untreated sheep can recover equally quickly after withdrawal of *G. filifolia* material. Furthermore, the increased levels of serum CPK indicate only mild bruising of muscles owing to normal handling; the concentration would have to have been increased ten-fold before muscular damage could have been diagnosed (F. Reyers 1982, Faculty of Veterinary Science, University of Pretoria, personal communication).

Although these experiments failed to produce a practical method for the treatment of vermeersiekte, this should not discourage further research in this field, as vermeersiekte remains one of the most important plant poisoning syndromes in the Republic of South Africa.

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REVIEW OORSIG

CANINE PARVOVIRUS IMMUNOPROPHYLAXIS: A REVIEW

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The presently available data on canine parvovirus is reviewed with particular emphasis on the degree of immunity provided by the various types of vaccines commercially available, the interference of active immunisation by maternally-derived antibody, and the recommendations as to age of vaccination.

Key words: Canine parvovirus, immunisation, antibody, immunity.

INTRODUCTION

Canine parvovirus (CPV) infection emerged as a potentially fatal, highly contagious viral disease of dogs in the United States of America (USA) during 1978[1-4 6 11 15 18 19 23 24 29 32 35]. Within a short period of time the infection had assumed world-wide epidemic proportions[1-4 6 10 14 16 23-27 29 32 33 37 38]. Serologic surveys conducted in the USA indicate an absence of CPV antibodies in dog sera prior to May 1978[15]. Serologic surveys in Belgium indicate that CPV antibodies were present in that country prior to 1978 and the serodynamics in Belgian dogs were as follows: 1976-0 %; 1977-20 %; 1978-36 %; 1979-60 %; since 1979 the percentage of CPV-antibody positive dogs has remained constant at approximately 60 % of the general canine population[15]. Further serologic surveys have demonstrated that between 20 % and 92 % of dogs in various populations have recovered from clinical or subclinical CPV infection[15 24 31].

Canine parvovirus is a small (20nm) DNA virus of the family Parvoviridae[1 21 24 29]. Parvoviruses are the smallest of the true viruses and those affecting vertebrates are subdivided into 2 groups[2 24]:

1. Adeno-associated parvoviruses (recombinant viruses) which require simultaneous infection by an adenovirus for their replication.
2. Autonomous (nondefective) parvoviruses which do not require the assistance of an adenovirus but do depend on host cell DNA for replication. Replication occurs in the nucleus of actively dividing cells and may produce intranuclear inclusion bodies. The autonomous parvoviruses include canine parvovirus (CPV), feline panleukopaenia virus (FPV), mink enteritis virus (MEV), Aleutian mink virus (AMV), minute virus of canines (MVC), porcine parvovirus and bovine parvovirus[2 24].

The origin of CPV remains speculative and may never be accurately determined[2 3 15 21 24 25]. Canine parvovirus, FPV and MEV are closely related antigenically[3 11 15 24 29 32]. It has been theorised that FPV mutated to render it pathogenic for mink and that a further mutation has now rendered the virus pathogenic for dogs[2 3 15 24]. The mutations may have taken place in either the cat or the dog or in members of the family Mustidae (mink, raccoon) which are susceptible to both cat and dog viruses[2 3]. A further hypothesis is that the mutation may have occurred during laboratory passage of either attenuated FPV or with a FPV or MEV strain[2 3 24]. Furthermore, it has been argued that CPV may be a mutant of the MVC (although they differ antigenically) or possibly a de novo virus[15].

Canine parvovirus can be identified by tissue culture, fluorescent antibody detection, electron microscopy, haemagglutination (HA), haemagglutination-inhibition (HI) and serum-neutralisation (SN) tests[2 3 4 11 15 24 29]. The HA phenomena differentiates CPV from FPV, MEV and MVC[3 11]. Serologic comparisons of the CPV, FPV and MEV by HA-HI and SN tests indicate that CPV, FPV and MEV are antigenically similar but are different from MVC[11]. Electron microscope examination of infected faeces can identify parvovirus viral particles but immunoelectron microscopy is required to differentiate CPV from MVC[11 24].

Laboratory diagnosis of active CPV infection requires evidence of active CPV shed in excretions or evidence that the serum antibody titre is of recent origin because prior vaccination or previous CPV infection could result in high antibody titres which may persist for many months[24 29-31]. The HI test is the most commonly used laboratory procedure to determine CPV serum antibody titres because it is rapid, sensitive and relatively inexpensive[3 11 29 30]. However, the HI test alone fails to differentiate between antibody titres of recent infection and titres of longstanding[24 31]. Recent CPV infection (less than 3 weeks previously) can be confirmed serologically by demonstrating that the CPV antibody is of the Immunoglobulin M (IgM) class due to the primary immunologic response[24 29]. The HI titre of serum from recently infected dogs is markedly reduced by the treatment of the serum with 2-mercapto-ethanol (2-M—E), a compound that dissociates IgM[24 29]. The CPV antibody titre from dogs infected more than three weeks previously is unaffected by treatment with 2-M—E as the antibody is primarily of the IgG class at this time[24 29].

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Parvoviruses are noted for their resistance to inactivation by extremes of temperature and various disinfectants which include numerous alcohols, iodides, phenolics, quaternary ammonium compounds, coal and wood tar derivatives and soaps[15,24,29,34]. A temperature of 80°C (176°F) for 15 minutes is necessary to completely inactivate CPV[32]. A 4 % formaldehyde solution or a one in 32 part dilution of 5,6 % sodium hypochlorite (household bleach) solution is able to inactivate CPV[15,24,29,32,34]. A more thorough means of sterilization is the use of formaldehyde gas fumigation[7,13,28]. Formaldehyde gas is released by the addition of potassium permangenate to a 40 % formaldehyde solution[13,28]. For routine fumigation, 40 g of potassium permangenate are added to 60 ml of 40 % formaldehyde solution for every 2,83 m^3 (100 cubic feet) to be sterilized[13,28]. However, successful sterilization can be accomplished by using 40 g of potassium permangenate to 120 ml of 40 % formaldehyde solution for every 2,8 m^3 to be sterilized[7]. The formaldehyde gas should be allowed to act for at least 10-12 hours[12,25]. As the chemical reaction causes considerable bubbling, and evolution of heat, a heat and splash proof container should be used[13,28]. Potassium permangenate stain can be removed with hydrogen peroxide or a dilute acid[13]. In the environment CPV may persist indefinitely[24,29].

Because CPV is extremely resistant to inactivation it is easily transmitted directly by contact with infected dogs[1,2,14,21-23,26,29]. Faeces from dogs with CPV infection serves as the primary source of virus spread[1,2,15,21,24-26,29,30,32]. Dogs with CPV infection may excrete as much as 10^9 median tissue culture infectious doses ($TCID_{50}$) per gram of stool for 5-8 d after the onset of clinical signs[29]. It has been established that the minimum infectious dose for dogs is approximately one tissue culture infectious dose, thus the potential for spread of infection by the faecal-oral route is enormous[29]. CPV may also be present in the vomitus, saliva and urine of infected dogs as a viraemia is a feature of the infection[15,24,29]. Virus cannot be isolated in tissue culture from faeces from infected dogs for more than 2 weeks after oral exposure (5-8 d after the onset of clinical signs) although susceptible in contact dogs may become infected for about 3 weeks[3,11,15,21,24,29,30]. Chronic shedding of CPV does not occur following clinical recovery as it appears to occur with FPV infection in cats[15,24,29]. Canine parvovirus infection can be transmitted via fomites such as contaminated food, feeding utensils, bedding, cages and clothing[15,24]. Humans in close contact with CPV infected dogs have not developed antibodies to CPV and the host range appears to be restricted to members of the family Canidae[15,29].

Although the morbidity rate in some dog populations during 1978 and 1979 was 100%, the mortality rate in affected dogs of all ages was probably between 8 % and 15 % while the mortality rate for the canine population as a whole was less than 1 %[15,21,24-26,29,32,33].

IMMUNISATION

When the devastating effects and extreme resistance of CPV were recognised, it was concluded that an effective immunisation programme would be the ultimate form of control[4,6,15,24,29,30]. Because of the fortuitous antigenic similarities between FPV and CPV, it was determined that FPV would immunize dogs against CPV[3,4,6,9,11,12,15,19,20,24-27,29-33,35,38]. Thus, inactivated and modified live FPV vaccines were adopted for use in the dog[3,4,6,9,11,12,15,19,20,24,27,29-33]. These feline origin vaccines were used, not as ideal immunising agents, but as expedients to protect a largely susceptible canine population[6].

The ideal vaccine should be safe, efficacious, easily administered and provide long-term protection[20,30,35]. It must protect not only the individual immunised dog, but also the canine population at large by preventing CPV shedding from a vaccinated but only partially protected individual, when exposed to oral CPV challenge[6,29,30].

There is a direct correlation between CPV antibody titre and the ability to resist oral CPV challenge[3,10,24,29,30]. In most dogs an HI titre $\geq$ 1:80 resists viral shed after oral challenge[24,29,30]. Hence, circulating CPV antibody must be present at high levels (HI titre $\geq$ 1:80) at the time of challenge to prevent initial viral replication, systemic spread of the infection and faecal shedding[27,30]. Lower levels of CPV antibody (HI titres < 1:40) appear to prevent viraemia and systemic distribution of CPV, although localised infection of intestinal lymphoid tissue occurs which results in infection of the overlying intestinal epithelium and virus shedding in the faeces in the absence of overt clinical signs (sub-clinical infection)[24,29,30]. Thus although dogs with a low CPV antibody titre may not exhibit overt clinical signs of CPV disease when challenged orally with virulent CPV, they are not in fact immune to CPV, in the strict sense[24].

Dogs that have recovered from CPV infection usually have high HI titres $\geq$ 1:640 and are immune to reinfection for at least 20 months[6,30]. It is probable that recovered dogs are immune to reinfection for life[29,30].

Inactivated Vaccines

Both inactivated (killed) FPV and inactivated CPV vaccines provide protection against CPV infection for a finite period after immunisation[6,24,29]. However, HI antibody titres usually decline rapidly after immunisation with inactivated vaccine, becoming undetectable in most dogs by 12 weeks after the second inoculation[5,24,29,30]. The rapid decline in antibody titre is well recognised with inactivated vaccines, because the antigens, consisting mainly of protein, are steadily degraded by normal catabolic processes[35].

The antibody response to inactivated CPV is superior to that of inactivated FPV[5,6,24,29,30,35]. Inactivated CPV and inactivated FPV vaccines usually result in a lower antibody titre than do modified live virus (MLV) FPV vaccines[6,24,29,30,35]. The MLV FPV vaccines usually produce a lower antibody titre than do the MLV CPV vaccines[6,24,29,30,35]. The duration of immunity derived from inactivated vaccine is influenced by the antigenicity of the vaccine virus, the method of virus inactivation and the adjuvant used[5,29,30,35]. Some inactivated CPV vaccines may result in prolonged protective antibody response[5].

Increasing the amount of viral antigen in the inactivated CPV and FPV vaccine increases the initial antibody response but does not alter the rate of antibody decline[29,30]. Inactivated CPV and FPV vaccines may be used in pregnant animals to establish a protective passive immunity in the young[18,21].

Serum antibody titres > 1:40 do not persist in most dogs for more than 12 weeks after immunisation with inactivated CPV or inactivated FPV[24,20,30]. Thus, many of these dogs, exposed to virulent CPV challenge after

12 weeks will shed faecal virus and become a source of infection for other dogs[20,30]. This state of "partial protection" persists for at least 5 months after immunisation with inactivated CPV and FPV and probably accounts for the apparent effectiveness of these vaccines in the field[5,12,21,24,29,30,33]. The probability is great that under field conditions the "partially protected" dogs will be exposed to CPV challenge and develop subclinical infections which will serve to boost the CPV immunity of the exposed dog and confer prolonged immunity[29,30]. Such subclinically infected dogs may shed virulent CPV in their faeces and thus serve to perpetuate CPV in the population[29,30].

Inactivated CPV and FPV vaccines provide immunity to CPV, but it is usually short lived[15,24,29,30,35]. Thus, it would appear that to ensure optimal protection to CPV, immunisation would have to be repeated several times annually[20,30,35]. The expense and inconvenience that this would impose limits the practical usefulness of inactivated vaccines[29,30].

Live Vaccines

The response of dogs to immunisation with MLV FPV is divided into 2 categories[30]:

1. Those that develop relatively low antibody titres which decline rapidly and thus resemble dogs inoculated with inactivated virus vaccine[30].
2. Those that develop much greater initial antibody titres which persists and thus resemble dogs that have recovered from CPV infection[30].

The proportion of dogs that develop prolonged protective antibody titres in response to MLV FPV is directly proportional to the amount of living virus in the vaccine dose[21,29,30]. A vaccine dose with $10^{7,5}$ $TCID_{50}$ of MLV FPV (approximately 100 times more live virus than in most licenced vaccines for use in cats) was successful in protecting 100 % of the vaccinated dogs[29,30]. If the vaccine dose contained $10^{5,5}$ $TCID_{50}$ of MLV FPV only about 62 % of vaccinated dogs developed protective CPV antibody titres[20,30]. If the vaccine dose contained $10^{3,5}$ $TCID_{50}$ of MLV FPV only about 38 % of vaccinated dogs developed protective antibody titres[29,30].

High antibody titres develop in dogs in which the MLV FPV is able to replicate and thus establish a productive infection which continues to stimulate the immune system[30,35]. The antigen mass of an inactivated vaccine is much greater than that of a MLV vaccine[35].

Following a single inoculation with MLV FPV vaccine 92 % of the dogs demonstrated seroconversion with HI antibody titres $\geq$ 1:10 although only 58 % developed HI titres $\geq$ 1:80 (the minimum titre necessary to prevent faecal viral shedding after oral challenge)[29]. If 2 inoculations were given 3 weeks apart some of the dogs with low initial titres developed protective antibody titres resulting in 78 % of dogs so inoculated having HI titres $\geq$ 1:80[29]. In a further survey it was determined that approximately one third of dogs inoculated twice with MLV FPV did not develop protective CPV antibody titres[20]. The dogs which had the highest antibody titres were those with a history of probable exposure to CPV infected dogs[20]. It was also determined that only 11 % of all dogs had a marked increase in serum CPV antibody titre after booster vaccination with MLV FPV, indicating that the serologic response to booster vaccination with MLV FPV vaccine was poor[20].

Modified live FPV vaccine appears to be safe for use in the dog and no spread of vaccine virus has been detected nor has there been any report of illness or adverse reactions in vaccinated dogs[12,20,21,27,29,32]. However, their use in dogs has been of recent duration and deleterious effects may yet be reported[29]. The use of MLV FPV vaccine is not recommended in pregnant bitches[21,35].

The route of vaccine administration may influence the antibody response by allowing the antigen rapid access to the reticulo-endothelial system[21]. A MLV FPV vaccine has been shown to provide a higher CPV antibody titre via the intravenous route than by either the subcutaneous or intramuscular route[21].

The ultimate protection of dogs against CPV infection has been the development of a MLV canine origin CPV vaccine[6,8,19,29,30]. The MLV CPV vaccine has been shown to provide a more uniform antigenic response utilising a smaller immunising dose[29,30]. The CPV antibody titre engendered by the MLV CPV is superior to that produced by inactivated CPV, inactivated FPV and MLV FPV[6,19,29,30,35]. Immunisation with MLV CPV consistently results in CPV antibody titres $\geq$ 1:320 which persists for at least 20 months[6,30]. Thus, prolonged immunity, including protection from sub-clinical infection can be induced following the administration of a single dose of MLV CPV vaccine[6,30]. The MLV CPV has the ability to overcome low levels of maternal antibodies, sufficient to block the immune response to inactivated CPV, inactivated FPV and MLV FPV, thus ensuring earlier successful immunisation of puppies with maternal CPV antibodies[6,29,30]. The MLV CPV appears to be innocuous to both pregnant bitches and newborn puppies[29].

An immunologic interference test has demonstrated that dogs developed protective antibody titres to all specific antigens in a combined MLV canine distemper virus – MLV adenovirus 2 – MLV parainfluenza virus – MLV CPV vaccine and a *Leptospira interrogans* serovars *canicola* and *icterohaemorrhagiae* bacterin[6,8]. In the reversion to virulence tests the MLV CPV strains remained non-pathogenic during 6 passages in seronegative test dogs[6,8]. Although certain vaccine strains of MLV CPV are shed in the faeces of vaccinated dogs, the reversion to virulence tests demonstrated that reversion of the vaccine strain to a pathogenic state is unlikely[6]. Some MLV CPV vaccine strains are not shed in the faeces of vaccinated dogs[8]. In the case of CPV, faecal shedding diminishes with the degree of attenuation of the infective virus strain[6]. Non-attenuated CPV strains are readily isolated from faecal content between 3 and 9 days after oral exposure of susceptible dogs[3,6,24,29,30]. The amount of virus excreted in the faeces declines as the number of passages in cell culture increases for a given CPV strain[6]. Thus, the MLV CPV vaccines are advocated as the immunising agents of choice in protecting dogs against CPV infection[18].

MATERNAL ANTIBODY

Canine parvovirus antibody is transferred from a recovered or successfully vaccinated bitch to her pups via the placenta and colostrum[15,19,24,29–32,35,36]. The amount of passively acquired maternal antibody is directly proportional to the CPV antibody titre of the bitch and is equivalent to 50 % of the dams' serum antibody titre[15,24,29–31]. This level of colostral-derived immunity relative to that of the dam is lower than that

reported for distemper (77 %) and infectious canine hepatitis (99 %)[31]. The half-life of maternally-derived CPV antibody is 9,7 d which is slightly longer than that reported for distemper (8,4 d) and infectious canine hepatitis (8,6 d)[30,31]. The highest level of maternally-derived CPV antibody is found in small litters of puppies as 90 % of the immunity is derived from the colostrum[20-31]. Studies with infectious canine hepatitis reveal that the absorption of colostral antibodies is virtually completed by 72 hours after birth[31]. Transplacental transfer of maternal CPV antibody accounts for about 10 % of the passive CPV immunity[29-31]. Nevertheless, the amount of antibody transferred to pups *in utero* from a successfully vaccinated or CPV recovered bitch is sufficient to render colostrum-deprived pups refractory to immunisation or infection for several weeks[29-31].

Vaccines that induce greater and more sustained humoral immune responses in breeding bitches would provide longer protection for pups through increased maternal antibody transfer[6,29-31]. Maternally-derived CPV antibody titres in pups decline regularly and exponentially with time[6,29-31]. The maternal CPV antibody in pups can be estimated if the titre of the dam and the age of the pups is known[6,29-31]. It is thus possible to determine the minimal CPV antibody titre that interferes with vaccination and to predict the earliest age at which pups would be expected to become susceptible to CPV infection or amenable to immunisation[6,29-31]. It has been suggested that the CPV immunisation programme for puppies should be instituted when the pups' CPV HI antibody titre is predicted to be below 1:5[29].

If pups are inoculated while sufficient maternal antibody persists ($\geq$ 1:10), there is no active response to vaccination and these pups may succumb to infection if exposed to CPV when the protective levels of maternal antibody are no longer present[6,29-31]. The age at which pups can be succesfully vaccinated depends on the CPV antibody titre of the dam[6,29-31]. Pups born to dams which have recovered from CPV infection receive enough maternal antibody to block active immunisation for up to 16 weeks of age[6,29-31]. However, it has been determined that there is a period of susceptibility during which maternal antibody levels in the pups block active immunisation yet are no longer protective against virulent CPV challenge[29]. Thus, apparent "vaccine failures" will continue to occur in some pups regardless of which type of vaccine is employed[29-31]. For this reason it is especially important that older, in-contact dogs are immunised with vaccines that prevent shedding of virulent CPV after challenge ("vaccine partial protection")[29-31]. This would minimise the possibility of exposure of pups to virulent CPV during the critical period of waning maternal immunity[29-31].

The MLV CPV is able to immunise pups that have sufficient maternal CPV antibody to block the response to MLV FPV as well as inactivated CPV and inactivated FPV[6,29-31]. This advantage reflects the inherent immunogenicity in dogs of the MLV canine origin CPV[6]. Thus, the MLV CPV would limit the spread of CPV in a susceptible of "partially protected" host population and control the disease in an epidemiologic sense[6].

IMMUNISATION GUIDELINES

The present recommendation for CPV immunisation by the American Veterinary Medical Association is that the vaccine be administered either subcutaneously or intramuscularly, that the first vaccination be performed at 6-8 weeks of age, and that the vaccination is repeated at 10-12 weeks of age and again at 14-16 weeks of age[9]. Dogs older than 16 weeks of age should receive 2 vaccinations administered 3-4 weeks apart[18]. Ideally, bitches should be revaccinated prior to breeding[18]. However, if a pregnant bitch's immune status is unknown or if a nonprotective CPV antibody titre is present, vaccination with 2 doses of killed CPV vaccine 3-4 weeks apart in the last trimester of pregnancy is recommended[18]. Annual revaccination is recommended[9,18,29-31]. Utilising this vaccination programme we can improve canine health examination which should be an important feature of a sound animal health programme[35].

This vaccination schedule may require future modification in order to protect all breeds of dogs[36]. Certain individuals of such breeds as the Rottweiler appear to remain refractory to successful immunisation despite the use of the recommended immunisation regime[36].

Canine parvovirus may exert an immunosuppressive effect on the canine immune response to MLV canine distemper virus (CDV) immunisation[17,23]. A MLV CDV vaccine may become pathogenic in a CPV infected dog and result in CDV encephalomyelitis[17,23]. Thus, the use of a MLV polyvalent CDV vaccine in a dog exhibiting clinical signs of CPV disease may be contraindicated[23].

IMMUNISATION FAILURES

Several viruses are presently recognised as a cause of canine enteric disease and these include: canine parvovirus, canine coronavirus, canine reovirus, canine rotavirus, minute virus of canines, canine distemper virus, canine adenovirus type I and canine herpesvirus[1,4,14,15,24,29]. Immunisation with MLV CPV, MLV FPV, inactivated CPV and inactivated FPV can only protect dogs against CPV infection[15,24,29]. Thus, in dogs vaccinated against CPV disease and exhibiting clinical signs of gastrointestinal disease the differential diagnoses may include not only the viral causes of gastrointestinal disease or vaccine failure, but must also consider haemorrhagic gastroenteritis; endotoxic shock; intestinal verminosis; intestinal protozoal, bacterial and fungal infections; toxin ingestion; foreign body obstruction and intussusception; pancreatitis; adrenocortical insufficiency and metabolic diseases[15,24,29,38].

The causes of a poor response to immunisation are numerous and may include: concurrent exposure to CPV or incubation of CPV infection at the time of vaccination; adrenocorticosteroid therapy resulting in suppression of the immunologic response; debility; intercurrent disease; parasitism; hypoproteinaemia; poor nutrition; immunodeficiency diseases; incorrect vaccination procedure and individual breed susceptibility[15,24,29,36].

DISCUSSION

In a retrospective survey of CPV diseases it has been shown that the immunisation of dogs with inactivated FPV, inactivated CPV and MLV FPV achieved commendable results in an epidemic situation[33]. It was estimated that CPV disease occurred in only 0,68 % of vaccinated dogs[33]. Experimental and clinical evidence

has demonstrated the beneficial effects and superior antigenicity provided by the canine origin MLV CPV vaccines[6 8 18 19 29 30 31]. The MLV CPV vaccines stimulate CPV antibody titres $\geq$ 1:320 and thus prevent viral shedding when exposed to virulent CPV challenge[30]. Antibody titres of this magnitude confer immunity for longer than one year[30]. In contrast, antibody titres of this magnitude and immunity lasting as long as a year develop only sporadically, if at all, when other types of immunising agents for CPV are used[30]. The MLV CPV vaccine is able to immunise pups that have sufficient maternal antibody to block an immunologic response to MLV FPV as well as inactivated FPV and CPV vaccines[6 29-31]. Thus, MLV CPV vaccines are the immunising agents of choice for CPV disease although vaccination failures have been observed[6 8 18 19 24 29-31 36]. There is a possibility that a combined MLV CDV CPV vaccine does not protect as successfully against CPV as a vaccine containing MLV CPV alone[18].

Further research into CPV disease will continue to improve the immunising agents presently available particularly once the importance of cell-mediated immune responses (CMIR) of CPV are known. To date only antibody-mediated immune response (AMIR) has been reported[3 6 8 11 12 18-21 24 29-33 36]. To evaluate the immune response stimulated by a vaccine the antibody titre (AMIR) and CMIR must both be evaluated[16]. It has been shown that the protection afforded to dogs against distemper by the measles virus vaccine is mediated via the CMIR[16]. The heterotypic immunity (protection against one disease by an antigenically-related virus causing another disease) conferred by the measles virus has a beneficial effect in dogs younger than twelve weeks of age[10 16]. The combined canine distemper-measles vaccine has definite advantages by protecting a greater percentage against canine distemper than either vaccine alone[10]. Whether a similar combined vaccine containing CPV and either of the antigenically related FPV or MEV would have a similar effect remains to be determined.

As the number of CPV immune dogs in the general population increases, either by vaccination or by natural exposure, CPV will be limited to a small number of dogs[18 35]. The most susceptible dogs will be those that do not have passive colostral immunity, those in which colostral immunity interfered with vaccination, those that were not vaccinated, immunosuppressed dogs ot those dogs who do not respond to the vaccine antigen[35]. Total eradication of CPV is unlikely and thus immunisation against CPV must be included in the routine vaccination program of dogs[18 29 35]. It is essential, however to fully forewarn dog owners as to the shortcomings of the present vaccines and the veterinary profession's incomplete knowledge, at the present time, of this new canine disease.

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BOOK REVIEW BOEKRESENSIE

HANDBOOK OF SMALL ANIMAL ORTHOPEDICS AND FRACTURE TREATMENT

W.O. BRINKER, D.L. PIERMATTEI and GRETCHEN L. FLO

W.B. Saunders Company, Philadelphia 1983 pp vi + 435 Figs 350 ISBN 0-7216-1991-6 Price R65,80

The authors state that this handbook was formulated to serve the practitioner and student as a readily available source of information covering the diagnosis, aetiology and treatment of those conditions affecting bones and joints and resulting in impairment of function.

The first part of the book (210 pages) deals with the classification, diagnosis and treatment of fractures. The basic principles involved in fracture repair, including the use of external and internal fixation, are outlined. The advantages and disadvantages of the various methods of internal fixation are discussed. This is followed by chapters on bone infection, transplantation of bone and complications of fracture repair. The major part of this section is devoted to a discussion of specific fractures. Various methods of fixation including their indications and complications are presented in a clear and understandable manner.

The second part of the book (192 pages) deals with lameness and joint surgery. A chapter is included on the complete physical examination of the orthopaedic patient, an essential and useful part of the diagnosis of lameness and deranged function. The structure and function of joints, the abnormalities of the internal structures and conditions generally affecting joints are described, followed by the diagnosis and treatment of specific orthopaedic conditions of the hind- and forelimb.

The third and last part of the book (20 pages) deals with miscellaneous diseases of the musculoskeletal system. Eosinophilic panosteitis, nutritional disorders, retained cartilage cores, injury to muscles, tendons and ligaments and other conditions are included.

This is a good book. It is well illustrated and the line drawings of which there are over a thousand are clear and make it easy to follow the surgical procedures described in the text. The authors are well known and experienced small animal orthopaedic surgeons and they have made full use of their wide experience to compile a book of a very high standard. The surgical procedures and techniques described are those that they found to be most successful in treating the conditions for which they were designed. It can serve as reference book for practitioners and students. Post graduate students interested in orthopaedic surgery will also find it a valuable source of information. It is a pity that such a good book was not published with a hard cover. It is, however a book that can be strongly recommended to anyone involved in or interested in small animal orthopaedic surgery.

D.G. Steyn

SHORT COMMUNICATION **KORT MEDEDELING**

ORAL ANTACID TREATMENT IN CLINICAL RUMEN ACIDOSIS

S.R. VAN AMSTEL*

ABSTRACT: Van Amstel S.R. **Oral antacid treatment in clinical rumen acidosis.** *Journal of the South African Veterinary Association* (1983) 54 No. 4, 265-266 (En). Department of Medicine, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

pH changes were measured in vitro in rumenfluid obtained from a natural case of rumen acidosis after the addition of 8 different antacids. Results showed magnesium oxide to be very potent with the potential danger of causing severe rumen alkalosis. Calcium hydroxide and magnesium carbonate gave satisfactory results while the alkalinising ability of magnesium hydroxide, magnesium trisilicate, calcium carbonate, aluminium hydroxide and sodium bicarbonate seemed both slow and ineffective.

Key words: Oral antacids, rumen acidosis.

INTRODUCTION

The use of oral antacids in cases of rumen acidosis can be of benefit. This would depend on the stage of the condition, the type of antacid and the dosage used. The advantage of using oral antacids in rumen lactic acidosis is the resultant rise in pH which will increase the concentration of undissociated lactic acid thus retarding its absorbtion. This in turn will limit the intensity of the metabolic acidosis resulting from lactic acid absorbtion. The disadvantage of using oral antacids is the conversion of neutralised lactic acid to bicarbonate in the liver from which a significant metabolic alkalosis could result. It therefore seems that oral antacids should be used at such a rate as to limit lactic acid absorbtion without causing a severe metabolic alkalosis. An experiment was carried out to test the efficacy of various commonly available antacids at various dose rates using rumen fluid from a natural case of rumen acidosis.

MATERIALS AND METHODS

Rumen fluid was obtained from a bovine which had overeaten on bread 12 hours previously. It had a pH of 4,1. Various antacids as shown in Table 1 were mixed with the rumen fluid at a rate of 1 and 2 g to 300 ml of the fluid. Accepting a maximum rumen capacity of 150 l this would correspond to total dosages of 500 and 1 000 g for an adult bovine. The pH of the rumen fluid was recorded immediately and then at 1, 2 and 2½ hours after mixing with the antacids. The results of the pH changes are shown in Table 1. The readings were done by using a radiometer pH meter PHM 27 with a glass (G202C) and a calomel (K401) electrode.

Table 1: pH RESPONSE OF RUMEN FLUID TO THE ADDITION OF ANTACIDS

Antacids used	Antacid to rumenfluid ratio. gms:ml 1:300				Antacid to rumenfluid ratio. gms:ml 2:300			
	Time after addition of antacids				Time after addition of antacids			
	Immediate	1 hour	2 hour	2½ hours	Immediate	1 hour	2 hours	2½ hours
	pH readings				pH readings			
Magnesium oxide	5,4	7,5	8,2	8,4	5,7	9,0	9,4	9,5
Magnesium carbonate	5,3	5,5	5,6	5,6	5,6	5,8	5,9	6,0
Magnesium hydroxide	4,4	4,9	5,0	5,2	4,5	5,3	5,6	5,9
Magnesium trisilicate	4,3	4,8	4,9	4,8	4,4	5,1	5,4	5,6
Calcium hydroxide	5,5	5,9	6,0	6,0	5,8	5,9	6,1	6,3
Calcium carbonate	4,6	4,8	4,9	5,0	4,7	4,9	5,0	5,1
Aluminium hydroxide	4,2	4,2	4,3	4,3	4,2	4,2	4,2	4,3
Sodium bicarbonate	4,8	5,0	5,0		4,9	5,4	5,4	

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0038-2809/83/04/0265-0266

DISCUSSION

Magnesium compounds are the most frequently recommended for cases of rumen acidosis[1,2,3]. General dosage rates recommended for these compounds correspond to the ratio of 1:300 as used in this experiment[1,3]. From the results shown in Table 1 it can be seen that magnesium oxide caused a rapid and dramatic increase in pH. pH response to magnesium hydroxide was poor at the general recommended ratio of 1:300. It seems therefore that it should be used at higher dosage rates. Magnesium carbonate had a more rapid response than magnesium hydroxide and the pH after 2½ hours was also higher. As with all the other antacids except magnesium oxide there was very little difference in the pH readings after 1 hour as compared with the 2½ hour readings. It therefore seems feasible that these antacids could be repeated 1 hour after dosing but possibly at a lower dosage ratio than 1:300. A disadvantage in the use of magnesium carbonate is its potential for gas formation which could lead to bloat in clinical cases. Magnesium trisilicate seems to be ineffective at a ratio of 1:300. Even at double that dosage the response is slow and repeat treatment may be necessary.

On the calcium compounds, calcium carbonate produced a slow and poor response at both dosage rates. Calcium hydroxide at a ratio of 1:300, however, generated a rapid response and brought the pH up to 6,0. Repeat treatment may thus not be necessary. Aluminium hydroxide at both dosage rates had virtually no effect on the pH. The response to sodium bicarbonate was also very poor. A distinct disadvantage is its gas formation potential which can be dangerous especially at ratios of more than 1:300.

CONCLUSIONS

The results of this in vitro investigation confirm the potent antacid properties of magnesium oxide. If used at the recommended dosage rates, magnesium oxide could cause a severe rumen and metabolic alkalosis. Magnesium carbonate produced a more rapid response and was more effective than magnesium hydroxide. When used at a ratio of 1:300 repeat dosage after 1 hour should be carried out at lower levels.

Calcium hydroxide generated a rapid response bringing the pH up to 6 at a ratio of 1:300. Repeat dosage may therefore not be necessary.

The remainder of the antacids tested showed a slow and poor response and is not recommended for use in clinical cases.

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BOOK REVIEW — BOEKRESENSIE

FORMULATION OF VETERINARY DOSAGE FORMS

Edited by JACK BLODINGER

Vol. 17 in the Series "*Drugs and the Pharmaceutical Sciences*", Marcel Dekker Inc., New York. 1983. pp. VIII and 332, numerous tables and figures, Price US $48.50 (ISBN 0-8247-1730-9)

This new textbook comprizes 6 chapters, each by different authors. The first chapter discusses the basis for selection of dosage forms. The concepts of drug absorption, distribution and elimination are introduced with the emphasis on species differences. The choice of different dosage forms (tablets, capsules, solution, suspensions, etc.) and different routes of administration are discussed and a basic discussion of bioavailability and pharmacokinetics is presented.

The second chapter details drug delivery devices (syringes, dosing guns, darts, etc.) most of which would be familiar to the veterinary practitioner. Formulation of drug dosage forms for animals is discussed in the third chapter which also contains information on antioxidants, preservatives and thickening agents.

The fourth chapter discusses the formulation of drugs for administration via feed or drinking water, a subject of great importance in intensive poultry, pig or feedlot situations. Such aspects as chemical stability and the compatibility of drugs in premixes and feeds are covered.

The fifth chapter deals with stability studies of veterinary formulations. The relevant parameters, e.g. odour, colour, clarity, pH, viscosity, etc. are listed and some analytical techniques are mentioned.

The final chapter concerns general principles for the regulatory control of veterinary drugs along with some specific requirements for Australia, Brazil, the European Economic Community, Japan and the United States.

This text has attempted to condense a tremendous mass of information. It should prove interesting to veterinarians and other involved in academic pharmacology, drug regulation, and the pharmaceutical industry but it should not be seen as an in-depth reference work.

C. Button

CASE REPORT GEVALVERSLAG

ADENOVIRUS PNEUMONIA IN A PUPPY

I.B.J. VAN RENSBURG* and M. GREENBERG**

ABSTRACT: Van Rensburg I.B.J.; Greenberg M. **Adenovirus pneumonia in a puppy.** *Journal of the South African Veterinary Association* (1983) **54** No. 4, 267-269 (En). Department of Pathology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

A case of adenovirus pneumonia in a puppy is described. The emphasis is on histopathology where an alveolitis with typical Cowdry type A intranuclear inclusions in macrophages, pneumocytes and bronchiolar epithelial cells were the most significant findings. The literature on the subject is briefly reviewed.

Key words: Pneumonia, adenovirus, intranuclear inclusions, canine.

INTRODUCTION

Adenoviruses are frequently associated with respiratory disease in man[5 8] and animals[1 2 4 15]. In the dog, 2 types of adenovirus are known to cause respiratory disease, namely canine adenovirus type 1 (CAV 1) which is the well known cause of infectious canine hepatitis (ICH)[9 10] and canine adenovirus type 2 (CAV 2) or Toronto A26/61 virus[3]. Both these viruses have been isolated from dogs suffering from respiratory distress[1 2 11 12 13 14], while pneumonia has been induced experimentally in puppies by the administration of CAV 1 without the production of ICH[13 14]. Few reports exist on naturally occurring adenovirus pneumonia in dogs and, in most of these (75 %), it was in association with canine distemper[4 6 7 11 12]. However, in a study of 42 pneumonic canine lungs in Belgium, Ducatelle et al.[4] found typical Cowdry type A intranuclear inclusions in 14 of them. Despite this relatively high incidence in Belgium, no cases of adenovirus pneumonia have previously been reported in the Republic of South Africa, although it has been diagnosed on several occasions.

CASE HISTORY, CLINICAL SIGNS AND MACROSCOPICAL PATHOLOGY

Eight days after having been inoculated against distemper, ICH and canine parvovirus with a vaccine containing modified live strains of CAV2, distemper virus, parainfluenza virus and parvovirus (DA2P + CPV vaccine, Norden), a male German Shepherd puppy was presented having shown respiratory signs for the previous 3 days. On examination it manifested a non-productive laryngo-tracheal cough, bilateral conjunctival congestion and a rectal temperature of 39,2 °C. Symptomatic treatment consisting of an antimicrobial (purbac, Lennon) and an antitussive (Benylin expectorant, Parke Davis and codein) was prescribed. On Day 9 post-inoculation (p.i.) clinical signs had progressed to a mucopurulent rhinitis, severe conjunctivitis and epiphora. A tentative diagnosis of canine distemper was made. Treatment remained the same. Haematology done on Days 11 and 12 p.i. revealed a leukopaenia (B-WBC 6,5000 and 5,500 × $10^9/\ell$ respectively) and marginal neutrophilia (88 %) and lymphopaenia (8 %) on Day 11 p.i. There was also a moderate thrombocytopaenia and abnormal platelet morphology present. At this stage antibiotic treatment was changed to tylosin (Tylan 200, Elanco). Despite treatment, pulmonary rales and pyrrhexia persisted, although clinically the puppy was happy and alert. By Day 16 p.i. the body temperature was normal, but purulent rhinitis was still present and a tracheal cough could easily be elicited by palpation. On Day 18 p.i. the pup's condition suddenly deteriorated; it showed anorexia, anterior abdominal pain and a decline in habitus. It died on the 19th day after vaccination.

On necropsy a generalised congestion, cyanosis and moderate dehydration was present. Plum-red areas of consolidation occurred in all lobes of the lungs. Specimens of lung, liver, kidney and myocardium were fixed in 10 % formalin for histopathological examination.

Microscopical findings

Examination of haematoxylin and eosin stained lung sections revealed an exudative pneumonia which varied in intensity in different localities. In areas the alveolitis was very marked with large numbers of monocytic cells and neutrophils filling the alveoli. Very little fibrin was present and no hyalin membranes were noticed. A necrotising bronchiolites was present but focal areas of bronchiolar epithelial hyperplasia, as described by Wright[13], were not noticed. The most significant finding, from a diagnostic point of view, was the large number of Cowdry type A intranuclear inclusions observed most commonly in large macrophages in the alveoli, but which were also present in lesser numbers in pneumocytes and bronchiolar epithelial cells.

Sections of the liver and kidney, particularly the renal glomerular endothelial cells, were carefully scrutinised for the presence of intranuclear inclusion bodies but none were found. Typical distemper inclusion bodies were not noticed in either the pulmonary of renal pelvic epithelium.

No bacterial or viral isolation were done.

DISCUSSION

The presence of numerous intranuclear inclusions typical of those occurring in adenoviral infection made

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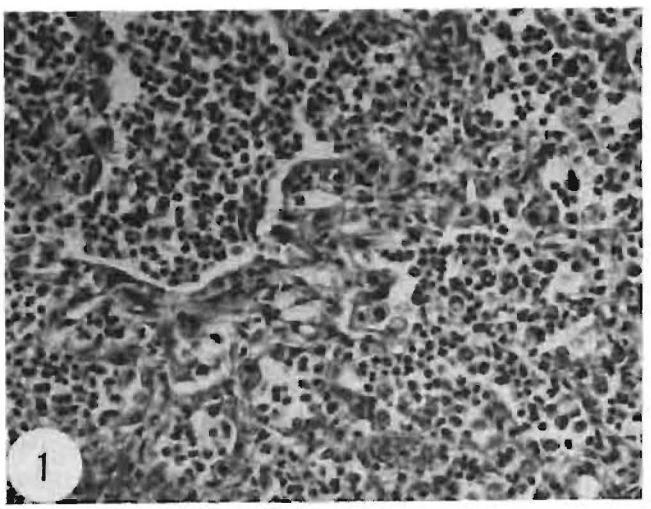


Fig. 1: Marked exudation of macrophages and neutrophils into alveoli.

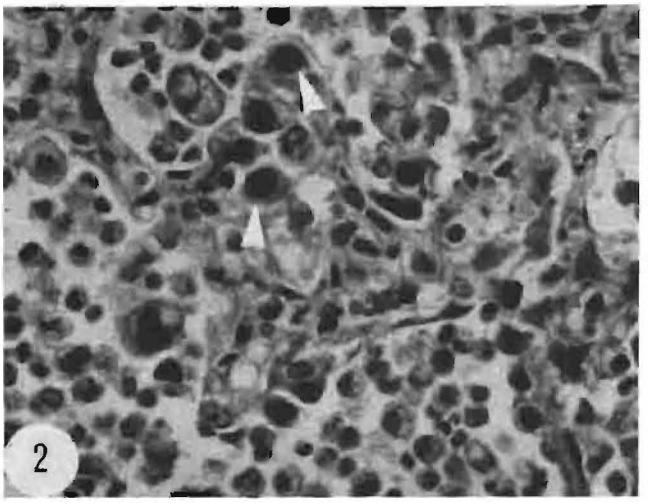
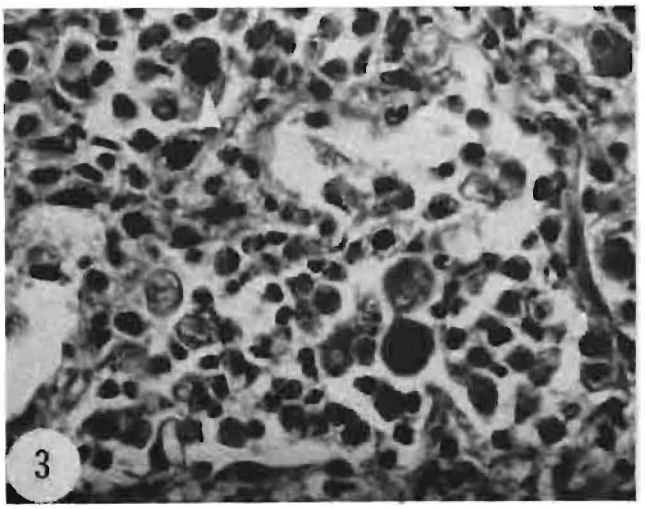


Fig. 2 & 3: Cowdry type A intranuclear inclusions (arrows) in large macrophages and pneumocytes.



the histopathological diagnosis a reliable and easy one. Most of these inclusions occurred in macrophages which were conspiciously enlarged while some were found in pneumocytes and bronchiolar epithelial cells. As in the hepatocytes bearing the inclusions of ICH, some inclusions in the case under review filled the nucleus, but most were surrounded by a halo with margination of the chromatin.

In the majority of the natural cases of adenovirus pneumonia reported[4,6,7,11,12] there was concurrent distemper virus infection evidenced either by typical inclusions in the respiratory epithelium or elsewhere in the body. In the case reported in this paper as well as in 7 of the 14 cases described by Ducatelle et al.[4], there was no evidence of distemper as manifested by the presence of typical inclusion bodies. In the opinion of these authors, the adenovirus seemed to have produced the most prominent pulmonary lesions despite the fact that evidence of distemper was found elsewhere in the body. There can be little doubt, therefore, that natural adenovirus infection is an important primary cause of pneumonia in young dogs, although it is not frequently diagnosed in the Department of Pathology, Faculty of Veterinary Science, University of Pretoria.

Of the 14 cases described by Fucatelle et al.[4] only one out of 10 showed Cowdry type A inclusions in the liver. In our case intranuclear inclusions were limited to the lungs. The upper respiratory tract was, however, not examined. *Bordetella bronchiseptica* was isolated from the lungs of 2 of the 14 dogs described by Ducatelle et al.[4] They thought that the low incidence of infection by this bacterium probably was due to intense antemortal antibiotic therapy of their patients. In the case described here bacterial isolation was not attempted, but in view of the finding of purulent foci of pneumonia in areas where inclusions were not prominent, it seems likely that the pup had suffered from secondary bacterial infection.

There can be no certainty whether natural infection with CAV1, CAV2, or, possibly, even the vaccine strain of CAV2 was responsible for the pneumonia in the puppy. The inoculation with a live vaccine containing CAV2 5 days prior to the onset of clinical signs compels one to speculate that this may have precipitated the syndrome. In 2 cases that have been described[12,13], adenovirus pneumonia developed 5 days and 2-3 weeks respectively after vaccination of puppies against ICH. However, in contradiction to this speculation is the experience of Wright et al.[13,14] that, when CAV1 is administered intravenously, it produces typical ICH and the only means to provoke pneumonia with it was by aerosol infection of puppies.

ACKNOWLEDGEMENTS

Professor R.C. Tustin is thanked for his constructive critisism in the preparation of the manuscript, Mrs V. Käber for the typing thereof and Mrs H. Smit for printing the microphotographs.

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BOOK REVIEW **BOEKRESENSIE**

THE VETERINARY ANNUAL

EDS. C.S.G. GRUNSELL & F.W.G. HILL

23rd Issue. Scientechnica, Bristol. Published by John Wright & Sons, 823-825 Bath Road, Bristol BS45NU, England. 1983 pp XII and 306, illustrations & figures 128, tables 28, Price not supplied (ISBN 0-85608-036-5).

As in previous issues this publication is in the form of a series of short review articles outlining recent developments in a wide range of Veterinary topics. Three special articles of general interest are included covering new findings in reproduction and infertility, animal husbandry and genetic engineering. There are 8 contributions on cattle including articles on respiratory diseases of calves, inherited resistance to mastitis and mycotic abortion. Two papers are concerned with staphylococcal dermatitis in sheep and sheep housing trends. The 4 articles covering pigs include one on biotin supplementation of sow diets and the 3 dealing with equines includes a contribution on limb fractures. There are 19 papers on small animal topics covering a diverse range of topics of interest to veterinarians including infectious diseases, opthalmology, surgery and pathology.

In short the Veterinary Annual contains diverse articles of a high standard which are of interest to both specialists and the general practitioner.

M.C. Williams

BOOK REVIEW **BOEKRESENSIE**

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

COMPILED BY: WILLIAM R. ATCHLEY, WILLIS W. WIRTH, CHARLES T. GASKINS and SANDRA L. STRAUSS

Available from: Office of Governmental and Public Affairs, U.S. Department of Agriculture, Washington, D.C. 20250, U.S.A. Bibliographics and Literature of Agriculture Number 13.

A bibliography is provided for the worldwide literature from 1758 to 1978 on the bloodsucking flies of the family Ceratopogonidae. The 3,527 references include both primary and secondary literature, and all foreign language titles are translated into English. In the keyword-in-context index, the references are sorted according to words or phrases appearing in the titles of the publications.

BOOK REVIEW **BOEKRESENSIE**

DISEASES OF CATTLE IN THE TROPICS

Editors: MIODRAG RISTIC & IAN McINTYRE

Vol. 6: Current Topics in Veterinary Medicine and Animal Science 1981 Martinus Nijhoff Publishers, P.O. Box 566, 2501 CN. The Hague, The Netherlands pp XII + 662 ISBN 90-247-2429-5.

The potential for cattle production in the tropics is considered greater than that realised at present. One of the major limiting factors is disease and it is the latter aspect that is pursued in this monograph.

The text is divided into 5 parts; each devoted to a particular aspect of the subject. The special topics in Part 1 include consideration being given to the ecologic (including zoonotic and epidemiologic) and economic implications of cattle production in the tropics. The major part of the work is encompassed in Part II which deals with the common and significant infectious diseases. Each disease is presented as an entity. Whilst recent advances in the epizootiologic, pathogenetic and prophylactic fields are emphasised, adequate recognition is given to the clinical and pathologic features of the disease. Part III consists of 2 chapters; the first, which considers calf rearing and problems related thereto cannot be praised highly enough as the thoughts offered reflect a welcome change of outlook and the second, which discusses infectious diseases of lesser frequency including actinobacillosis, actinomycosis, nocardiosis, eperythrozoonosis, haemobartonellosis and trichomoniasis. An appreciation of ticks and adverse climatic factors is provided in Part IV whilst the problems of disease control in the tropics is considered in Part V. An appendix in which the effects of toxic plants on livestock are grouped according to the clinical signs and systems affected is presented. The value of this appendix is difficult to appreciate. Besides the omissions (some serious) the mode of presentation precludes establishment of priorities and makes the entire exercise a little pointless.

Overall, the book offers a well balanced perspective on the problems related to the rearing of cattle in the tropics and, as such, may be recommended to veterinary student and bovine practitioner alike.

J.W. Nesbit

CASE REPORT GEVALVERSLAG

OSTEOSARCOMA IN A YOUNG GREAT DANE DOG

L.B. EVANS*

ABSTRACT: Evans L.B. **Osteosarcoma in a young Great Dane dog.** *Journal of the South African Veterinary Association* (1983) **54** No. 4, 271-273 (En). 8 Village Road, 3600 Kloof, Natal, Republic of South Africa.

A 10-month-old Great Dane dog was presented showing lameness in the left foreleg. Radiographic examination revealed a severe bony reaction of the left distal radius and ulna. The reaction together with associated soft tissue swelling increased dramatically over a 6 week period. A diagnosis of osteogenic sarcoma was confirmed histologically and the dog was euthanased. Metastases were found post mortem in the left prescapular lymph node and left lung.

Key words: osteosarcoma, young dog, Great Dane.

INTRODUCTION

Osteosarcoma is the most common bone tumour found in the dog. It is usually associated with middle-aged and older dogs, the highest incidence being reported in dogs more than 6 years old[1]. Large breeds such as Great Danes and St Bernards appear to be more commonly affected and there also appears to be a higher incidence in males than females.

These tumours are highly malignant and metastasize early to the lungs. Other possible sites of metastases include the liver, kidneys and the regional lymph nodes.

The most common sites of occurrence of this tumour are found in the proximal humerus, distal radius and ulna, proximal and distal ends of the femur and tibia, and the costo-chondral junctions[1,3].

Diagnosis of this tumour is generally dependent upon radiological findings, but it can be extremely difficult to differentiate this from other bone diseases such as osteomyelitis[3]. This is particularly so in the early stages when the only presenting signs may be mild localized pain, reluctance to excercise or slight lameness. Radiographs taken at this stage may show a small area of osteolysis or less frequently osteosclerosis. One or other of these processes may later predominate allowing the tumour then to be termed either an osteolytic or osteogenic sarcoma. The affected area of bone merges with the surrounding bone and is not clearly demarcated from it[3].

The case described here was thought worthy of recording because it was found in a relatively young dog and it also highlighted the difficulties first encountered in making a positive diagnosis of osteogenic sarcoma.

CASE REPORT

A 10-month-old Great Dane male dog was presented showing lameness in the left foreleg. The dog had recently been acquired from a welfare society and therefore the present owners had no knowledge of any detailed previous history. He had been fed a diet high in cereal and calcium content to which a commercial calcium supplement was added.

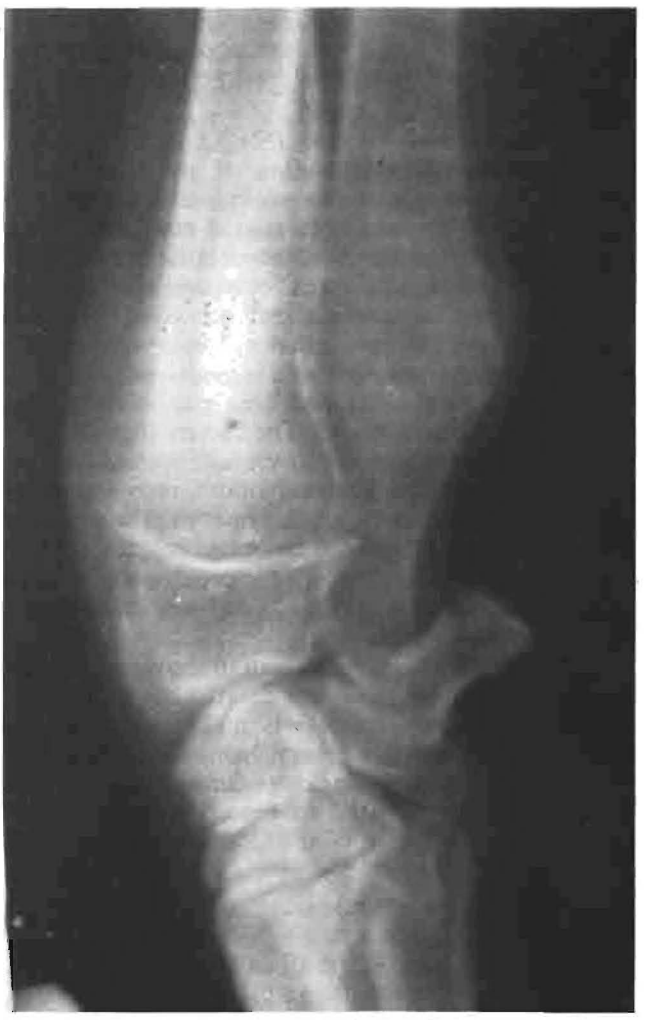

Fig. 1: Early stage of the osteosarcoma, seen as periosteal reaction on distal radius.

On examination the dog showed a limp on the left foreleg and slight pain evinced on palpation of the distal radius and ulna. No other signs were found.

Radiological examination of the leg revealed a severe bony reaction of the left distal radius and ulna (Fig. 1). A tentative diagnosis of panosteitis was made and treatment instituted with a preparation containing isopyrin, phebuzine and dexamethazone (Dexa tomanol,

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Hoechst). The dog returned a week later manifesting an even more severe lameness and a noticeable swelling which was present in the distal radius/ulna region. Differential diagnoses of an infective bone condition and hypertrophic osteodystrophy, or possibly even a bone tumour, were considered. The most likely diagnosis appeared to be an osteomyelitis and the treatment regimen was consequently altered to a combination of a preparation containing cortisone and an antibiotic (lincomycin). Over the course of the next week the dog's condition worsened. Blood samples were then taken for the determination of a white blood cell count and serum concentrations of alkaline phosphatase, and calcium and phosphorus. Further radiographs were taken of both forelegs, and a biopsy was taken from the medial surface of the left distal radius (the site of the greatest enlargement and which was most painful).

The following laboratory results were received:

WBC ($\times 10^9/\ell$)	8,5		
Polymorphs	0,71		
Lymphocytes	0,18		
Monocytes	0,09		
Alkaline phosphatase	310	(N = 250)	milliunits/ml
P – Calcium	2,86	(N = 2,25-2,75)	mmol/ℓ
P – Ionized calcium	1,46		mmol/ℓ
P – Phosphorous	1,93	(N = 0,8-1,6)	mmol/ℓ

The histological examination of the bone biopsy revealed the presence of dense collagenous fibrous connective tissue with associated related new bone formation and areas of pronounced osteoblastic proliferation. No evidence of neoplasia was found and a diagnosis of "periosteal new bone formation" was given. The comment was made, however, that a "rebiopsy should be considered if osseous neoplasia is suspected".

A swab from the affected area was submitted for bacteriological examination. The bacteriologist's report was "very scanty Gram negative bacilli present", but no growth was obtained. The antibiotic therapy was changed to chloramphenicol given daily per os. The dog's diet was also altered in order to reduce its calcium content.

Further radiographs revealed a progressive, marked periosteal bone reaction (Fig. 2) and surgical intervention was contemplated.

As the dog, at this point, began to show an improvement surgical intervention was delayed but the medication with chloramphenicol was maintained. The improvement in the dog's general habitus and in the affected leg, however, was of short duration and the condition suddenly appeared to become worse, i.e. the swelling of the distal radius and ulna increased drastically and appeared to progress proximally up the leg.

The dog was hospitalized and surgery was performed. During the operation the affected area was exposed, curetted and 3 drains inserted. In addition, swabs were taken for bacterial culture and sections of bone were taken for histopathological examination.

A light growth of *Staphylococcus aureus* was obtained from the swab, the organisms being sensitive to cotrimoxazole and certain other drugs. Treatment with cotrimoxazole was immediately instituted, but there was no obvious improvement. In fact, his condition rapidly deteriorated, the leg becoming enormously swollen.

The histopathological examination revealed the presence of an esteogenic sarcoma as well as large areas of necrosis and that the tumour had invaded the nedullary space of the bone.

After consultation with the owners, the dog was then immediately euthanased.

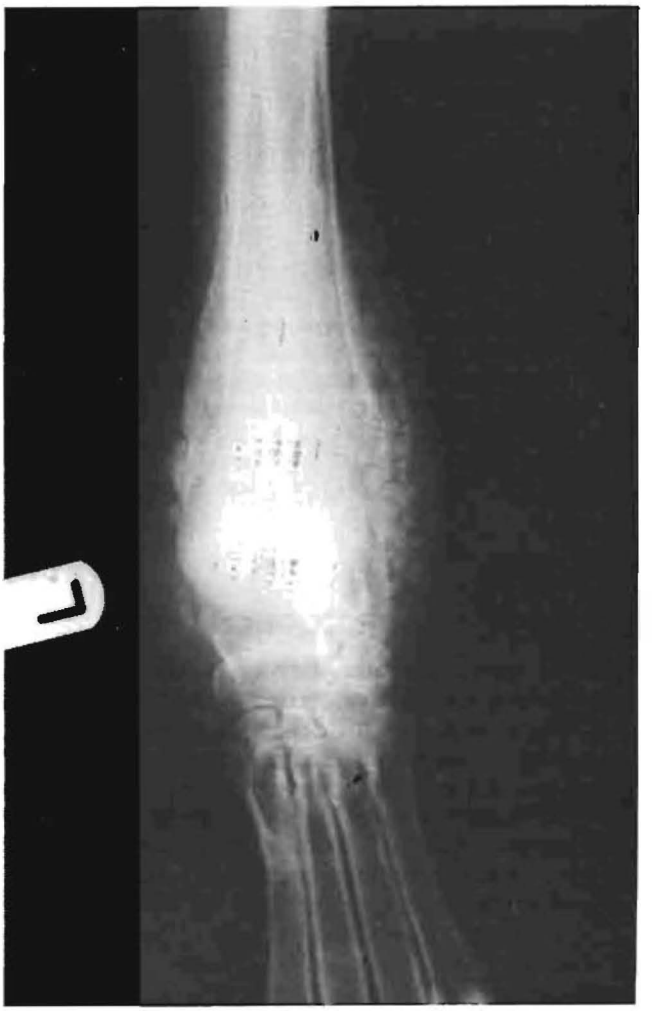

Fig. 2: Later stage of the osteosarcoma development.

The left prescapular lymph node and a section of the left diaphragmatic lung lobe were removed after the animals death for microscopical examination which revealed the presence of metastatic foci of osteogenic sarcoma.

DISCUSSION

This case is an extremely interesting one in view of the presence of an osteogenic sarcoma in a dog of such a young age[1]. It tends to highlight this possibility when considering the aetiology of lameness, especially as these problems are fairly frequently encountered in large breeds such as Great Danes[1,2].

The main differential diagnoses which should be considered are briefly as follows:

Panosteitis: This is an inflammatory bone condition, producing densities in the medullary cavity and possibly a very slight periosteal reaction, but there is no cortical destruction and it occurs near the nutrient foramen.

Osteomyelitis: This refers to any infective process occurring within the bone. It can occur anywhere in a bone and if it is present at the extremity of a bone, it tends to spread across the joint and into adjacent bones[3]. It will first be seen as a roughening of the trabecular outline, and later there will be evidence of bone absorption and

some reactionary bone formation. As a result of lifting of the periosteum some of the new bone formation may be external to the bone[3]. The process is essentially a destructive one and osteolysis is the prominent feature[3]. Bone sequestra may be seen.

Other bone tumours: *(a) Benign bone neoplasia.* Osteomas are rare in animals and are seen as localised regions of new bone formation, not involving other bones or adjacent soft tissues.

(b) Malignant bone neoplasia. These are relatively common in older dogs but rare in young dogs[1]. They can be difficult to differentiate from other bone conditions, e.g. osteomyelitis[3]. The lesion is usually confined to one bone but may spread to adjacent bones although rarely across joint spaces. Simultaneous areas of bone formation and bone absorption may be seen and the tumour may subsequently become classified as "osteogenic" or "osteolytic" if one or other of these processes becomes predominant.

Fibrosarcoma and chondrosarcoma are bone destructive and do not produce any visible periosteal reaction and new bone formation.

Osteosarcoma is typically highly osteolytic and causes extensive bone destruction. A small triangle of new bone formation may be seen external to the cortex at the edge of the lesion. This is the so-called "Godman's Triangle" and consists of bone development resulting from periosteal elevation caused by the tumour. It is considered typical of malignant bone neoplasia[3].

Metastases may be found in the regional lymph nodes and lungs, or more rarely in other bones, liver or kidneys.

ACKNOWLEDGEMENTS

I would like to gratefully acknowledge the assistance of Prof. C.J. Roos, Dr S. Burrows and Dr J. Duncan-Taylor. Their help with the radiograph interpretation, the surgery and the histology, respectively, was invaluable.

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BOOK REVIEW **BOEKRESENSIE**

COMPARATIVE VERTEBRATE ENDOCRINOLOGY

P.J. BENTLEY

2nd ed. Cambridge University Press, Cambridge. 1982. pp. XV + 485. Figs. 198. ISBN 0 521 28878 9 (paperback). Price: R30,45.

As its name implies, the book deals with comparative endocrinology of vertebrates. The evolution of endocrine glands and hormones are discussed in relation to the phylogenetic origin and the environmental adaptation of the different vertebrate species.

Special reference is made to hormones and calcium metabolism, the integument, osmoregulation and reproduction. A comprehensive list of references is included which greatly adds to its value to the researcher. From a veterinarians point of view, however, the book has little to offer as hardly any reference is made to domestic animals.

J.F.W. Grosskopf

PRAKTIESE ELEKTROKARDIOGRAFIE. 3. HIPERKALEMIE BY DIE HOND EN KAT

J. VAN HEERDEN*

ABSTRACT: Van Heerden J. **Practical electrocardiography 3. Hyperkalaemia in the dog and cat.** *Journal of the South African Veterinary Association* (1983) 54 No. 4, 274-276 (Afrik.). Department of Medicine, Faculty of Veterinary Science, Medical University of Southern Africa, 0204 Medunsa, Republic of South Africa.

The causes, electrocardiographic diagnosis and treatment of hyperkalaemia in the dog and cat are briefly discussed.

Key words: hyperkalaemia, dog, cat.

'n Gebrek aan kalium ontstaan selde by die gesonde dier omdat kalium goed verteenwoordig is in die meeste voedselsoorte. Absorpsie van kalium vanuit die spysverteringskanaal vind gewoonlik plaas as gevolg van 'n konsentrasie-gradiënt wat beperk word deur die elektron-negatiewe milieu in die lumen van die dermkanaal. 'n Aktiewe pomp-meganisme in selwande dra aktief by tot die veel hoër intra-sellulêre konsentrasie van kalium-ione. Die regulering van plasmakaliumkonsentrasie binne nou perke word hoofsaaklik teweeggebring deur aldosteroon, 'n produk van die bynierskors.

Omdat kalium goed verteenwoordig is in die meeste diëte en maklik vanuit die dermkanaal opgeneem word, is die liggaam van die meeste soogdiere goed daarop ingestel om kalium uit te skei. By meeste soogdiere wissel bloedkalium konsentrasies baie min as gevolg van sodanige doeltreffende uitskeiding van kalium. Wanneer die bloed kalium konsentrasie styg het dit gewoonlik ernstige en selfs noodlottige gevolge vir hartspiersametrekking in meeste soogdiere. Die onmiddellike oorsaak van dood is gewoonlik 'n noodlottige verandering in die geleiding-sisteem van die hart[3].

'n Gebrek aan kalium by die hond kan hoofsaaklik toegeskryf word aan maagdermkanaalsteurnisse (wat absorpsie verminder) en oormatige uitskeiding deur die niere (diurese).

Hiperkalemiese toestande ontstaan basies as gevolg van gebrekkige uitskeiding, oormatige vrystelling vanuit selle en oormatige toediening (iatrogenies) van dié katioon.

Die mees algemene kliniese toestande in die hond en kat wat lei tot 'n hiperkalemie is die volgende:

1) gebrekkige urienvorming en uitskeiding soos gesien met byvoorbeeld akute nierontsteking en obstruksie van die uretra
2) onderafskeiding van aldosteroon (Addison se siekte) – aldosteroon bevorder die terughouding van natrium en uitskeiding van kalium
3) asidotiese toestande – hoë waterstof-ioon konsentrasies lei tot verplasing van kalium vanaf intra- na ekstra-sellulêre vlak
4) anoksiese toestande – die geassosieerde daling in suurheidsgraad van die bloed en/of die wanfunksionering van die Na^+K^+ pompsisteem mag uitlekking van kalium-ione vanuit selle teweegbring
5) beskadiging van selle soos veral gesien met grootskaalse spierskade
6) oormatige kalium toediening soos byvoorbeeld met die gebruik van poli-ioniese oplossings intraveneus in pasiënte met slegs gedeeltelike funksionering van die niere.

Die kliniese diagnose van gevorderde hiperkalemie in die hond en kat is gewoonlik gebaseer op die vasstelling van 'n disritmie (bradi-aritmie of bradikardie) en die herkenning van geassosieerde orgaansiektes soos byvoorbeeld uretrale blok in die kat en hipoadrenokortisisme in die hond. Bevestiging van die kliniese diagnose van hiperkalemie geskied hoofsaaklik deur bepaling van serum kalium konsentrasies of deur middel van 'n elektrokardiogram. Die bepaling van die konsentrasie van kalium in die serum vereis gewoonlik gespesialiseerde tegniek en apparaat buite bereik van die algemene praktisyn. Die elektrokardiogram daarenteen bied 'n tegniek binne bereik van die algemene praktisyn waarvolgens 'n hiperkalemie op 'n praktiese wyse vasgestel kan word.

'n Opeenvolgende reeks van veranderinge vind plaas in die elektrokardiogram namate die serumkalium konsentrasies styg (Fig. 1):

1) die T-golf vergroot in amplitude en die vorm daarvan verander sodat dit skerper vertoon
2) die amplitude van P- en R-golwe neem af
3) die tydsduur van die QRS-kompleks en P-R interval neem toe
4) S-T segment onderdrukking tree geleidelik in
5) met kaliumkonsentrasies van hoër as 8,5 mmol/ℓ verdwyn die P-golf heeltemal en die ritme is dan gewoonlik 'n stadiger sinoventrikulêre ritme
6) die terminale ritme kan beskryf word as of ventrikulêre asistolie, ventrikulêre fladdering of ventrikulêre fibrillasie.

Elektrokardiografiese abnormaliteite stem egter nie baie goed ooreen met die absolute konsentrasie van serum kalium nie[3]. 'n Gegewe serum kalium konsentrasie is meer toksies by 'n hoë verhouding van ekstrasellulêre kaliumkonsentrasie tot intrasellulêre kaliumkonsentrasie. Die toksisiteit van so 'n hoë kaliumkonsentrasie word vererger deur lae ekstra-sellulêre kalsium en natrium konsentrasies asook 'n lae suurheidsgraad[4].

Die opeenvolgende veranderinge in die elektrokardiogram met 'n styging in bloedkaliumkonsentrasie asook die elektropatofisiologie daarvan word meer breedvoerig uiteengesit deur Coulter, Duncan en Sander[1], Fisch[2],

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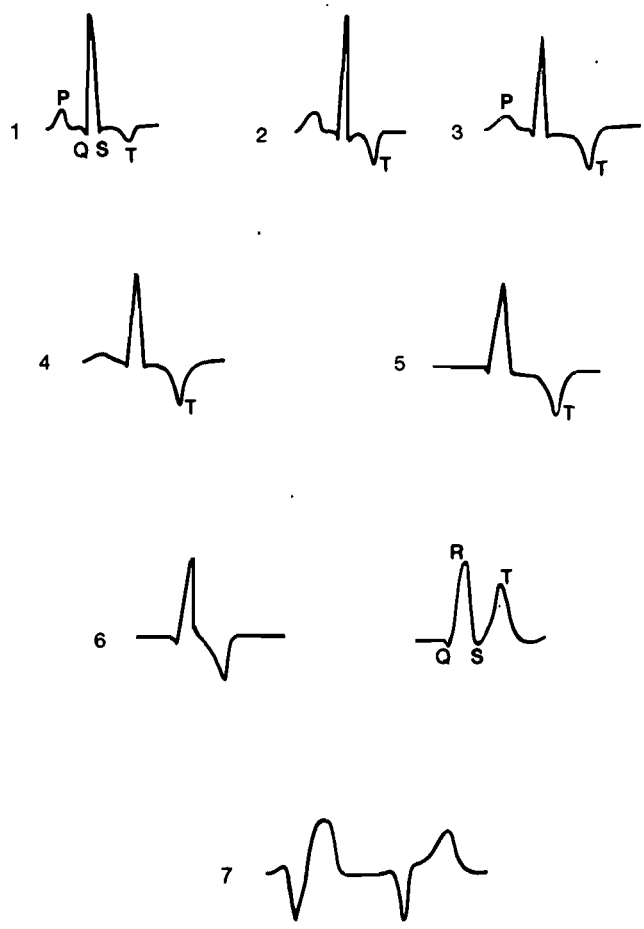

Fig. 1: 'n Voorstelling van opeenvolgende veranderinge (1-7) in die elektrokardiogram met 'n progressiewe styging in bloedkaliumkonsentrasies. 1. Normale kompleks soos verkry met afleiding II. 2. Prominente T-golf. 3. Prominente T-golf, verlenging van die tydsduur van die QRS-kompleks, verlenging van die QT-interval. 4. Verkleining van die amplitude van die P-golf. 5. Afwesigheid van die P-golf. 6. ST-segment onderdrukking; T-golf baie prominent en óf positief óf negatief. 7. Sinoventrikulêre ritme; geweldige verwyding van die QRS-kompleks; abnormale QRS-kompleks.

Schamroth[5], Tilley[6], en Van der Ark, Ballantyne en Reynolds[7].

Met die behandeling van hiperkalemie in die hond en kat word daar gepoog om bloedkaliumkonsentrasie te verlaag deur

a) verskuiwing van die katione na die intrasellulêre vlak
b) uitskeiding deur die niere en
c) verminderde absorpsie vanuit die dermkanaal.

Hierdie behandeling word gewoonlik in samehang en met inagneming van die onderliggende oorsaak van die toestand toegepas. Die behandeling van bloot net die ernstige kardiese disritmie sluit stapsgewys die volgende in:

a) 'n roete vir die intraveneuse toediening van vloeistowwe en/of geneesmiddels word geskep en daar word onmiddellik begin met die toediening van 'n 5 %-dekstrose oplossing – verhoging van bloedsuikerkonsentrasie prikkel die afskeiding van insulien wat op sy beurt die opname van kalium deur die selle bevorder. Dekstrose-toediening bevorder ook diurese wat die uitskeiding van kalium bevorder
b) die voortdurende monitor van die pasiënt met 'n elektrokardiogram
c) die intraveneuse toediening van natrium bikarbonaat teen 'n geskatte dosis van 1-2 milli-ekwivalente per kg – die onderdrukking van impulsgeleiding in die hart tydens hiperkalemie is tot 'n mate omkeerbaar deur die toediening van natrium. Die bikarbonaat verlig voorts die asidotiese toestand en bekamp waterstof-kalium ioon-omruiling.

In meeste gevalle sal die behandeling soos beskryf onder a – c voldoende wees.

d) As noodbehandeling in terminale gevalle, word die intraveneuse toediening van kalsiumglukonaat of kalsiumlaktaat aanbeveel. Kalsium-toediening prikkel miokard sametrekking. Kalsium verhoog die membraan potensiaal van enige prikkelbare neuromuskulêre weefsel en het as sodanig die teenoorgestelde effek as 'n oormaat kalium.
e) die intraveneuse toediening van insulien in kombinasie met 'n dekstrose oplossing en natriumbikarbonaat sal die verskuiwing van kalium na die binnekant van die sel verhaas.

Tydens behandeling vir 'n gevorderde hiperkalemie moet die pasiënt verkieslik met behulp van 'n elektrokardiogram gemonitor word. Die spesifieke behandeling van so 'n pasiënt sal bepaal word deur die onderliggende oorsaak. Kalsium sal byvoorbeeld liefs nie gebruik word in pasiënte wat onder behandeling met digitalis is nie. Desoksikortikosteroon en glukokortikoiede mag 'n belangrike komponent wees in die behandeling van pasiënte met adrenokortikale hipofunksie. In meer chroniese gevalle van hiperkalemie mag die gebruik van orale geneesmiddels wat kalium-opname verminder (soos lakseermiddels en sekere harse) oorweeg word. Lediging van die blaas in hiperkalemiese katte met uretrale obstruksie sal van kardinale belang wees.

Die volgende twee gevalle illustreer die gebruik van die elektrokardiogram in algemene praktyk:

Pasiënt 1: Die kat is opgeneem in 'n toestand van algemene ineenstorting, gevorderde dehidrasie en met 'n geweldig oorvulde blaas. Met nadere ondersoek is 'n volledige obstruksie van die uretra in die penis vasgestel. 'n Elektrokardiogram het die kliniese vermoede van 'n hiperkalemie bevestig (Fig. 2). Die elektrokardiografiese diagnose is bevestig deur die vasstelling van 'n bloedkaliumkonsentrasie van 8,5 mmol/ℓ in die laboratorium.

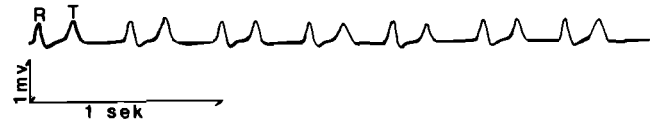

Fig. 2: Prominente T-golwe in die elektrokardiogram (Afleiding II) van 'n kat met 'n bloedkaliumkonsentrasie van 8,5 mmol/ℓ.

As noodbehandeling is die blaas onmiddellik dreineer deur middel van sistosentese. Daarna is 'n 5 % dekstrose-oplossing stadig intraveneus toegedien. Twee ure nadat begin is met die intraveneuse infusie van dekstrose het die kat klinies dramaties verbeter. Die afwesigheid van prominente T-golwe in die elektrokardiogram op hierdie stadium was opvallend (Fig. 3).

Die elektrokardiogram (Fig. 2) in hierdie pasiënt het die volgende uitstaande kenmerke van 'n hiperkalemiese toestand getoon nl. 1) geweldig prominente T-golwe wat maklik verwar kan word met QRS-komplekse en 2) die totale afwesigheid van P-golwe.

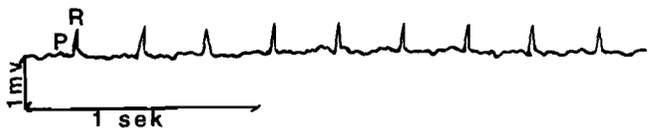

Fig. 3: Verdwyning van T-golwe na die toediening van 'n dekstrose-oplossing.

Die pasiënt het uiters gunstig reageer op die intraveneuse toediening van slegs dekstrose. Sommige outeurs beveel die gebruik van intraveneuse insulien aan as deel van die noodbehandelingsprofiel. Wanneer insulien gebruik word moet dit egter na of met dekstrose-behandeling geskied ten einde hipoglusemie te verhoed.

Pasiënt 2: Hierdie hond is opgeneem in 'n terminale toestand van kollaps met 'n geskiedenis van behandeling met 'n poli-ioniese oplossing intraveneus nadat 'n unilaterale nefrektomie 'n dag tevore op die pasiënt uitgevoer is. Die pasiënt het 'n hartspoed van 25 per minuut gehad. Die pasiënt wat vermoedelik aan hiperkalemie gelei het, is dood voordat geskikte behandeling uitgevoer kon word.

'n Elektrokardiogram (Fig. 4) wat van hierdie hond geneem is het die volgende uitstaande kenmerke getoon:

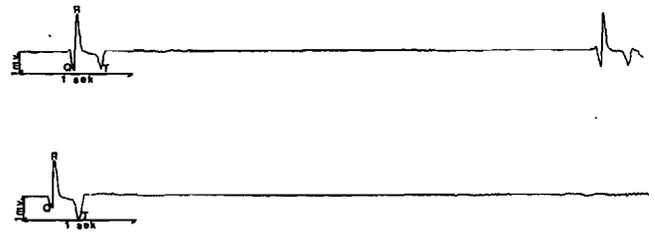

Fig. 4: Afleiding II. Uitgesproke bradi-aritmie, prominente T-golwe, wye QRS-komplekse en afwesigheid van P-golwe in 'n terminale geval van verdagte hiperkalemie.

1) uitgesproke bradi-aritmie
2) afwesigheid van P-golwe
3) prominente T-golwe
4) wye QRS-komplekse

Die verhoging van kaliumkonsentrasie in die bloedstroom van die pasiënt skep 'n gevaarlike noodtoestand waarby die lewe van die pasiënt in gevaar gestel word. Vroegtydige herkenning gevolg deur korrekte behandeling van hierdie elektrolietsteurnis is van kardinale belang vir die klinikus.

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OPPERVLAKKIGE HORINGVLIES EDEEM

S.W. PETRICK*

ABSTRACT: Petrick S.W. **Superficial corneal oedema.** *Journal of the South African Veterinary Association* (1983) **54** No. 4, 277-278 (Afrik). Department of Surgery, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

A 4-year-old Miniature Pinscher was presented with bilateral cataracts which were surgically successfully removed. Severe bilateral superficial corneal oedema developed within 6 days. A bilateral superficial keratectomy and membrana nictitans flaps cured the disease. It was found that after the lentectomy the owner had used a hypertonic salt solution as an eyewash.

Key words: corneal oedema, keratectomy, dog.

GESKIEDENIS

'n Miniatuur Doberman Pincher, 4 jaar oud en gesteriliseer, was op 18 Januarie 1983 verwys met bilaterale katarakke wat oor die afgelope jaar ontwikkel het.

EERSTE BEHANDELING

'n Bilaterale intrakapsulêre lentektomie is suksesvol uitgevoer. Twee dae later is die pasiënt huis toe gestuur met 'n voorskrif vir 'n antibiotikum en kortisoon salf en 'n midriatikum wat vir 4 weke gebruik moes word.

EERSTE HERBESOEK

Ses dae later is die pasiënt heropgeneem weens erge verslegting in die toestand van beide oë. Aanvanklik het dit voorgekom asof beide oë oopgebars het met 'n totale inhoudsverlies (Fig. 1). 'n Noodoperasie is op beide oë uitgevoer

Fig. 1: Die oog 6 dae na ontslag van die pasiënt.

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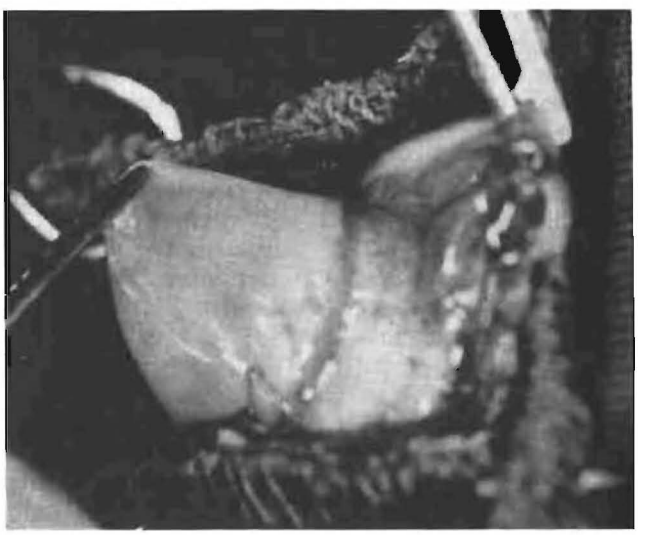

Fig. 2: Die oog met oppervlakkige horingvlies edeem.

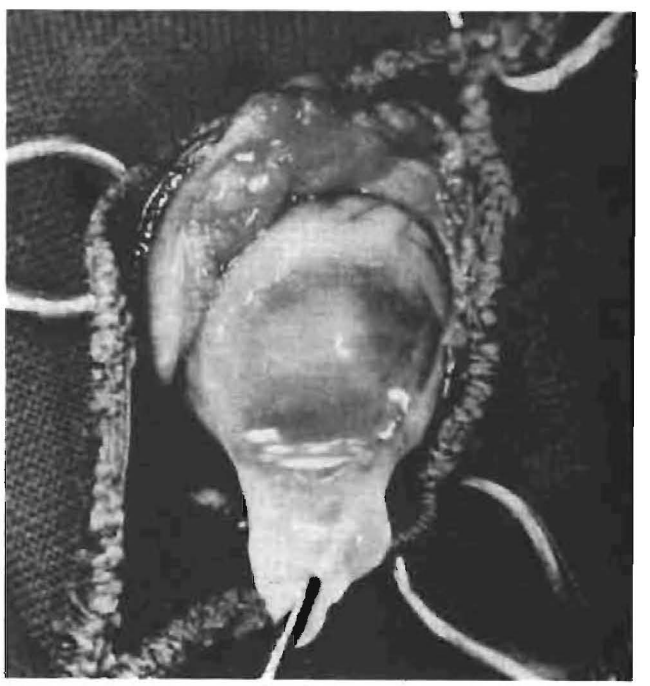

Fig. 3: Die oog na bykans voltooide oppervlakkige keratektomie.

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TWEEDE BEHANDELING

Na versigtige skoonmaak en voorbereiding vir horingvlieswondhegting, is gevind dat beide oë heel was en dat die massas tussen die ooglede niks meer as geweldige oppervlakkige edeem van die horingvliese was nie (Fig. 2).

'n Bilaterale oppervlakkige keratektomie is uitgevoer (Fig. 3) om die oppervlakkige edemateuse horingvliese te verwyder. In beide oë is die membrana nictitans oorgetrek en teen die boonste ooglede geanker.

Die dier is die volgende dag ontslaan met 'n antibiotikum salf as voorskrif en die opdrag om die pasiënt na tien dae terug te bring.

TWEEDE HERBESOEK

Met dié besoek is die steke uit die boonste ooglede verwyder om die membrana nictitans te bevry. Die horingvliese was op die stadium bykans normaal.

BESPREKING

Die na-operatiewe behandeling van die pasiënt na die lentektomie was noukeurig met die eienaar bespreek om seker te maak dat die regte voorskrif gebruik was.

Volgens die eienaar was alles presies gedoen soos voorgeskryf behalwe dat hy op eie inisiatief die oë daagliks met 'n soutoplossing van onbekende sterkte uitgespoel het.

GEVOLGTREKKING

Dit is bekend dat 'n hipertoniese soutoplossing die horingvlies sal dehidreer.

Dit blyk dat in hierdie pasiënt 'n baie gekonsentreerde soutoplossing verantwoordelik was vir vinnige dehidrasie en aansameling van vloeistof in die oppervlakkige lae van die horingvliese.

CASE REPORT **GEVALVERSLAG**

THE USE OF EQUINE ANTI-ENDOTOXIN HYPERIMMUNE SERUM** IN THE TREATMENT OF SEPTIC ARTHRITIS IN FOALS

MARIANNE THOMSON*

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Three thoroughbred foals were treated with anti-endotoxin hyperimmune serum. The serum was injected into the affected joint spaces. Two of the foals made a complete recovery.

Key words: Equine anti-endotoxin hyperimmune serum, septic arthritis.

INTRODUCTION

The use of equine anti-endotoxin hyperimmune serum** for its therapeutic properties in purulent arthritis in 3 Thoroughbred foals is described. Gaffin et al. state that "The serum has an anti-endotoxin IgG concentration of 160 mg/ml[2]. These antibodies were found not only to bind to "free" endotoxins from a wide range of Gram negative organisms, but also to bind to the endotoxin present in the outer coat of these bacteria. This causes complement activation, which in turn lead to the lysis and killing of these bacteria"[1].

They have administered the serum intravenously to successfully treat endotoxic shock resulting from foal diarrhoea and have also used it as a preventative measure in combating outbreaks of foal diarrhoea. It has, in addition, been administered directly into the uteri of 2 mares for the local treatment of *Klebsiella* metritis.

The following case reports will demonstrate how the author administered the serum intra-articularly into affected joints of foals suffering from purulent arthritis.

Case 1

A 30-day-old Thoroughbred filly was presented with acute lameness in the near stifle joint. The affected joint capsule was markedly distended. The filly had been born 31 days premature to a progesterone deficient dam. (From the 6th month of pregnancy the mare had been on a regimen of medroxy progesterone acetate (Depo Provera, Upjohn; 500 mg intramuscularly (i.m.) on a weekly basis) and melengestrol acetate (M.G.S., Upjohn; 200 mg/kg in soybean meal; 70 g on a daily basis in the food)).

The foal's rectal temperature was 40°C. She was anorectic and depressed.

An aseptic aspirate of the synovial fluid revealed a turbid, straw-coloured fluid that clotted in the syringe. A smear stained with Wright's Stain showed numerous neutrophils and cocci-type organisms. No organisms could be cultured from a swab sent to a laboratory in Ames medium.

The joint was flushed via a 16 gauge canula with Ringer's Lactate until the withdrawn fluid appeared clear to the eye (i.e. after about 800 ml had been used). A mixture containing 80 mg gentamycin (Baramycin, Schering) in 2 ml solvent plus 10 ml of a 4,2 % sodium bicarbonate solution was instilled into the joint.

Parental therapy was immediately initiated. Kanamycin (Kanamycin, TAD, Natterman) 500 mg three times daily i.m. and amoxicyllin (Clamoxyl, Beecham) 1,5 g twice daily i.m. for 5 days.

Days 2 and 3 The filly's rectal temperature was 38,5 °C and she was only slightly lame. She was given flunixin meglumine (Banamine, Schering) 2 ml intravenously (i.v.) on each of the 2 days as an analgesic.

Day 4 The patient was severely lame and the joint grossly distended. Rectal temperature was 39 °C. Her plasma IgG was 260 mg/ml (Miles Laboratories Horse IgG Test Kit). Plasma from 3 mares on the same farm was mixed in roughly equal quantities and 1,85 ℓ of this plasma was administered intravenously. Immediately prior to the plasma infusion, the foal received an intravenous administration of prednisolone sodium succinate (Solu-Delta-Cortef, Upjohn; 100 mg).

She was deeply sedated by the i.v. administration of a solution of 5 % GGE (Glyceryl Guaiacol Ether) in 10 % Dextrose Saline to effect. The joint was aseptically flushed with 1 ℓ of Ringer's lactate solution at body temperature. Ten million i.u. of Crystalline penicillin G was injected into and left in the joint. A smear of the joint fluid showed no reduction in the number of bacteria and neutrophils and the fluid had an offensive odour.

Day 5 There was a mild improvement and the rectal temperature was 38,5 °C.

Day 6 Her temperature was 39 °C, she was very lame and only nursed after administration of Banamine (2 ml i.v.).

The synovial fluid was still turbid, straw coloured and clotted in the syringe. Wright's stain of a smear of the fluid showed numerous neutrophils and bacteria. A sample for bacterial culture was taken, but no growth was obtained at the referal laboratory.

The joint was flushed with 1 ℓ Ringer's lactate solution and a mixture containing 80 mg gentamycin (Gentocin, Schering) and 10 ml of a 4,2 % sodium bicarbonate solution was instilled into the joint.

The parenteral antibiotic therapy was changed to one

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**Equine Anti-Endotoxin Hyperimmune Serum produced by S.L. Gaffin, B. Baker et al. University of Natal Medical School, Durban, South Africa.

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consisting of trimethoprin and sulphadoxine (Borgal, Hoechst) at the appropriate dosage rate, administered i.v. once daily.

Days 7-13 The joint was flushed once daily via a 16G canula after scrubbing the skin with Betadine for 5 minutes. Ringer's lactate solution was used for flushing and this was continued until the aspirate appeared clear and colourless which took approximately 0,5-1 ℓ on each occasion.

A mixture containing 80 mg gentamycin in 2 ml solvent plus 10 ml 4,2 % sodium bicarbonate, to correct the pH, was left in the joint after flushing.

During this period no sedation was necessary. In addition 7 ml of a preparation containing 40 mg trimethoprin and 20 mg sulphadoxine per ml (Borgal, Hoechst) was administered i.v. once daily. Banamine 2 ml i.v. was used on occasions to alleviate pain and encourage nursing. A multivitamin syrup was administered orally.

During this whole period the mare and foal were confined to a large stable with deep, clean bedding. The mare was given ample green food and on a few occasions she was milked out when her udder became distended.

Day 14 There was, after all this time, no sign of improvement in the status of the foal. On this day the joint fluid was a reddish colour, offensive and still rife with neutrophils and bacteria.

The use of Borgal was discontinued and a very gloomy prognosis was given. It was estimated that she had not gained any body mass at all in the previous 14 days.

The joint was once again flushed with 1 ℓ Ringer's lactate solution. (The foal by now so used to this that only minimal effort was needed to get her to lie down and almost no restraint was needed.)

125 ml Equine Anti-endotoxin Hyperimmune Serum plus 250 mg gentamycin (Gentocin, Schering) was injected into the stifle joint. This volume distended the joint very firmly, as intended; the rationale behind this being to induce the serum to penetrate into all synovial crypts.

The filly was in great pain when allowed to rise and received 2 ml Banamine and 3 ml of an anti-inflammatory agent containing phenylpyrazolidine i.v. (Tomanol, Hoechst).

Day 15 No treatment. The foal was still lame but her habitus was markedly improved.

Day 16 120 ml Anti-Endotoxin Hyperimmune Serum plus cephaloridine (Ceporan, Glaxo, 500 mg in 5 ml sterile water) was injected into the joint in the morning. In the evening she received another 500 mg of Ceporan in the joint. Banamine was given for pain relief.

Day 17 The joint was hardly distended and synovial fluid had a clear pale appearance, had a lubricant feeling and contained only a few pyknotic neutrophils and no bacteria. 500 mg Ceporan was instilled into the joint.

The foal was "bouncy" and only very slightly lame.

Day 18 Neither joint distension, nor lameness was evident. 500 mg Ceporan was injected into the joint.

From the next day on the foal received no further treatment except for oral multivitamins. She has remained sound to date and has kept pace in growth with her contemporaries.

Case 2

Day 1 An 8-week-old Thoroughbred colt was presented with acute lameness in the near hindlimb. No joint distension or lesions were visible and no treatment was administered.

Day 2 The consulting veterinarian to the Stud examined the colt. Body temperature was 41 °C, he was very lame with no weightbearing on the affected leg and was not nursing. No joint distension was visible.

He received 5 ml of a preparation containing trimethoprin and sulphadiazine (Tribrissen 48 %, Wellcome) intramuscularly once daily until Day 6.

The foal remained lame and nursed only when Butazolidone was administered to relieve pain.

Day 6 The consulting veterinarian aspirated the left stifle joint and withdrew a few clots of inspissated pus. The joint was not distended. The rectal temperature remained elevated and the foal was rapidly losing condition.

Day 7 The author was called to examine the foal at the request of the consulting veterinarian. The joint was prepared for aseptic flushing and aspiration by shaving and scrubbing with Betadine for 5 minutes.

No fluid could be withdrawn through a 16 Gauge canula. 200 ml Ringer's Lactate solution was infused into the joint but could only be aspirated with great effort. The canula repeatedly blocked with inspissated pus.

125 ml Equine Anti-Endotoxin Hyperimmune Serum plus 200 mg Gentamycin in 5 ml solvent was injected into the joint space. This, plus the remaining Ringer's Lactate solution very fully distended the joint. The severe pain after this overextension of the joint, was relieved with 2 ml Banamine and 3 ml Tomanol i.v. The Tribrissen was continued.

Day 8 Tribrissen and Butazolidin continued. Habitus of foal improved.

Day 9 The consulting veterinarian repeated the instillation of 120 ml Anti-Endotoxin Hyperimmune Serum plus 200 mg Gentamycin.

Tribrissen i.m. was repeated.

Day 10 Tribrissen 48 % 5 ml i.m.

Day 11 The foal was perfectly sound and nursing well. He was returned to pasture with his dam and to date, 8 months later, continues to do well and has remained sound.

Case 3

A foal with multiple joints affected by septic arthritis was treated by a colleague. The author never personally saw this foal, but supplied the Serum and described the method used in the previous 2 cases to the veterinarian concerned.

This foal received 250 ml of the Serum intravenously and a total of 250 ml divided into smaller doses was also injected into affected joints. Gentamycin was added to the intra-articular serum injections.

The response was unsatisfactory. Subsequently a *Staphylococcus* sp. was cultured and some response to chloramphenical was obtained. The foal then left with his dam for another part of the country and further follow-up has been unsatisfactory and vague. The poor response in this case was probably due to the fact that a Gram positive organism was present in the joint.

SUMMARY

The author is gratified by the results obtained by this rather novel and unconventional treatment of purulent arthritis. It is felt that anti-endotoxin hyperimmune serum is of benefit when used with strict aseptic techniques in conjunction with standard methods of combating this crippling condition.

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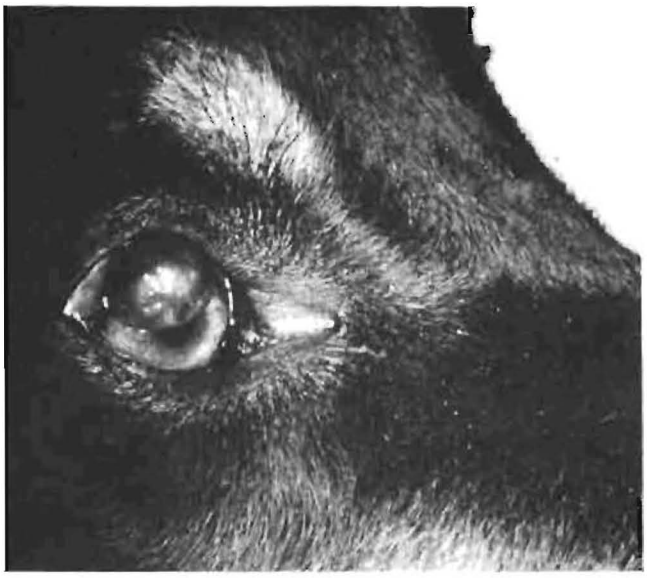

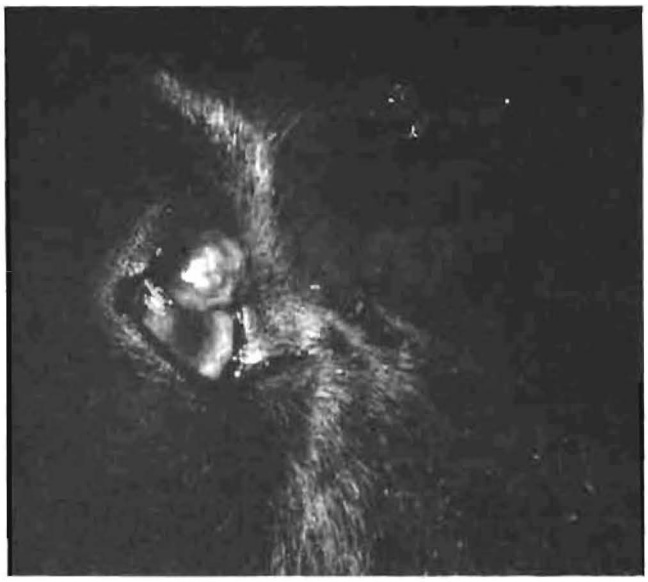

OKULÊRE MELANOOM IN 'N HOND

'n Dobermann reun van 18 maande was ingebring met 'n groeisel op die regter oog. Die ooglede kon nie meer sluit nie en die gevaar van horingvliesuitdroging het reeds bestaan.

Klinies is 'n melanoom op die dorsale limbus van die oog gediagnoseer.

Die gewas is suksesvol met 'n gedeeltelike oppervlakkige keratektomie verwyder en histologies as 'n melanoom bevestig.

Ingestuur deur: Prof. S.W. Petrick, Departement Chirurgie, Fakulteit Veeartsenykunde, Universiteit van Pretoria, Posbus 12580, 0110 Onderstepoort.

OCULAR MELANOMA IN A DOG

A Doberman male, aged 18 months, was admitted with a growth on the right eye. Closure of the eyelids was not possible and the danger of corneal dehydration already existed.

A melanoma on the dorsal limbus of the eye was diagnosed clinically.

With a partial superficial keratectomy the tumour was successfully removed and a melanoma histologically confirmed.

Submitted by: Prof S.W. Petrik, Department of Surgery, Faculty of Veerinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort.

TO THE EDITOR

AAN DIE REDAKSIE

UNCINARIA STENOCEPHALA IN DOGS IN THE REPUBLIC OF SOUTH AFRICA

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Naturally infested dogs from various sources near East London were used in a trial on the efficacy of oxfendazole against the nematodes of canines. These animals were all heavily infested with the whipworm, *Trichuris vulpis* (Frölich, 1789), as well as 2 hookworms, *Ancylostoma caninum* (Ercolani, 1859) and *Uncinaria stenocephala* (Railliet, 1884).

This appears to be the first record of *U. stenocephala* in the Republic of South Africa.

AAN DIE REDAKSIE

TO THE EDITOR

VEEARTSENYKUNDIGE DIENSTE – DEPARTEMENT LANDBOU OF GESONDHEID?

Op die oomblik resorteer veeartsenykundige dienste in die staatsopset onder die Departement Landbou. Die vraag ontstaan of veeartsenykundige dienste nie eerder tuishoort onder die Departement Gesondheid nie? Ons bespreek hierdie vraag aan die hand van die volgende stellings.

Stelling I. Departement Landbou en Veeartsenykundige Dienste het 'n historiese band

Die verbintenis tussen Departement Landbou en Veeartsenykundige dienste kom moontlik uit die geskiedenis, omdat veeartsenykundige dienste aanvanklik grotendeels op plaasdiere gerig was. 'n Mens herinner jouself aan die heroïese pionierswerk wat veral ten opsigte van bees- en perdesiektes om en by die vorige eeuwending, gedoen is. Perde is toe nog in 'n groot mate veral as trek- en rydiere vir landboudoeleindes gebruik. Navorsingswerk het met geen geringe prestasie uitgebrei na skaap-, vark en pluimveesiektes. Groot vrektes is bekamp. Hierdie werk is hoofsaaklik onder toesig van die staat gedoen en dit is gesien as dienste wat aan die landbou diensbaar is. So is die veebedryf bestendig en uitgebrei. Ten spyte van hierdie voordelige band wat veeartsenykundige dienste met Landbou gehad het, het veeartsenykundige dienste nie die volgehoue voorsiening van Landbou ontvang ten opsigte van vergoeding, uitbreiding en vernuwing nie.

Soos groot uitbreke van veevrektes deur die jare verminder het, so blyk dit of die belangstelling van die Departement Landbou in veeartsenykundige dienste afgeneem het. 'n Mens sou amper kon sê dat die sukses wat hierdie afdeling behaal het, tot sy nadeel begin strek het. Dit is net so dat as die siektes verminder, verminder die bekommernis daaroor ook. Om hierdie siening te ondersteun, haal ons graag aan uit 'n referaat wat Prof. C.F.B. Hofmeyr gelewer het voor die Suid-Afrikaanse Akademie vir Wetenskap en Kuns[2]. Hy wys op die agteruitgang in die voorsiening van die staat vir veeartsenykundige dienste en sê "laat ons 'n enkele siekte neem as illustrasie van hoe ons agterraak". Hy sê dat Kenia al in 1964 'n bek-en-klouseer instituut geopen het, terwyl so 'n eenheid in Suid-Afrika eers in 1980 voltooi is. Verder is Botswana Suid-Afrika ook voor, aangesien hulle reeds sedert 1979 bek-en-klouseer entstof produseer. Wat die opleidingsfasiliteite betref beweer prof. Hofmeyr dat die verdubbeling van studente-innames van 45 na 90 per jaar probleme met betrekking tot fasiliteite meegebring het, waaraan die staat nie onmiddellike aandag gegee het nie. Hy sê: "Hier is 'n krisis binne ons land – wat staan nog van dienslewering buite ons grense."

Prof. Hofmeyr konstateer "soms kry 'n mens die indruk dat daar verwag word dat die reputasie en momentum wat die veterinêre onderwys, navorsing en velddienste in die verlede behaal het, onbepaald sal voortduur sónder dat daar die nodige versorging van owerheidsweë is. As gevolg van owerheidsondersteuning wat dikwels te min en te laat is, is heelwat van die veterinêre inisiatief prysgegee en hierdie tendens sal voortduur onder huidige omstandighede."

En hy sluit af: "As Suid-Afrika sy vorige plek in Afrika wil herower op veterinêre gebied, is nuwe denke van owerheidsweë dringend en onontbeerlik."

Alhoewel die aandag van Departement Landbou ten opsigte van veeartsenykundige dienste verminder het, het hierdie afdeling nie stil gestaan nie. Die professie het skouspelagtig vooruit gegaan en met oorspronklikheid en vindingrykheid, wat daaraan eie is, 'n verskeidenheid nuwe terreine betree. Soos die biologiese wetenskappe ontwikkel het, is praktiese toepassings daarvan in veeartsenykundige dienste opgeneem. Verder het die professie se benadering tot die nuwe velde en selfs ten op-

sigte van landbou, verander. 'n Mens sou hierdie benaderings-verandering kon definieer as veeartsenykundige dienste wat meer medemens georiënteerd geraak het. So kom ons by ons tweede stelling.

Stelling II. Veeartsenykundige dienste is primêr 'n menslike gemeenskapsdiens

Ek dink dat die veeartsenykundige professie in ernstige filosofiese, etiese en morele dilemmas kan beland as hy die mens in sy dienslewering totaal uit die oog sou verloor, en sy lewe aflê bloot vir die welsyn van diere. Die mens kan hom nie onvoorwaardelik in diens van die dier stel asof die dier sy meerdere of gelyke is nie. Daarom is dit byna ondenkbaar dat veeartsenykundige dienste gelewer kan word sonder om die mens direk of indirek te betrek. Selfs dienste wat weens die mees humanitêre oorwegings gelewer word, word juis gelewer as gevolg van die mens se verantwoordelikheidsgevoel teenoor die skepping waaroor hy moet heers. Die mens se verantwoordelikheidsgevoel maak dus aanspraak op veterinêre dienste en nie die dier op sigself nie. Versorging van diere word gelewer vanuit hierdie verantwoordelike posisie óf waar die dier terwille van die mens se voordeel versorg word. 'n Mens kan dus uit bogenoemde sê dat veeartse nie vir diere werk nie, maar mét diere en vír mense. Dr J.B. Herrick sê in sy antwoord op die vraag wat 'n professie is: "a profession accepts as its main purpose, a contribution to society"[1]. Dr A.F. Hopkins stel dit so: "The basic ethical considerations of any profession must involve the caring and helping of other human beings"[3]. Daar is dus eers 'n menslike behoefte om van veeartsenykundige dienste gebruik te maak, voordat die dier daarby baat. Hierdie dienste kan immers nie bestaan sonder dat die mens dit ondersteun nie en kwalifiseer juis daarom as 'n professionele diens omdat dit primêr aan die mens aangebied word. Die dier is bloot die medium tot veeartsenykundige diens.

Kom ons kyk hoe lyk dit in die praktyk. Huisdiere word feitlik uitsluitlik tot die mens se eie voordeel aangehou en menslike gesondheidsaspekte speel hier 'n belangrike rol. As gevolg van hierdie noue kontak tussen mens en dier word sake soos soönoses, beserings, allergieë van groot belang vir menslike gesondheid. Aan die positiewe kant speel troeteldiere 'n belangrike rol ten opsigte van die mens se geestelike gesondheid, veral as spanningsontladingsvoorwerp en skakel terug na die natuur[6]. Dr P.R. Messent som die voordele van troeteldiere as volg op: Kameraadskap, vermoë om vriende te maak, verbeter selfbeeld van die mens, beskerming van die mens, die rol van die troeteldier in die huisgesin en met kinders, oefening saam met diere, speel saam met diere, psigologiese aspekte en psigoterapie, troeteldiere se spesifieke rol ten opsigte van senior burgers en werkende troteldiere[5]. Prof. B. Levinson sê in dié verband: "The veterinarian can no longer limit himself solely to safeguarding the physical health of the family pet. Now he must become involved in the mental health of the family whose pet he treats"[4]. Siek huisdiere wek ook spanning by die eienaar wat die mens kan beïnvloed. Ek is bewus van 'n ekstreme geval waar die eienares in die hospitaal beland het na die afsterwe van 'n 14-jarige troeteldier.

Die aanhou en bewaring van wild in reservate (en selfs in dieretuine wat bedreigde spesies beskerm) word vandag allerweë aanvaar as belangrik vir die mens se voortbestaan op die groen planeet. Daar word geargumenteer dat as die mensdom al die diere sou uitroei, daar so 'n ekologiese versteuring sal plaasvind dat die mens se voortbestaan in gevaar gestel kan word. Veeartsenykundige dienste wat in verband staan met wild en selfs marine-biologie, kan dus 'n bydrae lewer tot 'n gesonder en langer lewende mensdom.

Die perdesport (perderesiese, perdespring, ontspanningsritte, perdepolo, ens.) word vandag ook as belangrike spanningsontladings van die moderne gemeenskap gesien. Verder is veral beserings van deelnemers ook 'n gesondheidsaspek van die mens.

Proefdierwetenskappe staan direk in verband met menslike gesondheid en veterinêre dienste speel hier reeds 'n belangrike rol.

Veterinêre dienste aan diere- en welsynsorganisasies het veel te make met menslike gesondheid. Rondloperhonde kan onder andere nie net skade berokken nie, maar ook siektes versprei en uitgehongerde diere kan ook aggressief raak. Dierewelsynsorganisasies lewer ook veterinêre dienste aan minderbevoorregte mense.

Staats- en munisipale veeartse is ten nouste betrek by menslike gesondheid. 'n Mens dink hier maar net aan die tering en besmetlike misgeboorte skemas. Hierdie is belangrike voorkomingsaksies vir menslike gesondheid. Die staat se veterinêre dienste is ook betrokke by die aangeebare siektes (waarvan 'n hele aantal soönoses is). Die beheer van epidemiese siekte onder voedselvoorsienende diere deur die staat, dra ook by tot die mens se konstante beskikbaarheid van proteïene. In vleis- en suiwelprodukhigiëne lewer veterinêre dienste 'n onmisbare bydrae.

Hoe meer intensief boerdery word, soos die vark- en pluimveebedryf, voerkrale en die kuddebenadering, hoe meer sal die veearts sy regmatige rol speel in die landbousektor. Die veearts moet nie net op die laaste nippertjie uitgeroep word om 'n ekonomiese verlies vir die boer te probeer voorkom nie, maar die veearts moet eerstens daarop ingesteld wees om deur die instandhouding van dieregesondheid, menslike gesondheid te bevorder. Die veearts hoef nie in die éérste plek diens te lewer vir die landbou se ekonomiese voordeel nie. Landbou is daarop toegespits om sy produk te lewer op die mees ekonomiese wyse en teen die grootste wins. So verdien die boer sy brood. Veeartsenykundige dienste hoef nie noodwendig dieselfde ideale in die landbou te vervul nie. As die boer ekonomies baat by die bydrae wat hierdie dienste lewer – des te beter. Veeartsenykundige dienste wat vir die landbou gelewer word mag nooit sy bydrae tot menslike gesondheid uit die oog verloor nie.

Selfs veeartsenykundige dienste wat by reproduksie betrokke is behoort gerig te wees op beter gehalte en gesonder voedsel vir menslike gebruik eerder as om blóót 'n ekonomiese faktor in die landbou te wees. As voedselvoorsienende diere swak geteel of gegroei het, of aan besmetlike siektes of beserings ly, is die mens se proteïenbron daarby betrokke. Die oorwegende oogmerk moet dus wees: gesonde voedsel vir gesonde mense.

Samevattend kan mens in die algemeen opmerk dat veeartsenykundige dienste altyd 'n rol te speel het by die mens se fisiese en/of geestesgesondheid.

Stelling III. Veeartsenykundige dienste behoort as gelyke vennoot van medies en tandheelkundige dienste erken te word

Veeartsenykundige dienste soos hierbo uiteengesit kan geensins as para-mediese diens beskou word nie. Veeartse se opleiding plaas hulle ver bo para-mediese opleiding.

Veeartsenykundige dienste se bydrae tot die mens lê net op 'n ander vlak as mediese en tandheelkundige dienste. Hierdie dienste behoort dus in die breë volksgesondheidsfeer as 'n gelyke vennoot van mediese en tandheelkundige dienste erken te word.

As ons tot hierdie insigte kan kom, kan daar baie meer aanspraak gemaak word ten opsigte van samewerking, kommunikasie, professionele konsultasie en interdissiplinêre koördinasie. Net soos tandheelkunde as 'n dissipline op sy eie kan staan langs medies, so behoort veeartsenykunde ook sy plek in die ry in te neem. 'n Mens wil amper glo dat die nuwe mediese universiteit, Medunsa, 'n stap in die regte rigting geneem het om opleiding van die drie dissiplines onder een dak te plaas. 'n Mens dink ook aan die Weermag en Burgerlike Beskerming waar die drie dissiplines onder dieselfde hoof resorteer. Daar is ook die gesamentlike kongresse, veral saam met medici, wat in ons land gereël word. Op veeartsenykundige en mediese kongresse word ook van tyd tot tyd oor en weer bydraes gelewer deur medici en veeartse. Selfs die ou naam van ons vereniging, naamlik die Veterinêre Mediese Vereniging kon dalk 'n aanduiding gewees het van 'n nouer verband met die mediese veld (Vergelyk ook "Veterinary Medical Association of America"). Die Veterinêre en Mediese embleme is ook dieselfde, behalwe vir die "V" wat veeartsenykunde onderskei. Daar kan op hierdie basis beter skakeling plaasvind met onder andere die aptekersbedryf ten opsigte van oorvleuelende middels en spesieverskille, maatskaplike werkers en kliniese sielkundiges met betrekking tot troeteldiere in die gemeenskap, menslike ekoloë met betrekking tot wild- en natuurbewaring, mediese voedingkundiges met betrekking tot die vee-, vark- pluimvee- en suiwelbedrywe, ens. Veterinêre dienste kan met so 'n nuwe erkenning van sy rol as gelyke vennoot in gesondheidsdienste 'n baie groter en waardevoller bydrae lewer tot 'n gesonder mensdom.

Stelling IV. Veeartsenykundige dienste behoort onder die Departement Gesondheid te resorteer

Na aanleiding van ons voorafgaande stellings voel ons dat daar 'n belangrike regstelling vanaf die staat se kant moet kom ten opsigte van veeartsenykundige dienste. Dit het tyd geword dat hierdie dienste as volwaardige vennoot in volksgesondheid deur die staat erken word, en sy nuwe plek en rol in die verband inneem. Dit sal die regte sin en betekenis aan die professie se roeping verleen as ons tot so 'n stap kan vorder. So 'n regstelling deur die staat kan dalk lei tot ander regstellings soos behoorlike vergoeding van staatsveeartse, en toepaslike fondse vir opleiding en navorsing. Die "stiefkind" behandeling wat veeartsenykundige dienste van die Departement Landbou ontvang, behoort te verdwyn en sal veeartsenykundige dienste soos 'n "eie kind" versorg word. Om hierdie ideaal te bereik moet veeartsenykundige dienste onder die Departement Gesondheid resorteer. Is dit te ver gesog om te voorsien dat daar 'n Adjunk Minister van Gesondheid belas met Veeartsenykundige dienste gaan wees?

In 'n onlangse uitgawe van die blad SALUS wat deur die Departement Gesondheid en Welsyn uitgegee word, verskyn 'n belangrike voorblad berig. Die berig is geskryf deur prof. L.W. van den Heever en kan 'n wegbereider wees vir ons gedagtes[7]. Ons haal feitlik die hele artikel aan wat as hoofberig verskyn het onder die opskrif "'Vet' services care for public health".

"Members of the public at large, and even some of those persons who are active in community health, rarely think of the veterinarian as *a member of the health team*. Yet 'Veterinary Public Health' is a well recognised discipline which is recognised by international organisations such as the World Health Organisation as well as most national health authorities.

The WHO defines 'Veterinary Public Health' as "A component of public (community) health activities devoted to the application of professional veterinary skills, knowledge and resources to the protection and improvement of human health". Furthermore, "Veterinary public health activities involve a diverse range of functions within public health. *These reflect the broad community of interests between veterinary and human medicine and indicate the opportunities for profitable interaction.*"

With few exceptions, animals are essentially bred and maintained for the benefit of mankind. Farm animals produce essential food and fibre while the value to man's mental health of both large and small companion and recreation animals is being increasingly recognised. Being vitally concerned with all aspects of the production and health of animals, it can justifiably be said that *all veterinarians perform a public or community health function because they work with animals for man.*

More specifically, veterinarians contribute to man's physical wellbeing by

- bringing under control economically disruptive animal diseases as well as other costly but more insidious diseases of animals producing food and thus preventing malnutrition
- bringing under control diseases which are also zoonoses
- contributing to comparative and basic research which benefits human medicine
- participating in food protection and formulating many of the basic epidemiological concepts on which disease control in public health now rests
- promoting environmental hygiene
- training of workers in the health field.

In South Africa the veterinary profession has an outstanding record of contributing to the promotion of human health by controlling and eradicating diseases which hamper and prevent adequate food production, e.g. nagana and East Coast fever; dealing with diseases which are also transmissible to man, e.g. Rift Valley fever, anthrax and brucellosis and by controlling the hygiene of food of animal origin such as meat and milk. Both State and municipal veterinarians are particularly involved in the latter.

There is ample support and precedent in authoritative literature for *total involvement of the veterinarian in the promotion and maintenance of community health* and for active veterinary participation in environmental health." (Die kursivering is deur die skrywer gedoen.)

Die professie sal self moet besluit of hy sy rol moet bly vervul as 'n onder-afdeling van landbou en van tyd tot tyd deur landboukundiges sogenaamd "bedreig" word – of om as gelyke gesondheidsvennoot sy werklike bydrae te lewer. Ek glo dat die professie sy saak met die grootste oortuiging kan stel om die nodige regstelling te bewerkstellig.

Die klip is in die bos en 'n sinvolle gesprek oor hierdie stellings kan net tot die voordeel van die professie strek.

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