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THE PROBLEM WITH MASTITIS . . .

THE PROBLEM WITH MASTITIS IN OUR NATIONAL DAIRY HERD IS THAT WE ARE GETTING SIMPLY NOWHERE WITH ITS EFFECTIVE CONTROL!

If, as a country, we were ignorant of the fact that this most important and insidious erosion disease complex affects the economy of milk production as well as the quality of our milk and dairy products and that it can be effectively controlled, we might be excused. However, for decades every effort has been made to make us all **aware** of bovine mastitis. For decades, a host of authorities on the subject have spoken and written on the various aspects of mastitis. Farming journals regularly feature articles on some facet of mastitis, scientific associations and groups have organised conferences and invited speakers from other countries to show what can be achieved by organised control, open symposia have been held (and enthusiastically attended) in an effort to bring together all the parties concerned so as to precipitate action, heads of state departments and Ministers of State have made public statements indicating their concern, dairy technologists have expressed concern over the effect of mastitis on the quality of dairy products, health authorities have promulgated legislation concerning mastitic secretions in the milk supply, municipal and state veterinarians continue to advise and assist dairymen wherever the opportunity is presented, practicing veterinarians deal with the problems of their farmer clients as best they can, the veterinary pharmaceutical industry does its best to promote both therapy and prevention while research organisations look into various matters concerning mastitis and yet we still have a major mastitis problem! Why? *Because we have no organised mastitis control system in South Africa!*

The emphasis is on **organised** control. A great deal of extension work, advice, etc. is being given by a variety of qualified and unqualified persons in a number of organisations and disciplines, but such work is, with few exceptions, on an *ad hoc* basis and cannot be expected to yield the kind of results which a professionally supervised, integrated and organised control programme, directly related to quality of milk, has been shown to produce.

But to get back to the question of awareness of mastitis, one wonders whether there are still things that need to be said to make us all more aware thereof and to jolt us into action? Would it, for example, help to make some of the following known?

1. Using a relatively insensitive cow side test in 160 herds in the Transvaal and Free State, it was shown recently that an average of 20,9 % of quarters and an average of 45,5 % of cows had subclinical mastitis. In some of these herds as many as 49,3 % of the quarters and 84,2 % of the cows were mastitic.
2. In a survey of 496 herds producing industrial milk on the highveld it was found that 24,4 % were delivering milk containing somatic cells in excess of 1 000 000/ml and thus exceeding the rather lenient maximum permitted by Regulation under the Foods, Cosmetics & Disinfectants Act No. 54 of 1972. In 122 herds it was estimated that more than 32 % of udder quarters were mastitic while in only 3,6 % of herds fewer than 10 % of the quarters were affected.
3. In analysing available somatic cell counts of the fresh milk delivered to the 5 major consumer areas of the Republic it appears that 43,1 % of herds had a well established mastitis problem while in 9,4 % of herds the problem was severe to very serious. By placing the herds supplying these 5 large centres into various cell count categories, and taking into account the acknowledged probable loss in milk production for each category, it is estimated that mastitis in these herds resulted in production losses which varied from 5,3 % to almost 20 %.
4. In one province where collaboration between the municipal and state veterinarian has resulted in a voluntary mastitis control scheme being instituted, the percentage of herds producing milk which contains less than a million somatic cells/ml and thus meets present statutory requirements is significantly higher than the average in the other consumer areas. Simple identification of problem herds and gentle coercion to join the scheme has already had measurable benefit.
5. In the analysis mentioned above, an average of almost 10 % of herds were found to be producing milk which does not meet the requirements of the State Health Department regarding somatic cell content. Such milk must, of course, be considered unfit for human consumption.
6. The maximum permitted level of 1 000 000 somatic cells/ml of milk is indeed very lenient when considering the dilution factor in our large herds and many other countries have much stricter standards. It is known that herds producing milk with somatic cell counts ranging between 750 000 and 1 000 000/ml can be expected to produce almost 15 % less milk than those with normal milk cell counts. There is thus no room for complacency for dairymen whose milk meets the present requirements. It might well now be time for the authority concerned to consider decreasing the maximum permissible somatic cell content level to 750 000/ml or even less.

For those who might doubt the validity of the facts quoted above, one could refer to the appreciable range of published data elucidating in scientific detail the major problems related to mastitis in South Africa.

What is it, then, that is missing? What explanation can one offer for the rank apathy as regards specific action? Perhaps it is the lack of concerted official action by those authorities who have the powers to penalise and thus force into action those who sell milk which does not meet statutory requirements. Perhaps institution of a premium/litre of milk which contains less than a particularly low (say 250 000/ml) number of cells

would encourage, on the positive side, the dairyman to insist on the availability of a laboratory-backed professionally supervised mastitis control scheme in his area. If any thing is crystal clear in our situation it is that **it is the milk producer himself** who must become interested in and, in fact, demand realistic economic mastitis control services. How this interest can be fostered or promoted is another matter. Guidance and assistance by the State or Marketing Board, with perhaps a small levy on all milk produced in order to partially fund the programme, does not seem out of line. Close collaboration between i) authorities who by penalty or premium, can promote the farmer's interest in mastitis control and ii) the body which renders the herd mastitis control service is, of course, essential.

We pride ourselves on having a highly developed dairy industry; we breed and sometimes import cows, bulls, semen and even fertilised ova to ensure that we have dairy cows with excellent genetic production potential; we possess the know-how to feed and manage cows for maximum exploitation of that genetic potential and we have the professional knowledge and ability to effectively control bovine mastitis. Yet, if we do not fully utilise the latter ability, much of the former action is wasted. We simply must give adequate attention to that part of the cow that delivers the goods!

Everyone knows the virtues of milk and many love to extoll these virtues, particularly when a rise in the price of this essential commodity is in the offing. Such praise has rather a hollow ring about it when viewed in the light of the high incidence of disease in that very organ which produces the milk, i.e. the bovine udder. Unless this magnificient organ is kept healthy, even the best feeding, breeding and management will not provide sufficient reasonably priced milk to meet the future requirements of the rapidly growing South African population (±50 million by the year 2000!)

It is indeed greatly encouraging and well worth noting that so many of our rural practitioners are now actively engaged in promotive dairy cow health programmes which, of course, include a sensible herd approach to mastitis control. Despite their limited facilities their initiative has already borne fruit. One can hope that the more progressive dairy farmer will take note of what his practitioner can offer and make full use of his services, bearing in mind, of course, that mastitis control must become a permanent feature of dairy farming.

Concerning an approach on a wider front, however, the South African Veterinary Association has held an open symposium on mastitis control, arranged for publication of the proceedings and actively led the field to efforts to stimulate the authorities concerned into making money available and instituting mastitis control on an organised basis. To date none of these efforts have borne any obvious fruit.

It is clearly now up to the S.A. dairyman and organised agriculture to take the steps necessary to get to grips with this most pressing agricultural problem. As a developed country we simply cannot afford **not** to go into action on the mastitis problem – **now!**

BOOK REVIEW BOEKRESENSIE

PRACTICAL ANIMAL HUSBANDRY

T.K. EWER

John Wright & Sons Ltd, 42-44 Triangle West, Bristol BS8 IEX, England 1982. Pages 257. ISBN 0 85608 026 8
Price £10.50

Many specialist books on aspects of animal husbandry in different parts of the world have been published over recent years. To cover all subjects in a single book would be wasteful and impractical.

This book is presenting a contemporary outline of the main subject divisions of animal husbandry. Emphasis has been placed upon the practical implications of each.

The 8 chapters on Handling of animals; Breeding; Development; Reproduction; Feeding; Housing and Yarding; Preventive medicine and Animal Welfare, are very well balanced. Although a number of applications are more related to European situations, the principles stay the same and can be well applied in the R S A.

This book will most definitely be very useful to students at an early stage of their studies. With the emphasis changing in this country towards a herd approach, it will also assist in developing a practical approach to this problem.

The inclusion of information on the important variations imposed upon management by a tropical environment, widens the appeal of this book.

Although the chapter on preventive medicine is lacking information from a veterinary point of view regarding the diseases, it stresses the importance of husbandry very well and outlines its role as being of vital importance.

A practical and therefore good husbandryman must possess a synoptic understanding of his subject. This book will assist in realising those objectives.

L.J. Kritzinger

ANTIMICROBIAL AND PESTICIDE RESIDUES IN MILK

The Department of Health and Welfare, in close collaboration with the Department of Agricuture and several other organisations, has recently launched an extensive campaign to emphasize the hazards, dangers and undesirability of the presence of residues of antimicrobial and/or pesticidal substances in milk and dairy products.

The campaign will extend over several months. It is aimed at all who are directly or indirectly concerned. Apart from the producer of milk to be used as such or for the production of dairy products, the consumer is concerned as well as the manufacturers of dairy products. Dairy products are particularly involved because the manufacturing process often involves concentration of the components of milk.

The first stage of the campaign consists of some 12 short talks which have been serially broadcast by the SABC in the 'Calling all Farmers' programme. Editorial material has also been prepared for issue to agricultural journals and letters have been addressed to every known commercial milk producer.

The talks and editorial material are aimed at enlightening the public in general and the dairy farmer in particular about the need for the utmost care in the handling, use and storage of insecticides so as to prevent direct exposure of farm personnel and also indirect exposure of consumers of agricultural products as a whole and milk and dairy products in particular. As far as antimicrobials such as antibiotics and disinfectants are concerned, the radio talks and editorials emphasize the possible harmful effects to the consumer and the retarding effect of such inhibitory substances on the production of cultured dairy products such as cheese and yoghurt.

This information is provided against the background of the relevant legislation. Special mention is made of the registration, under Act 36 of 1947, of stock remedies and pesticides which are approved for agricultural use. These products are, of course, marketed with full and detailed instructions concerning use, application and precautions. The fact that some consignments of milk are still found to contain insecticides, such as dieldrin, indicates that there is a significant degree of wrongful application and even the illegal introduction of unregistered products. In the case of drugs and medicines prescribed, dispensed or administered by veterinarians in terms of Act 101 of 1965, the responsibility of advising on use and honouring withdrawal times lies primarily with the veterinarian.

Reference is also made to the maximum permissible levels of certain insecticides and the total ban on the presence of antimicrobial substances in milk and dairy products. These provisions are to be found in the Hazardous Substances Act No. 15 of 1973 and the Food, Cosmetics and Disinfectants Act No. 54 of 1972. This legislation indicates the degree of control which health authorities can exercise and the protection which the consumer is entitled to expect.

This campaign is not intended to scare the public but stems from the very real concern about the degree and extent of contamination of milk and milk products with what may be called agricultural chemicals. Clearly, something must be done and it is evident that once all the persons and bodies concerned have been informed of the situation, stronger action from health authorities may be expected when milk and dairy products are found to be contaminated.

The veterinarian, certainly as far as the use of insecticides for control of external parasites and the treatment of animal disease by means of preventive or therapeutic application of antimicrobial substances is concerned, has an important role to play. He is in a key position to advise and enlighten owners of dairy cows. He has, in fact, very definite responsibilities in this regard, negligence on his part leading to the imposition of fines or other penalties on the milk producer, would inevitably be viewed in a serious light and could even result in court action. Apart from these considerations, veterinarians are of necessity well aware of the dangers, hazards and implications of improper use of insecticides and antimicrobial therapeutic substances. As members of an informed profession, they therefore have a definite moral obligation to the community and the authorities in this regard, an obligation which no veterinarian can afford to ignore.

REVIEW OORSIG

SELENIUM IN LIVESTOCK PRODUCTION: A REVIEW

L.G. EKERMANS and JANE V. SCHNEIDER*

ABSTRACT: Ekermans L.G.; Schneider J.V. **Selenium in livestock production: A Review.** *Journal of the South African Veterinary Association* (1982) **53** No. 4, 223-228 (En) Department of Livestock Science, Faculty of Agriculture, University of Pretoria, 0002 Pretoria, Republic of South Africa.

A review of the literature concerning the role of selenium in livestock is presented. Selenium was originally known for its toxicity but in 1957 it was discovered that supplementation of this element prevented liver necrosis in rats. The occurence of selenium in soils and uptake by plants is discussed, as well as the biochemistry, metabolism, interactions with other elements and nutrients, known functions, requirements of livestock and its toxicity.

INTRODUCTION

Originally Se was primarily recognised for its toxicity. Certain plants accumulated Se in quantities that were toxic to animals. Marco Polo in his journals referred to a poisonous plant that when eaten by horses had the effect of causing hoofs to drop off[61]. Rosenfeld & Beath [46] provide quotes from a report by Madison in 1857 in which he describes a condition in the cavalry horses in South Dakota that resulted in sloughing of hoofs and loss of mane and tail hair. This condition was termed alkali disease or blindstaggers and was responsible for large losses of livestock in a number of the western states of the U.S.A.[61]. Only in 1934 was it discovered that Se was the causative agent[46].

Se was considered a toxic element until 1957 when Schwarts and Foltz[51] recognised Se to be the effective component of "factor 3" which prevented liver necrosis in rats. Schwarts and Foltz[51] further demonstrated that Se prevented exudative diathesis in chicks. Since then Se was found to play a role in preventing nutritional muscular dystrophy (NMD) in lambs and calves[34], hepatosis dietetica in pigs[12], pancreatic fibrosis in severe Se deficient chicks[35 58], Se responsive unthriftiness in sheep and cattle[3], reduced egg production and hatchability in poultry[8], and poor growth, increased mortality and gizzard myopathy in young turkey poults[52].

SELENIUM IN SOILS AND UPTAKE BY PLANTS

Se is a semi-metal similar to sulphur in its chemical properties. It exists in soils as basic ferric selenite, elemental Se and organic compounds derived from plant tissue. Se toxicity seems to be a greater problem in more alkaline soils where its uptake by plants is greater[61]. The relationship of Se in rocks, soils and plants can be divided in four main groups viz.

1. Where rocks with a high Se content (eg. sedimentary rocks, especially shales) decompose to form well drained soils in sub-humid areas (rainfall below 480 mm per annum), vegetation is produced that has potentially toxic amounts of Se. These are generally alkaline soils.
2. Where rocks with a high Se content weather to form soils, in humid areas, that are slightly acid, plants will not contain toxic amounts of Se but contain sufficient to prevent Se deficiency in animals.
3. Where rocks with high Se weather to form poorly drained soils or where Se from higher lying areas is deposited in poorly drained areas and soils are alkaline, vegetation with toxic levels are likely to be found. The more acid the soils in these areas, the lower the chances are for toxic amounts of Se.
4. Where rocks with low Se contents (eg. igneous rocks) decompose to form soils under humid or dry conditions, the plants are likely to contain insufficient amounts of Se to prevent deficiencies in animals. The more humid the area and the more acid the soils, the greater are the chances for low Se levels.

Seasonal conditions can influence the amount of Se in plants, as can climate and management practices[27]. Higher rainfall causes lower Se levels[48]. When climate favours a lush forage growth, especially in areas with low soil Se levels, deficiencies can occur. This could be due to the stem to leaf ratio which is greater under these conditions and most of the Se is found in the leaves[27]. Sulpher rich fertilizers are known to depress uptake of Se in plants because a downward movement of Se in the soils is caused[27] or this may be due to increased soil acidity[14]. Heavy grazing of pastures may also deplete Se in the plant[27]. The Se content of forages varies, legumes like clovers being lower than grasses. Arthur[4] found soybean meal to be low in Se whereas rapeseed and linseed meal were far higher and grains varied widely. The Se content of a few feed components are shown in Table 1.

BIOCHEMISTRY AND INTERACTIONS OF SELENIUM

In 1972 workers at Wisconsin established that Se is an integral part of the enzyme glutathione peroxidase (GSH-px) which functions primarily in protecting cell membranes agains peroxidative damage[47]. The action of Vitamin E is within the membrane itself where it prevents the chain reactive auto-oxidation of the membrane lipids and acts as a secondary defence mechanism against peroxidative destruction[35].

GSH-px destroys the peroxides which have already been formed by converting them to less harmful alcohols[64]. Erythrocyte or whole blood GSH-px activity has been shown to be a reliable indicator of Se status in animals[25 35 42]. Peter et al.[42] however warn that GSH-px activity of adult animals does not immediately attain a new equilibrium value when Se intake alters and this

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Table 1: Se CONTENT OF A FEW FEED COMPONENTS IN PPM

	Cantor, et al.[9]	Scott and Thompson[54].	Arthur[4].
Tuna meal	5	6,2-5,1	
Wheat	2	0,05-0,8	0,02-1,52
Brewers yeast	1,5	1,10	
Brewers grain	0,7	0,19	0,81-1,07
Menhaden meal	2,6	1,7	1,65-2,18
Herring meal	3,6	1,5-2,5	2,21-3,36
Corn	1,0	0,025-0,38	0,01-1,0
Soyabean meal	0,54	0,07-0,54	0,04-0,2
Blood meal		0,07	0,51-1,16
Linseed meal		1,0	0,65-1,51
Dehydrated alfalfa meal	0,21	0,09-0,38	0,02-0,14

must be taken into account when measuring responses to Se.

Noguchi et al.[35] found that GSH-px activity was directly related to the effectiveness of preventing exudative diathesis in chicks but was not correlated to the incidence of NMD in lambs[65].

Studies with ^{75}Se indicate the duodenum as the main absorption site and there is no absorption from the rumen or abomasum in sheep or from the stomach in pigs[63]. Se is excreted in the faeces, urine and expired air, the amount and proportions depending on the level and form of intake, the nature of the rest of the diet and the species of animals[63].

Se has a strong tendency to complex with heavy metals and Se metabolism can therefore be influenced by the dietary levels of several such elements. Jensen[26] quotes workers who found that silver interfered with the absorption of Se. It is suggested that Ag binds with GSH-px and inhibits peroxide decomposition[15]. Copper modifies Se toxicity by forming a copper-selenide complex which accumulates as a non-deleterious compound in tissues[26]. Oh et al.[37] quote workers who found that dietary sulphur increases the incidence of NMD and prevented the beneficial effect of Se. Hoffman La-Roche[20] say that sulphur probably inhibits the absorption of Se and displaces it competitively in its passage through the cell walls. Hintz & Hogue[23], too, stated that high sulphate intakes increased Se requirements of sheep. Whanger et al.[66] demonstrated that lambs from ewes on a Se deficient diet had a higher incidence of NMD when sulphate was fed and supplementing Se prevented this.

Rudert and Lewis[49] found an increase in NMD in progeny from ewes fed KCN. Cyanogenic glycosides are found in heavily fertilized pastures especially certain types, eg. Star grass (*Cynodon aethiopicus*) and a higher incidence of NMD occurred on these pastures[50]. It was found that cyanide reacted with Se in vitro to form a seleno-cyanate but whether this occurred in physiological systems is unknown. There are indications that cyanide affects normal Se and possibly Vitamin E metabolism.

Jensen[26] quoted work in which 5 ppm arsenic fed to rats counteracted Se toxicity. Interactions between Se and protein have also been found. Rosenfeld & Beath [46] said that high protein diets afford some protection against Se toxicity whereas Julien et al.[28] found that increasing protein which contained some Se decreased Se responsive diseases in dairy cattle.

The interactions of Se with other elements is thus important when deficiency symptoms occur, for the deficiency may be due to an interaction and not a Se shortage as such.

FUNCTIONS OF SELENIUM

Since Schwartz & Foltz[51] discovered Se to be an essential nutrient, many researchers supplemented Se to investigate possible responses.

Peter[54] obtained no significant effect on ewe fertility or lamb growth whereas Oh et al.[36] found improvement in 18 week body weight gain of lambs supplemented with Se. Most researchers found that by supplementing 0,1 ppm Se no deficiencies occurred but Whanger et al.[66] discovered that sheep needed 0,2 ppm when fed legumes. Oh et al.[36] saw no difference in supplementing 0,5 or 0,1 ppm Se.

Ullrey et al.[62] found 30-65 ppm Se in salt to be safe and effective in preventing Se deficiencies. Sharman[56] noted that lambs reaching maximum growth rates (300 g average daily gain) and being fed intensively showed NMD and ill thrift.

An injection of Vitamin E and Se prevented this. Byers & Moxon[7] showed that cattle respond positively to Se especially when growth rate is high and diets are deficient or marginal in protein. Their results indicate that the need for Se is highest when protein requirements are greatest and response to supplemental Se was highest when protein levels were below optimum requirements.

Julien et al.[28] increased Se from 0,02 ppm to 0,06 ppm in a diet by simply increasing the protein which contained some Se; the incidence of retained placentae in dairy cows decreased accordingly from 50 to 20 %. They further noticed that by supplementing Se three weeks prepartum to increase intake from 0,23 mg to 0,92 mg daily, retained placentae decreased from 38 to 0 %. By a single injection of 50 mg sodium selenite they reduced the incidence of retained placentae from 51,2 to 8,8 %. An injection of 680 IU Vitamin E plus 50 mg Se had the same effect.

Gwazdauskas et al.[18] found that Vitamin E-Se injections did not influence reproduction or production in dairy cows. Incidence of retained placentae was 11,4 % and showed no response to Se treatment.

Segerson et al.[55] found that by supplementing superovulated cattle with 100 mg Se over 3 injections, 100 % fertilised ova were obtained as compared to 41 % or less without Se supplementation. These researchers found that Se was an interacting nutrient in promoting sperm penetration of the zona pellucida of the

transferred ova. Julien et al.[28] saw Se as an effector of increased sperm motility.

Michael et al.[33] noticed Se deficiencies in commercial swine herds and Ullrey[61] detected the same type of deficiency lesions in pigs shortly after weaning. These lesions were described by Eggert et al.[12] in pigs on a low Vitamin E-Se diet and unsupplemented pigs died within 58 days showing, at post mortem, lesions of hepatosis dietetica. No deaths occurred in supplemented pigs. Mahan & Moxon[32] also noticed Se deficiencies in pigs soon after weaning when a corn-soybean diet was fed; 0,4 ppm supplemental Se prevented this whereas the officially allowed supplement of 0,1 ppm did not.

Piatkowski et al.[45] and Wilkinson et al.[68] found Se to have no beneficial effect on growth or reproduction in pigs if tissue Se levels were adequate.

Bengtsson et al.[55] and Hakkarainen[19] found that both Vitamin E and Se were needed to prevent the Vitamin E-Se deficiency syndrome in pigs. Given separately they were inadequate in preventing this syndrome. Five mg DL-α-tocopherol and 135 μg/kg Se was found to be sufficient to protect pigs.

In chicks Se supplementation prevented exudative diathesis[35] and pancreatic fibrosis[10] and improved egg production and hatchability on low Se diets[8].

Interesting work is quoted by Peplowski et al.[41] wherein it was demonstated that Se enhanced the primary immune response in mice. Peplowski et al.[41] found that by adding Vitamin E and Se (5 ppm Se + 220 IU Vitamin E/kg) the immune response was enhanced. Vitamin E and Se fed separately had the same effect as was shown by hemagglutination assays. Table 2 summarises the results obtained by Se supplementation.

SELENIUM REQUIREMENTS

The minimum requirement of animals varies with the form of the Se injected and the nature of the diet[63]. A dietary intake of 0,1 ppm for grazing sheep and cattle is recommended as a supplement by Allaway & Hodgson[1] but Whanger et al.[66] indicate that sheep fed legumes required 0,2 ppm. Oldfield et al.[38] found 0,06 ppm supplemental Se sufficient to prevent NMD in lambs but Oh et al.[36] found this amount insufficient and that 0,1 ppm was the minimum required. These differences show that other factors influence Se requirements, e.g. high

Table 2: RESULTS OBTAINED BY Se SUPPLEMENTATION

Animals used	Se supplement	Response	Reference
Dairy cows	50 mg Se + Vitamin E injection, initially	100% fertile ova recovered as against 40 % in unsupplemented group	Segerson et al.[55]
	25 mg Se + Vitamin E at 30 & 40 days after initial injection		
	50 mg Se + Vitamine E 21 days prepartum	Retained placenta reduced from 38 % to 0	Julien et al.[29]
	Vitamin E, Se injection 28 days prepartum 15 mg Se	Retained placenta reduced	Trinider et al.[59 60]
	Se 15 mg + Vitamin E	No effect on retained placenta	Horvath 1975 as quoted by Julien et al.[29]
	21,0 mg sodium selenite before calving	No influence on calving retained placenta	Gwazdauskas et al.[18]
Pigs	0,05 ppm as sodium selenite	Decrease in mortality, no effect on growth	Ewan et al.[13]
	0,1 ppm Se 0,5 ppm Se	Decrease in mortality, no effect on growth	Groce et al.[17]
	0,05 ppm Se as selenious acid	Increase in growth, feed intake, feed efficiency, unsupplemented pigs died between 3 & 21 days post weaning	Glienke & Ewan[18]
	0,1 ppm Se or 1,0 ppm Se	No effect on growth, reproduction or feed efficiency	Wilkinson et al.[67 68] Young et al.[69]
Sheep	0,94 mg/ewe/day as 132 ppm Se in salt 25 mg Se drench	Greater gains in lambs and decreased mortality, no effect on fertility, growth but lambs of supplemented ewes were heavier from 49 days of age onwards	Paulsen et al.[40] Peter[43]
Hens	0,015 ppm or 0,03 ppm	Increase in egg production and hatchability	Cantor & Scott[8]
	0,1	After 5 weeks egg production increased steadily, hatchability 90 %, for unsupplemented group 42 %, after 20 weeks hatchability 90 %, for unsupplemented group 10 %	

sulphate intakes reduce Se availability to animals so that Se requirements are greater. The availability of Se in the Se supplement varies and different types of supplement differ in effectiveness in preventing a deficiency symptom[9].

Scot et al.[52] found 0,06 ppm Se as sodium selenite adequate to prevent exudative diathesis and pancreatic fibrosis in chicks. They determined that turkeys required 0,17 ppm to prevent gizzard and hart myopathies but 0,28 ppm was required when Vitamin E was low in the diet.

Cantor et al.[9] established that the Se requirements of chicks were higher when animal products were included as protein sources (availability of Se lower than 25 %) than when plant products were used.

Abnormally high dietary levels of silver, copper and zinc increased Se requirements[63]. Underwood quotes Jensen[26] who increased Cu and Zn in chick diets and observed high mortality, exudative diathesis and NMD when the diet had 0,2 ppm Se. Adding 0,5 ppm supplemental Se prevented the deficiency signs and significantly reduced mortality. Similar results were noticed when silver nitrate was added to the diet.

In pigs 0,1 ppm supplemental Se is required to prevent deficiency symptoms [16 31]. Examples of Se requirements of livestock are shown in Table 3.

Table 3: SUPPLEMENTAL Se REQUIREMENTS

Turkey poults	0,17 ppm Se 0,28 ppm Se	Scott et al.[53] Low Vit. E Diet
Laying hens	0,1 ppm Se	Combs & Scott[11]
Pigs	0,1 ppm Se	Jenkins & Winter[25]
Sheep	0,11 ppm	Oh et al.[37]
	0,2 ppm	on legume forages, Whanger et al.[66]
	0,1 ppm	on non legume forages – Whanger et al.[66]
All animals	0,5 – 1,0 ppm 0,1 – 0,15 ppm	on low Vit. E diet – Hoffman & La Roche[20] on sufficient Vit. E – Hoffman & La Roche[20]

Se requirements vary but this is due to the complex interactions with other metals and due to the difference in avilability of Se in animal diets. In some cases the same level of Se supplementation produced opposite results, some researchers obtained positive response and others none. It is therefore important when analysing results to look at the composition of rations being fed.

AVAILABILITY OF Se AND SUPPLEMENTATION METHODS

Schwartz & Foltz [51] divided Se compounds into three main categories according to their ability to protect rats from liver necrosis.

The first group includes elemental Se and compounds practically inactive due to poor absorption with no protection against liver necrosis. Cantor et al.[9] demonstrated that elemental Se was also unavilable to prevent exudative diathesis in chicks.

The second group consists mainly of inorganic salts such as selenites, selenates and Se analogs such as selenomethionine and selenocystine. These have more or less equal values in preventing liver necrosis in rats. Cantor et al.[10] found sodium selenate and selenocystine to have high values in preventing exudative diathesis whereas low values were given to seleneothionine, selenomethionine, selenopurine and sodium selenite. Sodium selenite and selenomethionine resulted in high GSH-px activities and gave protection against exudative diathesis.

The third group consist of Se compounds in organic forms. These are more active per unit of Se than those in the second group.

The Se levels in plants vary widely and depend mainly on the Se status of the soils on which they are grown[63]. Pastures and forages free from Se responsive diseases usually contain 0,1 ppm Se or more while in areas where deficiency symptoms occur Se levels are below 0,05 or 0,02 ppm[63].

Se can be given as injections, oral dosing of Se salts, salt licks containing Se, use of feeds rich in Se, Se-containing heavy pellets for ruminants or treatment of soil with Se compounds[63]. Injections and dosing of animals seems to be the best method, having the advantage of knowing exactly how much each animal receives but it is expensive and individual handling of animals is required which makes it time consuming[3]. Kuchel & Buckley[30] investigated using Se-containing pellets (1 g elemental Se + 9 g iron); the pellets were retained in the reticulo-rumen and Se levels in blood and tissue were significantly raised for 12 months. No evidence of toxicity or excessive Se accumulation in tissues was noticed, therefore making pellets safe and effective for grazing ruminants. Jenkins and Hidiroglou[24] used slow release Vitamin E-Se pellets in ewes and lambs. Sodium selenite pellets were implanted in the loose connective tissue behind the shoulder. Implantation increased Se levels in blood and the incidense of NMD was reduced in lambs. Hidiroglou[22] found that when Se pellets were implanted into neonatal beef calves born to cows on a Se deficient diet, NMD was effectively prevented.

Rotruck et al.[47] used 30-65 ppm sodium selenite in salt licks and proved this amount to be safe and effective.

TOXICITY OF Se

The most well documented Se toxin disease is blindstaggers or alkali disease. This disease generally occurs in animals eating Se accumulator plants which have the

ability of taking up Se in the soil unavailable to other plants and converting it to a more available form for animals and other plants when these accumulator plants die. Selenosis can be mild, chronic or acute[63].

Chronic Se poisoning is recognised by dullness, lack of vitality, emaciation, roughness of coat, loss of hair from the mane and tail of horses and body of pigs, soreness and sloughing of hooves, stiffness and lameness due to erosion of the joints of long bones, atrophy of the heart, cirrhosis of the liver and anemia[63].

In acute poisoning blindness, abdominal pain, salivation, grating of teeth, some degree of paralysis, respiratory failure and starvation due to loss of appetite are symptoms found in cattle, horses and pigs. In sheep loss of weight and reduced weight gains are evident. Low egg production and hatchability are observed in hens and reduced feed intake and growth in chicks. High Se intakes affects reproductive performance in farm animals. Underwood[63] quotes workers who state that a reduction in reproduction is the main effect of toxicity and that this effect can be severe even though no other effect or lesions appear. Abnormal development of embryos in rats, foals, sheep and cattle as well as chicks have been noticed.

Underwood[63] advises the feeding of high protein or sulphate diets to alleviate Se toxicity but he mentions that this is difficult under range conditions. Arsenic compounds can effectively be used (5 ppm in drinking water, 20-40 ppm arsenic or 50-100 ppm arsanilic acid in feed) to treat chronic poisoning[48]. Toxic levels of Se are shown in Table 4.

Table 4: TOXIC LEVELS OF Se

Supplement to a basal diet: 5 ppm Se 7 ppm Se 9 ppm Se 10 ppm Se	 Hatchability decreased Hatchability decreased + low egg weight Low egg prod, hatchability decreased + low egg weight abnormal embrios	Ort & Latshaw[39]
5 ppm in edible herbage on a dry basis	toxic to grazing stock	Underwood[63]
2 mg of Se per kg body weight supplied by intake of plants	Blind staggers in cattle and sheep	Rosenfeld & Beath[46]
Consumption of cereals, grasses and hays with moderate levels of Se (10-30 ppm)	Alkali disease in cattle and sheep	Rosenfeld & Beath[46]

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A REPORT ON THE SAFETY OF AN INTRAVENOUS COMPOUND CALCIUM SOLUTION AND THE LEVELS OF PLASMA TOTAL AND IONIZED CALCIUM FOLLOWING INJECTION

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ABSTRACT: Belonje P.C.; Jaros G.G.; Van Den Berg A. **A report on the safety of an intravenous compound calcium solution and the levels of plasma total and ionized calcium following injection.** *Journal of the South African Veterinary Association* (1982) **53** No. 4, 229-231 (En) Department of Human and Animal Physiology, University of Stellenbosch, 7600 Stellenbosch, Republic of South Africa.

An intravenous compound 5 % calcium solution was subjected to three trials in S.A. Mutton Merino X Merino ewes to establish its safety and also to resultant levels of blood total and particularly ionized calcium. In the first trial, 10 animals each received 20 ml of the compound but the administration time progressively decreased from 90 seconds in the first to only 15 seconds in the last ewe. Plasma total calcium levels more than doubled in all the animals and, except for transient disorientation in the last ewe, nothing untoward was noted. In the second trial 4 ewes again received 20 ml of the compound while in the third trial 5 ewes received the compound at the rate of 0,5 ml/kg. In both trials the administration time was about 30 seconds. Plasma total and ionized calcium levels more than doubled initially and, while there was a steady and parallel decline in both, they were still more than 60 % higher than pre-injection levels after as long as 2 hours.

Key words: Calcium solution, safety, sheep, plasma levels

INTRODUCTION

It is well known that excess blood calcium levels may lead to respiratory and cardiac failure[1]. In practice it is often difficult to determine whether "downer" sheep are in fact hypocalcaemic and it is therefore important that intravenous calcium solutions should be safe to use even in animals who are normocalcaemic. In addition veterinarians are often confronted with a large number of "downer" sheep and time cannot be wasted with slow injections. An intravenous calcium solution must therefore also be safe even if injected fairly rapidly.

A recently formulated compound intravenous high calcium solution (Surcalce, Sandoz) was made available for testing and three short-term experiments were conducted. The solution consisted of 45 % calcium gluconate monohydrate, 2 % calcium gluconolactobionate, 3,7 % calcium acetate monohydrate and 3 % magnesium hypophosphite-6-hydrate. The first experiment was conducted to establish whether rapid intravenous injections of Surcalce at ambient temperatures were fatal. The second and third experiments were designed to follow the total and ionized calcium levels after intravenous injection of either 20 ml Surcalce in a random weight class or at 0,5 ml/kg body mass. The ionized calcium determinations were most important as it had to be established whether this solution in fact raised this the physiologically important fraction of total calcium.

MATERIALS AND METHODS

Experimental procedures

Experiment 1

Ten healthy 2-tooth S.A. Mutton Merino X Merino ewes were selected at random. They were all non-pregnant, non-lactating and their average body mass was 48,1 kg. During the 2 months previous to this trial their weekly plasma total calcium and magnesium levels had been monitored for other purposes.

On the day of this trial each of the 10 animals received 20 ml Surcalce intravenously at a rate far higher than the recommended duration of injection of 5 minutes. The first animal received the dose over 90 seconds and this time was progressively decreased until Sheep 10 received the full dose in only 15 seconds. Also the infusion was given at ambient temperature and not at body temperature as recommended. This procedure would possibly mimic emergency conditions in the field. The sheep were bled 5 and 10 minutes after the injection for plasma total calcium and magnesium determinations.

Experiment 2

Four healthy S.A. Mutton Merino X Merino ewes were used. Their ages ranged from 6 months to 6 years. Each animal was fitted with a 2-way intravenous cannula which allowed for flushing after any infusion, and clearing before taking a blood specimen. Before the experiment commenced each sheep received heparin intravenously (500 IU/kg body mass). Five minutes before Surcalce was administered, a base-line blood speciment was taken. At time zero each sheep received 20 ml Surcalce intravenously. Blood speciments were taken at 1; 10; 20; 30; 40 and 50 minutes after this injection. An aliquot of each blood speciment was analyzed immediately for ionized calcium while the rest was centrifuged for plasma which was frozen and later analyzed for total calcium and magnesium.

Experiment 3

Five healthy adult S.A. Mutton Merino X Merino ewes were used. The animals were fitted with intravenous cannulae as in Experiment 2. Food and water were withheld for 24 hours and then the animals were weighed immediately before the experiment began to establish fasting body mass. The dose of Surcalce was then calculated at 0,5 ml/kg body mass.

The animals were heparinized as before. Thereafter blood specimens were taken 25; 15 and 5 minutes before Surcalce administration and 1; 5; 10; 15; 20; 30; 60; 90; and 120 minutes thereafter. An aliquote of each blood specimen was analyzed immediately for ionized calcium

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while the rest was centrifuged for plasma which was frozen and later analyzed for total calcium and magnesium.

Analytical methods

1. Ionized calcium levels were determined by means of a Neutral Carrier Disc Electrode[2].
2. Total calcium and magnesium levels in Experiments 1 and 2 were determined by means of an atomic absorption spectrophotometer (Varian Techtron Model 1200) using a nitrous oxide-acetylene flame and potassium as an ionization suppressor[4].
3. Total calcium levels in Experiment 3 were determined by means of an atomic absorption spectrophotometer (Varian Techtron Model 1275) using an air-acetylene flame and lanthanum chloride as a releasing agent[3].

RESULTS AND DISCUSSION

Experiment 1

Weekly blood specimens analysed during the two-months prior to this experiment revealed that plasma total calcium levels in the animals varied between 2,42 and 2,57 mmol/ℓ while plasma magnesium levels varied between 0,82 and 1,03 mmol/ℓ. Both these ranges are normal.

The 5 and 10 minutes levels after the intravenous injection of Surcalce are given in Table 1.

Table 1: PLASMA TOTAL CALCIUM AND MAGNESIUM LEVELS IN SHEEP 5 AND 10 MINUTES AFTER THE INTRAVENOUS ADMINISTRATION OF 20 ml OF SURCALCE AT INCREASING RATES OF ADMINISTRATION

Sheep No	Rate of administration in seconds	Plasma calcium mmol/ℓ		Plasma magnesium mmol/ℓ	
		After 5 min	After 10 min	After 5 min	After 10 min
1	90	7,06	6,80	1,70	1,33
2	85	6,47	5,70	1,64	1,26
3	85	6,49	5,50	1,37	1,22
4	60	6,88	5,78	1,06	0,95
5	60	7,06	6,14	1,37	1,14
6	35	6,68	5,87	1,32	1,16
7	30	6,87	6,26	1,59	1,21
8	30	7,10	6,32	1,39	1,18
9	20	5,98	6,43	1,11	1,12
10	15	5,63	6,05	—	1,28

Note that in all cases the total calcium more than doubled while there was about a 25 % increase in plasma magnesium. In all the animals lip-licking, teeth-grinding, defaecation and urination were noted soon after the injection. It was only in Sheep 10 which received the Surcalce within 15 seconds (the fastest one could express the solution through an 18 gauge needle), that the animal appeared slightly disorientated for a few minutes. Other than that all the animals appeared normal and they all commenced feeding when they were placed on pasture immediately after the last specimen was taken.

It is obvious that Surcalce was a safe form of intravenous calcium in this group of sheep and should it be used in animals that are not hypocalcaemic it should not cause adverse effects.

Experiment 2

The results of this experiment are given in Table 2. Once again there was the expected rise in total calcium but it is pleasing to see a concommitant rise in ionized calcium.

Table 2: THE MEAN (±SE) PLASMA TOTAL CALCIUM, IONIZED CALCIUM AND TOTAL PLASMA MAGNESIUM LEVELS IN 4 SHEEP BEFORE AND AFTER AN INTRAVENOUS INJECTION OF 20 ml OF SURCALCE

Time relative to injection (min)	Plasma total calcium mmol/ℓ	Plasma ionized calcium mmol/ℓ	Plasma total magnesium mmol/ℓ
−5	2,56±0,13	1,03±0,03	0,87±0,05
1	6,77±0,63	2,72±0,19	1,23±0,06
10	5,03±0,29	2,24±0,15	1,06±0,03
20	4,71±0,20	1,98±0,17	1,06±0,03
30	4,31±0,14	1,85±0,10	1,04±0,04
40	4,08±0,10	1,71±0,10	1,01±0,04
50	4,06±0,11	1,67±0,07	1,01±0,05

Both these levels decreased in an almost identical fashion with time (correlation coefficient r = 0,98). Magnesium levels followed a similar course, but were of a much lower magnitude.

From these data one can state that Surcalce not only raises the total calcium levels, but also the physiologically important ionized calcium levels.

Experiment 3

The results of this experiment are given in Table 3.

Table 3: THE MEAN (±SE) PLASMA TOTAL CALCIUM AND IONIZED CALCIUM LEVELS IN 5 SHEEP BEFORE AND AFTER AN INTRAVENOUS INJECTION OF SURCALCE AT THE RATE OF 0,5 ml/kg BODY MASS

Time relative to injection (min)	Plasma total calcium mmol/ℓ	Plasma ionized calcium mmol/ℓ
−25	2,30 ± 0,14	1,06 ± 0,07
−15	2,32 ± 0,14	1,05 ± 0,07
−5	2,32 ± 0,13	1,06 ± 0,07
1	7,98 ± 1,52	3,13 ± 0,37
5	6,70 ± 0,89	2,93 ± 0,30
10	5,50 ± 0,40	2,60 ± 0,17
15	5,12 ± 0,41	2,47 ± 0,12
20	5,01 ± 0,25	2,34 ± 0,13
30	4,58 ± 0,28	2,22 ± 0,13
60	4,30 ± 0,20	2,08 ± 0,10
90	3,89 ± 0,23	1,95 ± 0,13
120	3,87 ± 0,24	1,77 ± 0,09

These calcium results are similar to those in Experiment 2, and again total and ionized calcium levels were closely correlated (r = 0,978). It is also pleasing to note that the rate of decline in the two calcium levels was similar to that in Experiment 1 and, in addition, that these levels remained elevated by more than 60 % above the base levels even 2 hours after the injection.

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BOOK REVIEW — BOEKRESENSIE

GENETICS FOR DOG BREEDERS

ROY ROBINSON

Pergamon Press, Oxford, New York, Toronto, Sydney, Paris, Frankfurt 1982 pp 264, Figs 32, Tabs 19, ISBN 0-08-025917-0, £9,95

The writing of this book stems from the belief that the continued advancement of dog breeding will depend upon appreciation of modern trends in animal breeding. It should be acknowledged that the world of animal breeding is moving steadily away from "rule-of-thumb" methods in favour of more balanced programmes of selective breeding. The thoughtful breeder would be wise to ponder on the fact that once his or her dogs are provided with a good home, are properly nourished and under expert veterinary supervision, the only avenue left for substantial improvement resides in the science and art of breeding.

The early chapters present an outline of the basic principles of heredity. About 35 pages are set aside for "elements of heredity" which are followed by the more practical aspects. The many facets of selection, inbreeding, line breeding and creation of strains and blood lines are discussed.

Later chapters deal in some detail with an account of colour and coat inheritance, with particular attention to the fundamental types which often cut right across many breeds. The topic is of considerable interest in itself, in addition to the contribution of an understanding of breed evolution.

No book on dogs is complete without a discussion of the various anomalies which can afflict these animals. The publicity given to a few of the more common and serious should not obscure the fact that the great majority are rarely encountered. The conscientious dog breeder should be aware of those defects and ailments which are primarily genetic in nature. He or she should be aware of the clinical signs and have an idea of the steps to be taken to combat them. This chapter will certainly be of service to veterinary surgeons in the competent pursuit of their profession. The latest information on immunogenetics and biochemical genetics is also presented, but the behavioural aspects and the genetics thereof are only mentioned in passing. This is the only but a real shortcoming of the book which otherwise can be recommended to veterinarians, veterinary students and all serious dog breeders.

D.R. Osterhoff

ON THE INACTIVATION OF *BRUCELLA ABORTUS* IN NATURALLY CONTAMINATED MILK BY COMMERCIAL PASTEURISATION PROCEDURES

L.W. VAN DEN HEEVER*, K.W. KATZ+ & LESLEY A. TE BRUGGE†

ABSTRACT: Van den Heever L.W.; Katz K.W.; Te Brugge, Lesley A. **On the inactivation of *Brucella abortus* in naturally contaminated milk by commercial pasteurisation procedures.** *Journal of the South African Veterinary Association* (1982) 53 No. 4, 233-234 (En). Faculty of Veterinary Science, University of Pretoria, Box 12580, 0110 Onderstepoort, Republic of South Africa.

Following concern about the recent publication indicating the possible ability of *Brucella abortus* to survive commercial pasteurisation of naturally contaminated milk, such milk was subjected to biological (BT), serological and bacteriological tests. The raw milk was Brucella Milk Ring Test positive and biotype I was isolated from 4/5 BT guinea pigs, the one tested being seropositive to the Rose Bengal Test, and with serum agglutination and complement fixation titres of 160 and 36 international units respectively. After batch (63 °C/30 min) and high temperature, short time (72 °C/15 sec) pasteurisation, all 10 BT guinea pigs were bacteriologically and serologically negative, indicating that officially approved methods of commercial pasteurisation rendered naturally *Brucella*-contaminated raw milk safe for consumption.

Key words: Brucella, milk, inactivation, pasteurisation

INTRODUCTION

The title of this report does not purport to reveal hitherto unknown facts but to confirm what has been reported by various authors and has been generally assumed to be the case by all those concerned with the control of zoonoses [3 7 8].

Bovine brucellosis still awaits total eradication in South Africa and the fact that some 53 % of representative herd milk samples react positively to the Brucella Milk Ring Test (BMRT) while some 17 % of herd milks supplied to the City of Johannesburg are found to contain viable *B. abortus* according to the guinea-pig biological test, has resulted in heavy reliance upon commercial pasteurisation to safeguard the consumer against infection. Appearance in September 1981 of an article by Swann, Garby & Schnurrenberger[9] therefore caused some concern. These authors investigated the safety aspects of serological identification of field strains of *B. abortus*. In examining forty field strains obtained from the milk and vaginal secretions of naturally infected cattle they found i.a. that 95% survived heating at 65 °C for 120 minutes while 55% survived heating at 75 ° for 120 minutes. These finding brought these workers to suggest that these organisms should then be capable of surviving the low temperature (batch) process (65,5 °C for 30 min) and probably also the high temperature short time (HTST) process which heats milk for 15 seconds at 71,6 °. They concluded that many field strains of *B. abortus* were more thermoduric than previously recognised and state that studies are in progress to determine a survival curve for the heat denaturation of *B. abortus* as affected by subculturing and time since isolation.

Furthermore, temperatures at which milk is required to be commercially pasteurised in South Africa are 63,0 °C for 30 min. (batch) and 72,0 °C for 15 seconds (HTST)[10] – the batch temperature being somewhat lower than that referred to by Swann et al.[9].

In view of this uncertainty it was decided to investigate the efficiency of the two commonly employed commercial pasteurisation methods, using naturally contaminated fresh milk

MATERIAL & METHODS

One hundred litres of chilled milk (4 °C) was obtained a few hours after the milking of a herd well known to be infected with *B. abortus,* the milk of the herd having been regularly found to be both BMRT and biologically positive for *B. abortus* over some 30 months.

Fifty litres of chilled raw milk was batch pasteurised at 63 °C for 30 min and immediately cooled to about 4 °C. Fifty litres of chilled raw milk was pasteurised in a HTST plate pasteuriser at 72 °C for 15 seconds. Samples of both batch and HTST pasteurised milks were drawn immediately for application of the 'Lactognost' Heyl & Co., (National Dairy Equipment Co., Johannesburg) screening and Aschaffenburg-Mullen conclusive tests for alkaline phosphatase. The raw milk was subjected to the BMRT and both raw and pasteurised milks were biologically tested[2]. Biological testing consisted of intramusclar injection into guinea pigs of 1,0 ml of mixed sediment and cream obtained from centrifugation of 10 ml of milk, the animals being sacrificed after 24 and 45 days for serological examination and attempted culture of *B. abortus* from spleen and other organs[1]. Serological examination consisted of the Rose Bengal test (RBT), the bovine brucellosis serum agglutination test (SAT) and the bovine brucellosis complement fixation test (CFT). The RBT and CFT were carried out as set out by Herr, Bishop, Bolton and Van der Merwe[4] and the SAT as described by Herr, Te Brugge and Guiney[6]. SAT and CFT results were recorded in international units (IU) of anti-*Brucella* antibody per ml.

Bacterial cultures of the spleens were made by plating out tissue onto 5 different media, using a bacteriological needle. The media were the same as those used by Herr & Roux[5]. All plates were incubated at 37 °C in 10 % CO_2 (Forma Scientific CO_2 incubator) and read at 48 and 96 hours.

The isolates were typed by using agglutination reactions to monospecific antisera and by using dye and antibiotic sensitivity tests as set out in the World Health Organisation standards[4].

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1. Raw milk: the BMRT was clearly positive and *B. abortus* biotype I was isolated from the tissues of all 3 guinea pigs subjected to biological testing and sacrificed at 24 days. The serum from only one of these guinea pigs was tested, the results being as follows: RBT positive, SAT 160 IU/ml and CFT 36 IU/ml. Of the two guinea pigs sacrificed at 45 d, one yielded *B. abortus* biotype I; serologically both were RBT positive and SAT negative, both sera giving anticomplimentary reactions in the CFT so that no titre was recorded.
2. Pasteurised milk: None of the 10 guinea pigs in the biological testing of both batch and HTST pasteurised milks yielded any serological reaction and no *B. abortus* could be cultured from their tissues.
3. The 'Lactognost' screening test failed to disclose any alkaline phosphatase in either the batch or HTST pasteurised milks. However, the Aschaffenburg-Mullen test, executed on refrigerated samples 5 days later, disclosed that 18μg of p-nitrophenol/ml was liberated by the HTST milk.

CONCLUSIONS & DISCUSSION

These results clearly indicate that a strain of the common South African biotype I of *B. abortus,* when present in the milk of a heavily naturally infected herd, is completely inactivated by the batch and high temperature short time methods of pasteurisation as approved by regulation under the Foods, Cosmetics and Disinfectants Act No. 54/1972[10]. The biological test consisting of serological and bacteriological examination of the guinea pig, is considered to be highly sensitive and it appears unlikely that any *B. abortus* not detected by this method is likely to present an infection hazard to the human consumer of such pasteurised milk.

The presence of 18 μg of p-nitrophenol/ml of HTST milk (maximum permissible 10 μg) may represent either a degree of under-pasteurisation or regenerated alkaline phosphatase, the latter being a possibility because of a 5 day delay in executing the Ascraffenburg-Mullen tests. Assuming that the HTST was, in fact, slightly underpasteurised, the proof of complete inactivation of the organism shows that although not to be condoned, even such slightly underpasteurised milk can be considered safe as far as *B. abortus* is concerned.

No attempt has been made to offer any explanation for the discrepancy between these findings and those of Swann, Garby & Schnurrenberger[9]. The fact that these authors worked with American strains instead of a South African one is not considered significant. The relatively high concentration of organisms in bacterial culture suspensions in comparison with the smaller numbers likely to occur even in heavily contaminated milk may be a factor.

According to Herr (Dr. S., Veterinary Research Institute, Onderstepoort, personal communication) the great majority of South African isolations are of type I, only 10 % being of type II while none of biotype IV have been isolated. In a previous series of 90 different herd milk samples, five yielded biotype I and four biotype II. (K.W. Katz, personal communication). Further tests on the efficacy of pasteurisation procedures on milk contaminated with biotype II are recommended to amplify this report.

ACKNOWLEDGEMENT

We gratefully acknowledge the assistance of Mr. P. Pietersen in the bacterial culture and typing procedures. The Department of Food Science, Faculty of Agriculture, University of Pretoria and the Animal and Dairy Science Research Institute, Irene, kindly undertook the batch and HTST pasteurisation procedures respectively.

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SERUM CORTISOL CONCENTRATIONS IN CAPTIVE TAMED AND UNTAMED BLACK-BACKED JACKALS (*CANIS MESOMELAS*) AND TRANSLOCATED DOGS

J. VAN HEERDEN* and H.J. BERTSCHINGER**

ABSTRACT: Van Heerden J.; Bertschinger H.J. **Serum cortisol concentrations in captive tamed and untamed black-backed jackals (*Canis mesomelas*) and translocated dogs.** *Journal of the South African Veterinary Association* (1982) **53** No. 4 235-237 (En) Department of Medicine, Faculty of Veterinary Science, Univeristy of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

Serum cortisol levels were found to be higher in a group of captive untamed black-backed jackals (*Canis mesomelas*) than in a group of tame black-backed jackals. Repeated venipuncture of domestic dogs in an accustomed environment did not result in a rise in serum cortisol levels. Translocation of these dogs to a new environment resulted in a rapid but transient rise in serum cortisol levels.

Key words: Serum cortisol, jackals, dogs, stress.

INTRODUCTION

Blood cortisol levels provide a good indication of the activity of the glucocorticoid producing portion of the adrenal cortex. The activity of this portion of the adrenal cortex is subject to fairly distinct diurnal rhythms which closely parallel the metabolic, physical and emotional activity of individuals.

Ultimately the hypothalamus is the controlling centre for blood cortisol diurnal rhythms. Stressors of any kind exert a profound positive effect on the rate of glucocorticoid synthesis and thus cortisol blood levels. The effects of stressors are also mediated via the hypothalamus.

Emotional arousal in response to social interaction in animals that live or are housed in groups can be classified as a non-specific stressor. Approaching, catching and restraining an animal for the purpose of examination or collection of a blood specimen will also cause a non-specific endocrine response. This applies especially to untamed animals.

In this preliminary investigation serum cortisol levels of tamed and untamed captive black-backed jackals *Canis mesomelas* were determined. The effects of technique and environmental change on serum cortisol levels in domestic dogs were also investigated.

MATERIALS AND METHODS

Two groups of black-backed jackals kept in different localities were used. Both groups were born in the wild and subsequently raised in captivity from the age of approximately 6 weeks. Group A consisted of 5 approximately 6 month old animals housed together in a single group. They were untamed and shied away on human approach. For all practical purposes they were considered to be wild. Group B consisted of 6 approximately 20-month old tamed jackals. They were housed in pairs in adjacent runs. These animals were tamed and allowed fondling and handling by humans.

Group A animals were caught with some difficulty using a noose at the end of a long handle. Individuals had to be held down by force during collection of the blood specimens. This group was sampled on 3 different occasions as indicated in Table 1.

Group B individuals were only briefly restrained during the course of the bleeding procedure. The animals were bled in numerical order from 1 to 6.

All animals were bled from the saphenous or jugular vein between 09h00 and 10h00. Blood specimens from both groups of animals were all collected between 10 minutes to one hour of having approached them. The blood was collected in "Venoject" evacuated tubes (Comopharm) without anticoagulent. The serum was removed after clot contraction and stored at −20 °C until assayed for cortisol.

Samples for serum cortisol assays were obtained from 3 female mongrel dogs, S1, S2 and S3, respectively 18 months, 5 years and 6 years of age, routinely used for the collection of blood specimens by venipuncture. These animals were familiar with the venipuncture procedure and blood specimens were obtained in their respective kennels on three successive days as indicated in Table 3. On the third day the animals were bled 7 times at 5 minutes intervals to determined the effect of repetitive bleeding.

On another occasion two of the dogs (S1 and S2) were translocated to a strange environment to determine whether this had any effect on serum cortisol levels. The dogs were bled immediately before translocation (08h30) to obtain baseline values. They were then taken to strange kennels and bled at the intervals indicated in Table 4.

Serum cortisol levels were determined by means of a commercially available radioimmunoassay kit (^{125}I Cortisol RIA "Premix" kit, Diagnostic Products Corporation, U.S.A.). This RIA kit has been used routinely by our laboratory and has proved, with a few minor modifications to the method, to be suitable for assaying cortisol levels in animals.

RESULTS

Approaching, catching and restraint of the jackals in Group A caused considerable excitement of the individuals concerned, as well as of the rest of the animals in the group. Individuals ran and jumped around to evade the noose.

Approaching the animals in Group B caused no excitement. They were free to run around while others were bled. As in the case of the other group the handling and bleeding of individuals could be seen by the re-

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mainder of the group. Whenever Jackal No. 5 in Group B was bled, it was extremely agressive attempting to bite the people handling him. On the second occasion he was successful. The results of the cortisol assays are shown in Tables 1 and 2.

Table 1: GROUP A. SERUM CORTISOL LEVELS IN UNTAMED CAPTIVE BLACK-BACKED JACKALS

Jackal No.	Serum cortisol concentration nmol/ℓ Day 1	Day 8	Day 12	Mean
1	110	198	198	169
2	98	148	175	140
3	168	160	145	158
4	150	130	175	152
5	202	80	202	161

n = 15 Group mean 156 SD 38,5

Table 2: GROUP B. SERUM CORTISOL LEVELS IN TAMED BLACK-BACKED JACKALS

Jackal No.	Serum cortisol concentration nmol/ℓ Day 1	Day 120	Day 124	Mean
1	39	93	100	77
2	48	9	28	28
3	32	—	—	—
4	24	—	—	24
5	168	—	253	211
6	153	127	—	140

n = 12 Group mean 90 SD 74,2

The group mean serum cortisol concentration for all the untamed black-backed jackals sampled on 3 occasions (Group A) was considerably higher (156 nmol/ℓ) than the group mean for the tamed black-backed jackals (90 nmol/ℓ) in Group B. It was also considerably higher than those for domestic dogs bled in a familiar environment (Table 3). The serum cortisol levels of black-backed jackals in Group B (tame group) compare well to the normal range seen in dogs (Bertschinger 1982, Department of Genesiology, Faculty of Veterinary Science, University of Pretoria).

The results of the cortisol assays on the dogs are shown in Tables 3 and 4. Dogs bled in a familiar environment did not show a rise in serum cortisol values (Table 3). Dogs bled after having been translocated to a new environment showed a substantial but transient increase in serum cortisol values (Table 4).

DISCUSSIONS

It is interesting to note that the higher cortisol levels in 2 of the animals in Group B, Nos 5 and 6, can be explained: Number 5 was an extremely aggressive animal displaying the signs of aggression as described by Ferguson[1], whenever approached by human beings. It was in fact more difficult to restrain this animal than the animals in Group A. Jackal No. 6 was always bled last on both occasions. On the second day of sampling this was after an unsuccessful attempt to restrain jackal No. 5. It was quite obviously excited by the behaviour of its aggressive run-mate

Table 3: SERUM CORTISOL LEVELS OF DOGS BLED IN A FAMILIAR ENVIRONMENT

Day and time of collection		Dog No. 1 nmol/ℓ	Dog No. 2 nmol/ℓ	Dog No. 3 nmol/ℓ
Day 1	08h30	22	67	43
	08h50	15	43	26
Day 2	08h30	98	67	58
	08h50	60	56	39
Day 3	08h30	33	41	61
	08h35	48	42	48
	08h40	45	21	43
	08h45	28	31	36
	08h50	24	36	42
	08h55	10	45	72
	09h00	30	62	107

Table 4: SERUM CORTISOL LEVELS IN DOGS AFTER TRANSLOCATION TO A STRANGE ENVIRONMENT

Sample	Dog No. 1 nmol/ℓ	Dog No. 2 nmol/ℓ
Before translocation	25	45
After translocation:		
5 min	22	32
10 min	85	88
15 min	127	142
20 min	132	165
25 min	108	130
30 min	91	113
1 h	123	55
1 h 30 min	70	42
2 h	39	70
4 h.	32	41
6 h	53	34
8 h	37	43
24 h	51	56
30 h	22	53
48 h	25	55
54 h	55	29
120 h	30	43
126 h	53	59

Handling the domestic dogs in a familiar environment and bleeding them in an accustomed way did not produce a rise in serum cortisol values. A change in environment however did result in a tremendous upsurge in serum cortisol values. The acute rise observed indicates the necessity of collecting blood specimens for cortisol assay within a period of 10 minutes after approaching an animal and preferably in a environment familiar to the animal. It also indicates the value of serum cortisol determination in evaluating acute stress. The rapid return of serum cortisol levels to resting values as well as the diurnal fluctuations observed in dogs after translocation to a strange environment make cortisol concentrations probably less valuable as an indicator of chronic stress (Table 4). Chronic stress can perhaps be better evaluated by other parameters such as signs of phychosomatic disease and lack of resistance to pathogenic agents.

These preliminary findings indicate that

a) mere handling of an animal unaccustomed to procedures such as the collection of blood specimens, acts as a physical stressor which arouses the animal emotionally. Obtaining "normal" values from stressed animals is extremely difficult. Approaching,

catching, handling and bleeding animals should not take longer than 10 minutes;
b) a change in environment arouses an endocrine response;
c) taming wild animals considerably reduces the fear-response to humans and
d) repetitive subjection of animals to a relatively non-painful technique would reduce the stressor-effect of such a technique.

It is of extreme importance to minimize stressful situations as far as is possible whenever handling or dealing with experimental animals. Endocrine responses to stressors certainly have a profound effect on certain haematological and blood chemical parameters[3]. Continuous arousal of animals may also jeopardize their physical well being eg. a reduced resistance to internal parasites and increased susceptibility to cardiovascular diseases[2].

It is therefore highly desirable to use tamed individuals whenever research is undertaken on wild animals kept in captivity. Emotional arousal is thus kept to a minimum. It is however almost impossible to exclude stressor effects of handling and management altogether.

ACKNOWLEDGEMENT

The support of Dr Anna Verster is greatly appreciated.

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BOOK REVIEW **BOEKRESENSIE**

EQUINE CLINICAL CHEMISTRY AND PATHOPHYSIOLOGY

JAMES R. COFFMAN

Veterinary Medicine Publishing Company, Bonner Springs, Kansas 66012 1981. pp xi 275, several diagrams and tables. ISBN 0-935-78-17-7.

The title of the book is somewhat misleading in that it is neither a dissertation on equine clinical chemistry and the pathophysiology thereof, nor does it restrict itself to these subjects. In the forward the purpose in writing the book is stated as "to help equine practitioners in their efforts to enhance their diagnosis". Predictably, the emphasis is therefore heavily on a practical clinical approach, employing the laboratory in an economic way. Anybody expecting and fearing heavy academic discussions or explanations on subjects such as acid-base balance and urology, will be pleasantly surprised.

The introductory, somewhat philosopical chapter, deals with thought-provoking physiological and anatomical adaptations in the horse and the relevance thereof in equine practice.

The first section of the book, entitled "Clinical chemistry and related concepts", deals with selected topics such as serum protein electrophoresis, plasma lactate determination and adrenocortical pathophysiology.

The last section of the book deals with problem – specific data based on three important medical fields in equine practice namely abdominal pain, weight loss and diarrhoea and polyuria: polydypsia.

The book is welcomed as an important contribution towards enhancing the skill of the equine practitioner. It is therefore recommended for practitioners and students with an interest in equine medicine.

J. van Heerden

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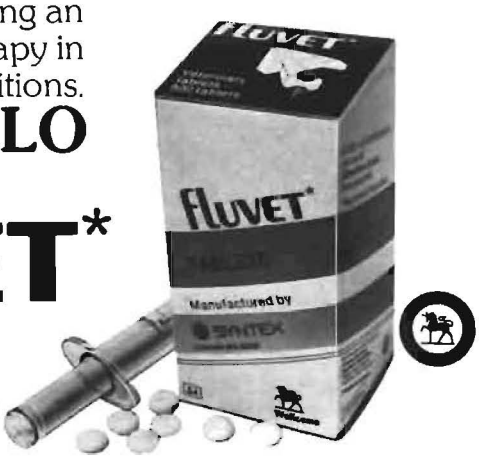

SYNTEX

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CLINICAL EXPERIENCE WITH PROSTAGLANDIN $F_{2\alpha}$ THAM AS A LUTEOLYTIC AGENT IN PREGNANT AND NON-PREGNANT BITCHES

E.E. OETTLÉ*

ABSTRACT: Oettlé E.E. **Clinical experience with prostaglandin $F_{2\alpha}$ THAM as a luteolytic agent in pregnant and non-pregnant bitches.** *Journal of the South African Veterinary Association* (1982) 53 No. 4 239-242 (En) Department of Genesiology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0100 Onderstepoort, Republic of South Africa.

Prostaglandin $F_{2\alpha}$ THAM was administered to 6 bitches at various stages after service at dosages ranging from 30-250μg/kg for 2-6 days. Four bitches treated between Days 21 – 42 after second service did not produce pups irrespective of dosage and duration of administration. A bitch treated in the first trimester and another in the third trimester did not abort but gave birth to normal litters. Two unserved bitches were also treated in the first 20 days of metoestrus with no drop in plasma progesterone being evident. It would seem that prostaglandin $F_{2\alpha}$ THAM is useful as a luteolytic agent only in the second trimester. Although side effects of mild panting with occasional vomition and/or defecation were sometimes noted at all dosage levels, they were milder at the recommended lower dosage of 30 μg/kg administered twice daily subcutaneously for 4 days.

Key words: Prostaglandin $F_{2\alpha}$ THAM, bitch, luteolysis.

INTRODUCTION

Population control in dogs is a problem of considerable magnitude. Most of the methods of control are aimed at preventing pregnancies but are often not effective. Stilboestrol may be used for misalliance in bitches within the first week of gestation[2]. However, pregnant bitches are often presented for abortion in more advanced stages of pregnancy.

Jöchle et al.[3] studied the effects of intravenously administered prostaglandin $F_{2\alpha}$ THAM ($PGF_{2\alpha}$) in second and third trimester bitches. They infused 50 μg/kg/h for 6 hours, and on another occasion infused a total of 4 mg (2×2 mg, with a 7 hour interval), but with no resultant abortion. All the dogs vomited within 30 seconds of the infusion, and at the 2 mg dose there was slight incoordination, which ceased when the treatment was stopped. There was a transient drop in plasma progesterone concentrations, but these returned to normal within 72 hours.

Conannon & Hansel[1] used subcutaneous doses of $PGF_{2\alpha}$ spread over 3 days. Of the 7 bitches treated by these workers between Days 28 and 53 post-oestrus, 4 aborted successfully. The dose regimen used by them was 20 μg/kg three times daily or 30 μg/kg twice daily. The developing corpus luteum was found to be refractory to the drug, and bitches showed a permanent drop in their plasma progesterone concentration only when treated after Day 25. They found that when the plasma progesterone concentration dropped below 2,1 ng/ml (6,8 nmol/*l*), the bitches aborted. Those that did not abort had higher plasma progesterone concentrations at the start of the experiments, and they suggested that the luteolytic effect of the PG is dependent on the degree of luteal activity, and that if higher and/or longer dose regimens had been used, the other 3 bitches might have aborted as well. The only side-effects of the PG administration noted were a transient drop in rectal temperature, (averaging 1,39 °C) detectable within 15 minutes and reaching a maximum at 45-60 minutes. This was not seen in overiectomized bitches, and so it was assumed to be associated with the drop in plasma progesterone concentration.

Sokolowski & Geng[6] have claimed luteolysis in the bitch with $PGF_{2\alpha}$ but only at very high doses (1 mg/kg). However, they did not specify the stages of the cycle that the bitches were in at the time of the experiment.

This trial was run to test further the efficacy of $PGF_{2\alpha}$ THAM as a luteolytic agent in the bitch.

MATERIALS AND METHODS

Seven purebred Beagle bitches and one Pyrenean Mountain Dog were used in the trial. Five of the Beagle bitches (Nos 5, 9, 11, 13 & 16) were served at 2 day intervals throughout their oestrus periods by fertile males. The remaining 2 Beagle bitches (Nos 15 & 18) were not served. The Pyrenean Mountain Dog was presented for abortion at 40 days of pregnancy. Four Beagle bitches (Nos 5, 15, 16 & 18) and the Pyrenean Mountain Dog were treated with $PGF_{2\alpha}$ (Lutalyse, Upjohn) at a dose of 30μg/kg administered subcutaneously twice daily for 3-9 days (Table 1). Bitch No. 13 received 60 μg/kg twice daily for 2 days and bitches 9 & 11 received 250 μg/kg once daily for 6 and 4 days respectively. Bitch No. 13 was also treated with 0,3 mg ergometrine three times daily for 12 days for the uterine haemorrhage which followed on the $PGF_{2\alpha}$ treatment.

In an attempt to suppress vomiting and/or diarrhoea, 2 bitches (Nos 5 & 9) also received 30 mg proquamezine (Myspamol, Maybaker) before the $PGF_{2\alpha}$ administration. Bitch No. 11 was also treated with 2,5 IU ocytocin in conjunction with the $PGF_{2\alpha}$ in an attempt to initiate parturition.

Blood was drawn daily in heparin from 6 bitches during the time of $PGF_{2\alpha}$ administration and at various time thereafter. Plasma progesterone was assayed with a competitive protein binding assay[7]. The dosage rates and times of drug administration are outlined in Table 1.

RESULTS

Three of the 4 bitches treated between Days 21-42 after the second service did not produce litters (Nos 5, 9 & 16), while the fourth (Pyrenean Mountain Dog) aborted 10 pups on Day 43. Bitch No. 13, treated in the first week after service, developed a bloody vaginal discharge on the second day of $PGF_{2\alpha}$ administration, was treated with ergometrine as described and eventually whelped a normal litter of 4 pups on Day 61. Bitch No. 11, which

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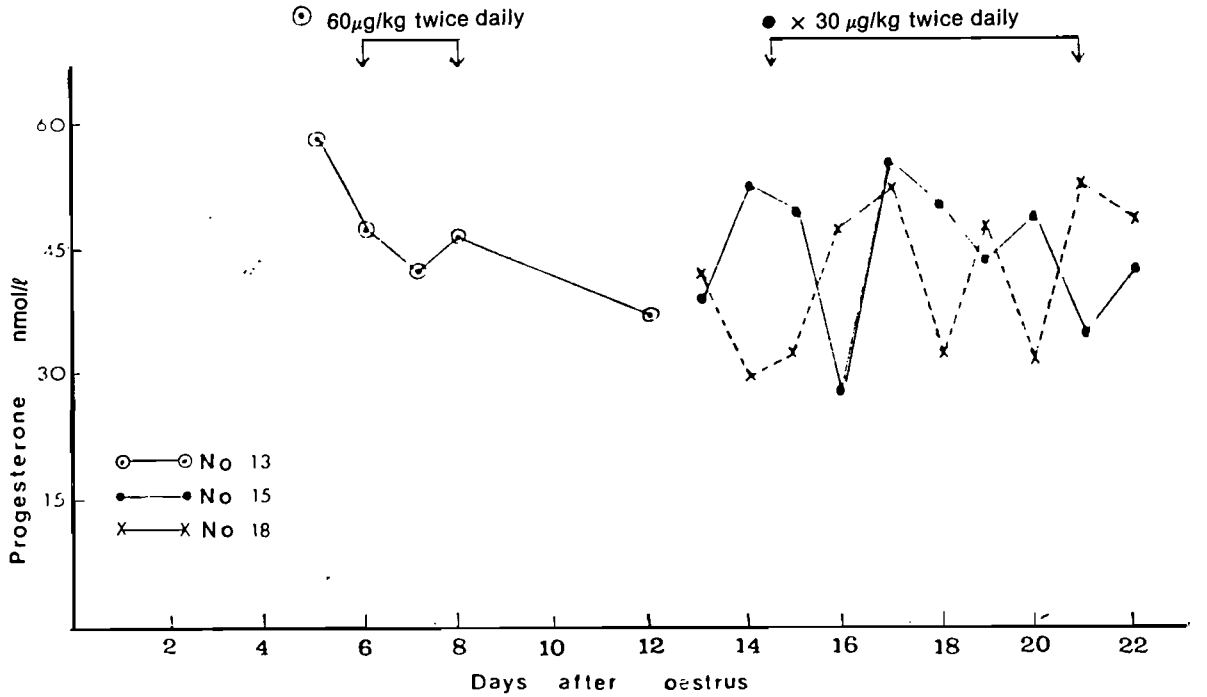

Fig. 1: Plasma progesterone concentrations of 3 bitches during treatment with $PGF_{2\alpha}$

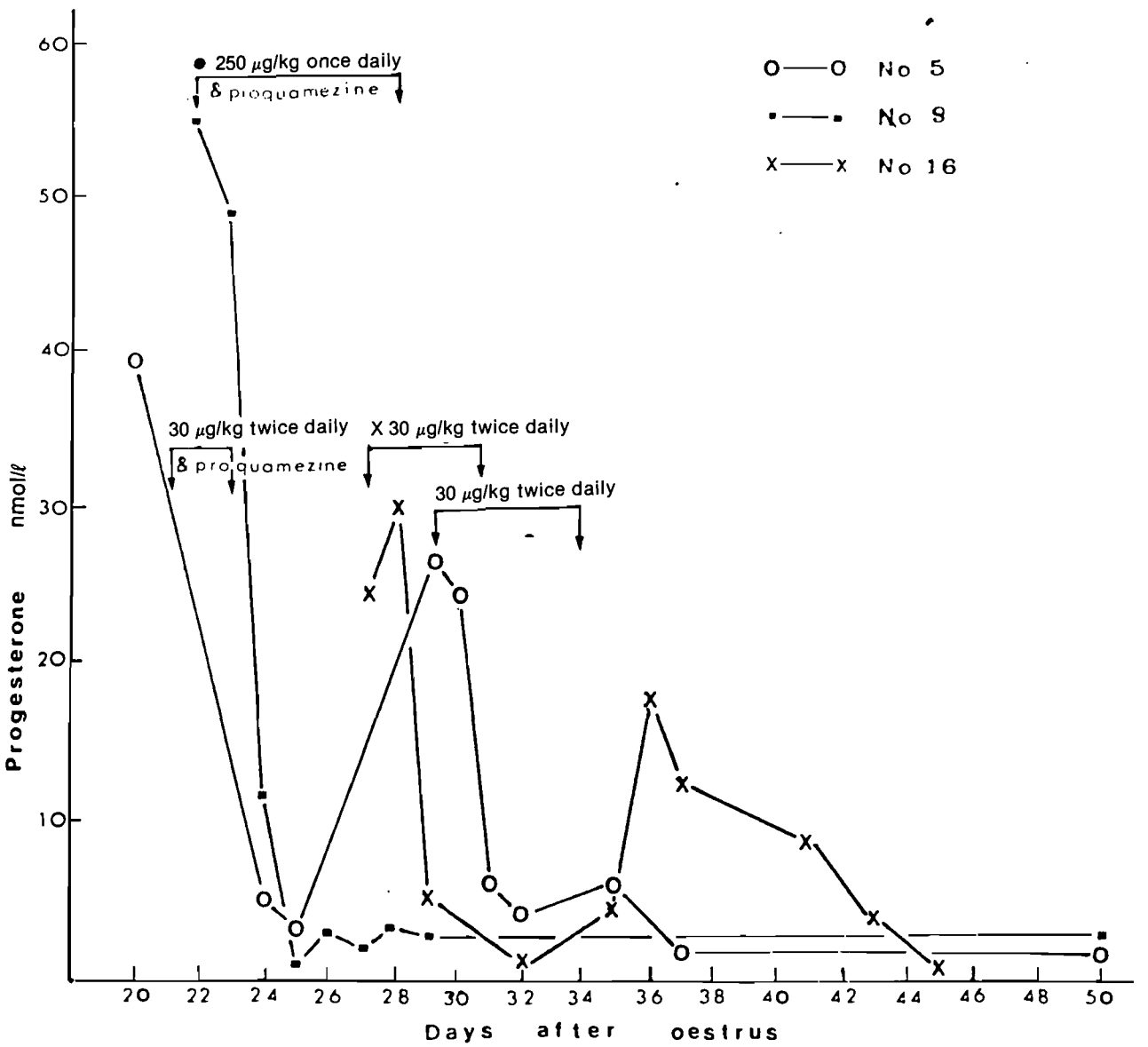

Fig. 2: Plasma progesterone concentrations during treatment with $PGF_{2\alpha}$ and proquamezine

Table 1: DOSAGE RATES AND TIME OF DRUG ADMINISTRATION

Bitch No.	Mated	Days when PG was administered.	Dose of PG	Other treatment
13	Yes	6-7 *	60 µg/kg twice daily	0,3 mg ergometrine three times daily for 12 days, starting on Day 8
15	No	13-19**	30 µg/kg twice daily	—
18	No	13-19**	30 µg/kg twice daily	—
5	Yes	21-23*	30 µg/kg twice daily	30 mg proquamezine 30 minutes before PG
		29-33*	30 µg/kg twice daily	—
9	Yes	22-27*	250 µg/kg once daily	30 mg proquamezine 30 minutes before PG
16	Yes	27-30*	30 µg/kg twice daily	—
		49-52*	250 µg/kg twice daily	—
Pyrenean Mountain Dog	Yes	40-42*	30 µg/kg twice daily	—
11	Yes	52-55*	250 µg/kg once daily	2,5 IU ocytocin once daily for 4 days
		58*	250 µg/kg once	2,5 IU ocytocin once daily for 3 days

* Days after second service
** Days after end of oestrus

Table 2: RESULTS OF $PGF_{2\alpha}$ TREATMENT

Bitch	Mated	Drop in plasma progesterone	Comment
13	Yes	No	Whelped on Day 61
15	No	No	—
18	No	No	—
5	Yes	Partial	Complete luteolysis on second treatment. No abortion products seen.
9	Yes	Yes	No abortion products seen
16	Yes	Yes	No abortion products seen
Pyrenean Mountain Dog	Yes	Not assayed	Aborted 10 pups on Day 43
11	Yes	Not assayed	Whelped on Day 60

was treated in the last 10 days of pregnancy whelped normally on Day 60. These results are presented in Table 2.

It is evident from the maintained plasma progesterone concentrations that luteolysis did not occur in the 2 non-mated bitches treated between Days 13 & 19 after the end of oestrus. This also holds true for one mated bitch (No. 13) also treated during this period (Fig. 1). Partial luteolysis was observed in bitch No. 5 treated on Days 21-23 after the second service, and again on Days 29-33. The plasma progesterone rose after the first treatment (Fig. 2) but showed a marked decrease after the second treatment.

A precipitous drop in plasma progesterone was seen in bitch No. 9. Bitch No. 16 showed an initial fall in progesterone levels, followed by a rise and then a second drop without further treatment (Fig. 2). This bitch showed mammary development on Day 49 with the presence of colostrum in the teats. Radiographs gave a negative result for pregnancy at this time, and the possibility of pseudopregnancy was considered. Plasma progesterone was already low; but in an endeavour to cause an increased tone of the uterus and enhance expulsion of any uterine contents, she was treated agian, this time with high doses of $PGF_{2\alpha}$ (Table 1). Mammary development regressed slowly and she showed a normal oestrus on Day 59.

Two of the bitches (Nos 5 & 9) treated after Day 21 returned to oestrus 3-4 months after the previous oestrus.

Side effects of the PG were mild, amounting to panting with some of the bitches vomiting and/or defecating within 15 minutes of the injection, and lasting for not more than 30 minutes. They were noted at all dose rates, but not in all bitches. They were however much milder at the lower dose rates. The bitches which received the proquamezine did not show any vomiting or diarrhoea.

DISCUSSION

Concannon & Hansel[1] have found that the corpora lutea of bitches in their first trimester of pregnancy are refractory to the effects of $PGF_{2\alpha}$. The results of the present experiment appear to concur with those of Concannon & Hansel[1]. Jöchle & Andersen[4] have stated that the developmental cycle of the corpus luteum in the bitch is divided into 3 stages, namely, formation, full development, and retrogression, and each of these occupy approximately one third of the gestation period (Day 0-21, Day 22-43, Day 44-63). It is an established fact that in cattle the developing corpus luteum is unaffected by $PGF_{2\alpha}$ for 5 days after oestrus[5]. When correlated with Jöchle & Andersen's concept of corpus luteum development in the bitch[4], it offers a possible explanation as to why the corpus luteum of the bitch is refractory to the effects of $PGF_{2\alpha}$ for a relatively long time, that is prior to Day 22, while the corpus luteum is developing.

Abortions do occur in bitches treated with $PGF_{2\alpha}$ in the middle trimester, i.e. when the corpus leteum has fully developed and implantation, which starts around Day 22 post oestrus[4], is taking place. While plasma pro-

gesterone indicated that incomplete luteolysis occurred in bitch No. 5, which was treated from Days 21-23, this bitch responded to a second treatment of $PGF_{2\alpha}$ from Days 29-33 (Fig. 2). It appears therefore that the abortificient effect of $PGF_{2\alpha}$ is primarily due to luteolysis of the mature corpus luteum with little influence on myometrial contraction while the uterus is under high progesterone influence. This appears to be borne out by bitch No. 13, who was treated before implantation with $PGF_{2\alpha}$ and ergometrine, both of which cause uterine contractions, and yet carried her pregnancy to term.

The 3 Beagles which were treated in their second trimester (Nos. 5, 9 & 16) all showed some degree of shortening of their inter-oestral periods. This was most marked in beagle No. 16 which showed heat 2 months after the previous oestrus. Beagles Nos 5 & 9 showed heat 4 months after their previous oestrus periods, which is 2 months shorter than the average inter oestral period of 6 months in our beagle colony. The possibility exists for using $PGF_{2\alpha}$ as a means of bringing bitches into heat earlier, or even as a method of synchronisation in a breeding colony, and warrants further investigation.

Since the drug was as effective in the low and high doses, 30μg/kg administered subcutaneously twice daily for 4 days is the recommended average dose since side effects were fewer and of shorter duration at this dose regimen. Bitches which received proquamezine did not show any vomiting or diarrhoea, but since it was only given to 2 bitches (Nos 5 & 9), no definite conclusions were drawn concerning its use in conjunction with $PGF_{2\alpha}$. Vickery & Mc Rae[8] achieved results similar to those of Concannon & Hansel[1] with a more potent experimental PG analogue (dl 9α; 11α, 15α-trihydroxy-16-phenoxy-17, 18, 19, 29-tetranorprosta 4, 5, 13, transtrienoic acid), which required only a single subcutaneous dose of 20 μg/kg. After Day 30, complete abortion occurred in all cases, and the products of pregnancy were recovered. When treated between Days 20 and 22, pregnancy was terminated in 2 out of 4 cases, but no products of pregnancy were observed. At Day 9, there was no effect on pregnancy and the pups were born normally at term. All patients showed salivation, vomiting, diarrhoea and digging movements, which ceased within 3 hours. The interval between treatment and abortion was 3-10 days. The advantages of this type of product for the practitioner are obvious, but as the product is not yet commercially available, the use of $PGF_{2\alpha}$ as a second trimester abortifacient remains the only means available at this stage.

While $PGF_{2\alpha}$ is not an infallable means of terminating pregnancy in the bitch, it may be tried in cases of unwanted pregnancies, since side-effects at the recommended dose are of short duration and of such a nature that the drug may be used with impunity even in valuable bitches, despite the chances of not procuring an abortion. This work also confirms the value of plasma progesterone assays as a diagnostic aid for the clinician in monitoring the response to the PG, as shown by Beagle No. 5 (Fig. 2). It also highlights the possibility of using plasma progesterone concentrations more frequently as a diagnostic aid in other forms of cyclic abnormalitites in the bitch.

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THE FEASIBILITY OF A RENOGRAM STUDY IN DOGS WITH RADIOPHARMACEUTICAL ^{99m}Tc-DTPA

D.C. LOURENS*, IRENE DORMEHL** and D.J. GOOSEN***

ABSTRACT: Lourens D.C.; Dormehl Irene C.; Goosen D.J. **The Feasibility of a Renogram Study in Dogs with Radiopharmaceutical ^{99m}Tc-DTPA.** *Journal of the South African Veterinary Association* (1982) 53 No. 4, 243-248 (En) Department of Medicine, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

The present study is one of in vivo ^{99m}Tc-DTPA renography, successively involving conscious healthy dogs under acepromazine maleate sedation and dogs under the narcotic sodium thiopental.

It was found that during the running of the renogram the animal had to be kept completely immobile and that constant infusion narcosis with sodium thiopental produced this immobility without affecting the renograms unduly. However administration of a thiopental bolus did have an adverse effect on the renogram.

Sedation with acepromazine maleate significantly increased the time to peak and the excretion phase as presented by the slope. These effects are thought to be due to a decreased blood pressure with concomitant renal bloodpooling and retarded bloodflow.

The radioisotope renogram appears to hold great promise for both clinical and research applications. The equipment required for this application however, is so costly that it would only be financially feasible for major centres.

INTRODUCTION

The clinical assessment of renal function in veterinary science has generally been based on the following facets: history, clinical signs, urine analysis, non protein nitrogen, electrolytes, clearance tests, plasma proteins, radiography and biopsy[7 8 9 11 19 22].

Several substances including radioisotopes have been utilised for determining clearance rates by the kidneys eg. phenosulfonphthalein (P S P) creatinine; inulin; ^{125}I-sodium iothalamate and ^{131}I-sodium iodohippurate[3 10 18 20 22 25]. Handling of the test substance by the kidneys involves glomerular filtration and/or tubular reabsorption and secretion.

A more precise evaluation of renal function can usually be obtained and abnormalities detected earlier with renal clearance methods than with clinicopathological methods[25]. Clearance methods however have not received wide clinical acceptance due to added time, inconvenience and costs. The studies themselves and more particularly the subsequent analytical procedures are time consuming[3 19]. The best methods available to measure the functional integrity of the kidney involve constant infusion techniques, accurately timed urine collections and complicated laboratory procedures. These procedures are not practical for the busy veterinary clinician and are therefore seldom employed[18].

Renal radionuclide investigations in veterinary medicine are presently largely limited to in vitro radioimmunoassays[6 13 19 20 24].

It is possible to gain a great deal of information concerning the integrity of renal function in a non-invasive manner using a suitable radioactive compound intravenously and the conventional instrumentation of nuclear medicine viz scintillation counting and data processing equipment to evaluate the handling of the substance by the kidneys[2 15].

Diethyltriamine penta-acetic acid (DTPA) labelled with ^{99m}technetium (^{99m}Tc) is in general found useful for the measurement of a number of parameters which relate to kidney function[16]. It appears to be excreted at a rate that approximates the glomerular filtration rate (GFR) as to ^{131}iodine -0- iodohippurate which is to 70-80 % extracted from the plasma by the renal tubule[1]. The short half-life of ^{99m}Tc (T½ = 6,06 h) and its high yield of photons of suitable energy (E = 140 kev) make it a very useful isotope for nuclear medical procedures and is therefore chosen as a label whenever possible.

The way the kidney handles ^{99m}Tc-DTPA can be represented by a curve – the renogram – depicting radioactivity as a function of time. The renogram is normally a rapidly changing curve relatively easily interpretable by visual inspection but also rendering numerical parameters of diagnostic value.

The present study was undertaken to evaluate the applicability of the radiorenogram in dogs. To this effect renograms were obtained for healthy dogs using ^{99m}Tc-DTPA. Because of the problem foreseen to keep dogs immobile for any period of time, the effects of sedation and narcosis were studied. These could also serve as a baseline for subsequent research on kidney disease in dogs.

MATERIALS AND METHODS

Renogram technique

The radiorenograms were obtained by administering ^{99m}Tc-DTPA (approximately 42μci/kg body mass) intravenously to each animal and the activity counted externally across the renal areas using a large field gamma-camera (ON Sigma 410) interfaced to a data processor (A2 multiterminal MDS).

Scanning started on a count down of the injection and serial scintigrams were obtained and stored at a rate of 10s/frame for 20 min, giving a total of 120 frames. These 120 images will, with a healthy animal, comfortably cover the excretion of the isotope by the kidneys. The renal areas could be displayed from disc on a video screen for each of the 10 second frames (Fig. 1).

The regions corresponding to these areas were chosen and recorded by selecting the relevant spatial coordinates through the complete series of images and the count rate was then registered as it changed with time. In this way curves were generated for each kidney.

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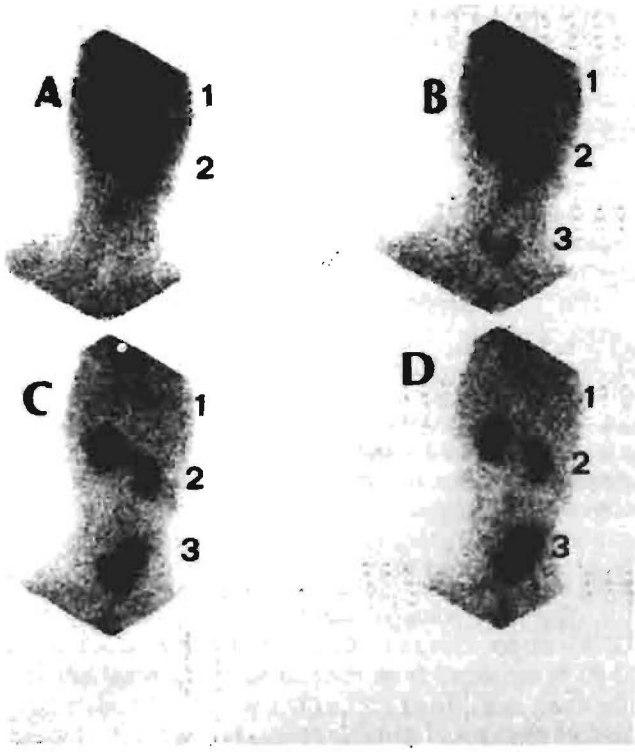

Fig. 1: Scintigram illustrating 4 frames (A to D) at different times of the renogram. Note the progressive reduction in cardiac activity (1) the prominent kidneys (2) and the filling of the bladder (3).

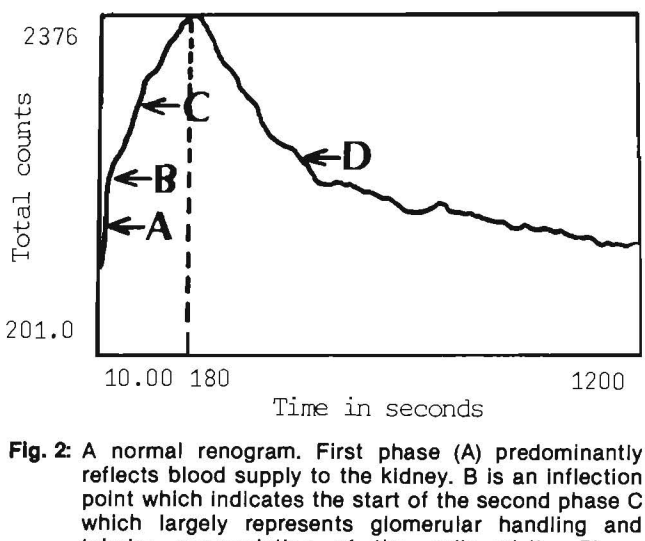

Fig. 2: A normal renogram. First phase (A) predominantly reflects blood supply to the kidney. B is an inflection point which indicates the start of the second phase C which largely represents glomerular handling and tubular accumulation of the radioactivity. Phase three(D) is the urinary excretion phase.

From these curves abnormalities could be identified visually and by analysing the recognised parameters which thus became available.

Analysis of the renogram is generally directed towards 3 sections of the curve[26] (Fig. 2). These 3 phases can be described as follows: Within 15 – 20 s following the injection of the radiopharmaceutical there is a rapidly rising count rate (A) across the renal areas which at about 20-40 s reaches an inflection point (B). The first phase of the renogram reflects the arrival of radioactivity within the area of interest in the form of an early unmixed bolus. Although the blood supplied to the kidney does affect this initial phase it is also influenced by changes of renal function and by background activity. The second phase (C) of the renogram is represented by a less rapid increase in activity reaching a peak at around 2-4 min i.e. The so-called time to peak (TP). All the factors previously mentioned are once more influencing the count rate during this phase but the glomerular handling and tubular accumulation of the radioactivity dominate.

The radioactivity which had by now accumulated in the tubule, at this stage begins to leave the area of interest via the collecting system and reaches the bladder (see Fig 1). Hereafter the curve represents a rapidly falling count rate across the kidneys i.e. the urinary excretion phase 3 (D in Fig. 2). The time it takes for half the maximum activity to disappear (T½) is an indication of the urinary excretion rate. So also is the slope of the curve which is by definition the reduction of counts per time interval at a particular time.

In reality the total renogram is affected by hydration, the position of the kidneys with respect to the camera and medication. It is consequently advisable to keep as many of these variables constant for a series of comparative studies. Background subtraction techniques and computer analyses do however, add to increased accuracy and facilitate the evaluation of the curves.

Animals

Eight purebred female Beagles, approximately 15-20 months old and 11,5 – 12 kg body mass, were used. They were maintained on balanced commercial dog food mixed with meat and milk. Throughout experimentation they were allowed free access to water and food to assure normal hydration. The dogs were healthy as determined by repeated clinical examination, urinalysis, blood urea nitrogen, serum creatinine and haematology. They were trained to accept the experimental procedures without any discomfort.

Isotope

The radiopharmaceutical ^{99m}Tc-DTPA was prepared according to specifications. A vial of DTPA was mixed together with 3-5 ml $^{99m}TcO_{-4}$ (Tc pertechnetate) obtained from a generator (Nuclear Development Corporation of S.A.). A calibrator was used to measure 500μCi of the drug for intravenous administration.

Experimental studies

The 8 Beagles were studied whilst completely unmedicated (Study A), while sedated (Study B) and while under general anaesthesia (Study C).

Study A

Studies were performed in 8 conscious, unmedicated Beagles which were restrained in sternal recumbency. The crystal of a large field gamma camera interfaced to a data processor was positioned dorsally over the lumbar area. The radiolabelled compound (42μCi/kg body mass of the animal) was injected into the cephalic vein through a 20 gauge needle.

Scanning started on a count down on the injection and serial scintigrams were obtained and stored at a rate of 10 s/frame for 20 min (See Renogram technique).

The renograms thus obtained were evaluated and results tabulated. Immediately after the test the dogs were placed in a radio-isoptope excretion room for 24 h.

Study B

Studies were performed on 8 Beagles sedated with 4 mg acepromazine maleate (Acetylpromazine, Milvet) intravenously into the cephalic vein. The positions and procedures were exactly the same as described in Study A.

Study C

Renogram studies were performed on 7 anaesthetized Beagles. Anaesthesia was induced with sodium thiopental (Intraval, May Baker) and maintained at a constant infusion rate with the aid of an Infusomat (B. Braun Melsungen A.G.). This preparation consisted of a solution of 1 g sodium thiopental in 200 ml normal saline which was delivered at a rate of 70 ml/h ie 1,17 ml/min intravenously.

Intra-arterial blood pressure and heart rates were continuously measured with a physiological polygraph monitor (Nihon Kohden) and recorded at 0 min, 5 min, 10 min and 20 min after onset of the experiment in the case of 5 of the 7 dogs. For this purpose an intra-arterial catheter was inserted into the femoral artery. The tip of the catheter was connected to a Statham pressure transducer with a 3 way stopcock for saline flushes to keep the catheter open.

In this study endogenous creatinine clearance was performed on 5 dogs as follows:
The urinary bladder was catherized and emptied. The urine obtained was assayed for its creatinine concentration (Jaffè reaction on Astra by Beckman). Arterial blood was collected for serum creatinine determination (Jaffè reaction on SMA² by Technicon). To avoid radioactive contamination, urine and serum creatinine levels were determined before administration of the isotope. After the isotope was administered the urine volume was recorded for ± 30 min.

In study C the dogs were supinely positioned to permit dorsal imaging of the kidneys. The reason for this position was to facilitate arterial and bladder catheterization (Fig. 3).

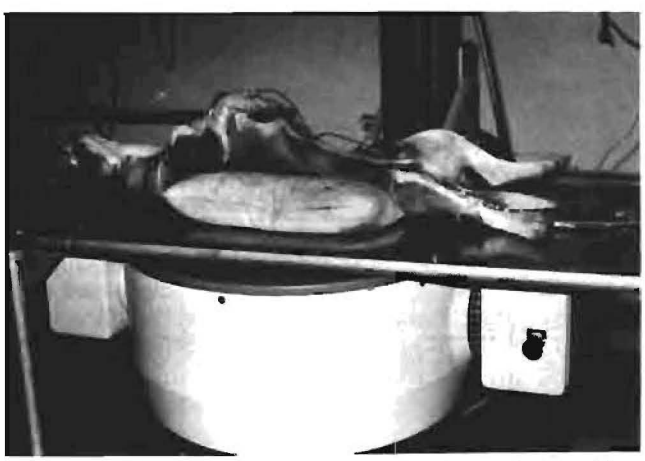

Fig. 3: Example of anaesthetized dog in the supine position for dorsal imaging of the kidneys by the gamma camera.

RESULTS

Table 1 summarizes the results as obtained from the renograms (Fig. 4). The mean heart rate, blood pressure and endogenous creatinine clearance of dogs under thiopentone anaesthesia are also included.

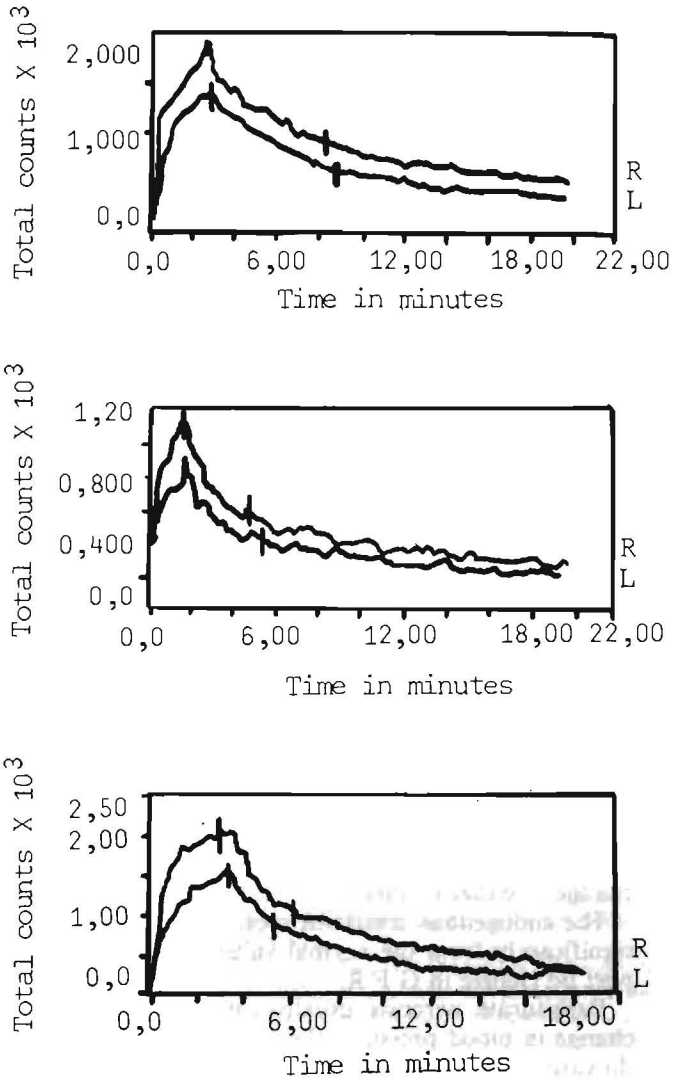

Fig. 4: Right and left kidney renograms for the same animal under the following conditions:-
a. Awake and unmedicated
b. Thiopentone infusion anaesthesia
c. Acepromazine maleate sedation

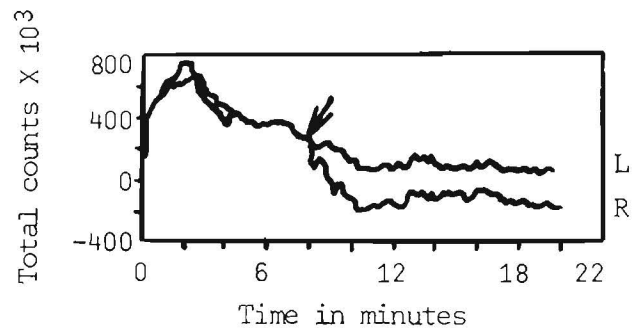

Fig. 5: Renogram of an unmedicated dog. The irregularities indicate movement (Arrow) by the dog due to restlessness.

Fig. 4 is an example of a typical renogram for the same animal under the 3 different studies. Fig. 5 is a renogram of a conscious dog showing the irregularities due to movement while Fig. 6 is that of an anaesthetized dog to show the effect of a bolus injection of thiopentone.

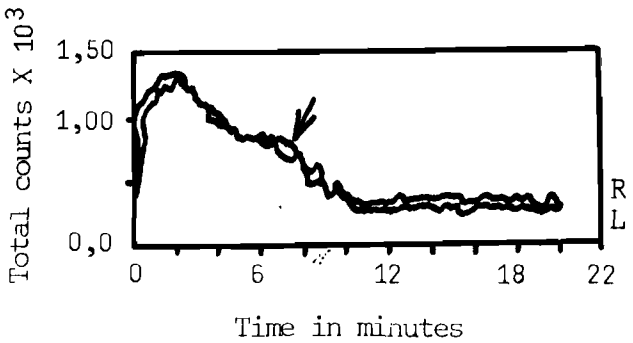

Fig. 6: Radiorenogram of a dog under anaesthetic viz a bolus injection of thiopentone. Arrow indicates where a booster anaesthetic was administered.

DISCUSSION

From Table 1 it can be seen that there is often a difference between the left and right kidney parameters. These differences are however statistically insignificant ($P<0,05$).

It seems as though a slight reduction in TP occurs during thiopentone sodium anaesthesia with respect to the awake dogs. The difference (~13-14%) is, however, statistically insignificant ($P<0,05$).

Since TP is influenced by renal bloodflow and/or G F R this fact could imply that there is no observable change in either of these.

The endogenous creatinine clearance does not differ significantly from the normal value which would support no change in G F R.

Barbiturate narcosis usually causes a slight or no change in blood pressure; the effect on the whole cardiovascular system is usually mild. Hypotension is partially caused by suppression of ganglionic transmission and the vasomotor centre in the medulla during deep narcosis. The general impression gained from the literature is that barbiturates suppress renal function[5,12].

The mean heart rate and blood-pressure of the Beagles under thiopentone anaesthesia were 162/min and 105/50 mm Hg respectively. The mean heart rate for healthy Beagles is given as 138/min[23]. Indirect blood pressure measurements give values of 112/57 and 111/69mm Hg[17]. Our results show a very slight reduction in blood pressure and a somewhat increased heart rate. In spite of this it seems that renal function is unchanged by the anaesthetic. Various physiological mechanisms are activated by a lowered systemic blood pressure. The renin/angiotensin mechanism will lead to vasoconstriction of the efferent arterioles which will tend to keept the filtration rate constant[21]. Sympathetic stimulation during decreased blood pressure can also lead to selective constriction of the efferent arteriole[4] and an increased heart rate. These mechanisms can thus be responsible for maintaining renal perfusion and GFR within normal limits.

A factor which may play a role is the infusion of normal saline together with thiopentone at a rate of 70 ml/h. It may be speculated that even a small amount of Nacl could be enough to stimulate renal function.

The TP obtained from the dogs under acepromazine sedation is found to be significantly longer ($P<0,05$) than values pertaining to conscious dogs. This could correspond to reduced renal blood flow and/or a reduction in the glomerular filtration rate and could be secondary to induced hypotension.

The effects of phenothiazines such as acepromazine on the autonomic nervous system are complex and often unpredictable. It is a reversible α-adrenergic blocking agent and can thus result in decreased blood pressure after parenteral injection[12].

Table 1: MEAN RENOGRAM RESULTS OF STUDIES A, B AND C. THE MEAN BLOOD PRESSURE, HEART RATE AND ENDOGENOUS CREATININE CLEARANCE RATE OF BEAGLES UNDER THIOPENTONE ANAESTHESIA IS ALSO GIVEN

Parameters	Unmedicated (Study A) Eight dogs		Thiopetone infusion (Study C) Seven dogs		Acepromazine maleate (Study B) Five dogs		% difference and statistical significance	
							Unmedicated thiopentone	Unmedicated Acepromazine
	R	L	R	L	R	L	R L	R L
TP(min)	2,38±0,33	2,39±0,29	2,07±0,26	2,05±0,36	3,43±0,969	3,43±1,002	−13% −14% statistically not significant P<0,05	+44% +44% statistically significant P<0,05
T½(min)	8,39±2,11	9,05±4,26	6,48±1,8	6,20±2,2	7,07±1,23	7,53±1,8	−23% −31% statistically not significant P<0,05	−16% −17% statistically not significant P<0,05
Slope	−92,5±36	−77,0±50,0	−133±80	−108±47	−266,3±145	−244,2±160	+40% +40% statistically not significant P<0,05	+188% +217% Statistically significant P<0,05
Blood pressure (mm Hg.)			105/50					
Heart rate (per min)			162					
Endogenous creatinine clearance			2,45 ml/min/kg					

R – Right Kidney
L – Left Kidney

The arterioles of the kidney contain both ∝ (+ + +) and ß2(+) receptors. The kidney also contains dopaminergic receptors of which stimulation will lead to vasodilation. The physiological importance of this has not yet been evaluated[12]. The importance of increased TP with acepromazine is speculative. It may possibly be secondary to hypotension or may be the reflex sympathetic effect when the efferent arteriole in the kidney is blocked. The receptors in the renal arterioles can be blocked which may lead to vasodilatation and subsequent pooling of blood in the kidneys. There may be a systemic decrease of hydrostatic blood pressure as well, which is the most important factor for filtration to occur.

The T½ which is, as previously explained, an indication of the rate of urinary excretion is found not to be significantly affected ($P < 0,05$) by either pentothal or acepromzaine. The state of hydration of the dog has a significant influence of the T½ values making it an unreliable parameter for diagnostic purposes. Note the large standard deviation.

A third parameter which is often deduced from the renogram is the slope of the curve between TP and T½, defined as: $\frac{\text{½ Maximum count rate}}{(T½ - TP)}$

From the definition the slope would likewise point to changes in urinary excretion rate. The slopes are statistically unaffected ($P < 0,05$) bv sodium pentothal, as would be expected from the T½ results.

On the other hand the slopes obtained from dogs under acepromazine sedation differ very significantly from those of both conscious dogs and dogs under sodium pentothal.

This very large increase in the slope could not be explained in terms of changes in T½ or TP and is found to be due to an increased maximum count rate across the kidneys. The administered activity never varied by more than 10 % during the entire study and this cannot account for increased accumulation of activity in the kidney. It seems as though a degree of pooling of radioactivity takes place during the early stages of acepromazine sedation. As speculated before, it is possible that this could account for increased maximum count rate.

As regards technique the single most important problem was movement in the unmedicated as well as the acepromazine sedated dogs, e.g. only 5 out of 8 acepromazine attempts were successful.

In Fig. 5 the steplike effect of even mild movement can be seen; this can lead to wrong interpretaion and evaluation of the renogram. This steplike effect is due to a spatial shift of the kidneys with respect to their original position to which coordinates the computer software repeatedly refers.

A further disadvantage is the exhaustion of both animal and technicians to keep unmedicated dogs immobile for a 20 min test period. The most important disadvantage was that parameters like heart rate, blood pressure, and endogenous creatinine clearance could not be measured in the unmedicated and sedated dogs.

All these problems were excluded when the renogram study was performed while the animals were anesthetized. In this study it was found to be important to keep the level of narcosis constant with the Infusomat as a bolus adjustment could also effect the renogram curve (Fig. 6). This could probably be due to a cardiovascular effect. Similar results have been found in baboons (*Papio ursinus*)[14].

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BOOK REVIEW — BOEKRESENSIE

GROWTH AND THE DEVELOPMENT OF PATTERN

Edited by R.M. GAZE, V. FRENCH, M. SNOW and D. SUMMERBELL

1981, Cambridge University Press, Cambridge, London, New York, New Rochelle, Melbourne & Sydney, being a Supplement to Volume 65 of The Journal of Embryology and Experimental Morphology. ISBN 0 521 24557 5. Supplement, ISSN 0022-0752; vol. 65. South African price: R52,50.

In pure biological research one finding in the pursuit of a particular problem more often than not uncovers a number of subsidiary ones, the solution of each of which is germane to the understanding of the *main* issue. The researchers, for practical reasons, are forced to narrow their range and intensify their focus, until eventually sets of concepts and series of terms are developed that only the initiated can understand fully and handle ably. To further the interests of parties concerned and to overcome the 'dilution effect' of publication in a number of different journals, new specialist journals are created, but these, for reasons of locality and human nature, soon multiply. In an effort to overcome the inevitable dichotomies and drifting apart, there has been an increasing tendency to hold meetings, discussions, conferences, colloquia and symposia, and to publish the proceedings in book form, either as a volume in a series or as a supplement to a journal.

The book under review is no exception to this practice: it forms the supplement to Volume 65 of The Journal of Embryology and Experimental Morphology and is the outcome of a Discussion Meeting on the topic of 'Growth and the Developmental Pattern', held under the auspices of the Company of Biologists Ltd at White House, Chelwood Gate, Sussex from 25 to 30 May 1981. In the words of the editors: 'The main theme of this meeting was specifically to examine the connexions between two aspects of development that have become somewhat dissociated over the years: growth and development of pattern.'

Twenty-four authors, mostly from the United Kingdom, but also from the United States of America, France, the Federal Republic of Germany and Yugoslavia, have contributed 17 papers. Five of these are more of the overview type, in which the results of a number of experiments, mostly done by the authors themselves, are reviewed and discussed in the light of the main theme of the meeting. The other twelve are reports of experimental work in the usual journal style.

A number of diverse aspects of the central theme is covered comprising observations on and analysis of:

(a) urodele and chick limb bud regeneration, and of genetic limb malformation in mice;
(b) wing disc regeneration, genetic wing malformation and the effects of mosaicism in populations of mutant cells with different growth rates in wing discs of *Drosopila;*
(c) the pathways taken by the axons of ectopic sensory neurons in fruit flies, crickets and locusts;
(d) the potentialities of pre-primitive streak and early primitive streak rat embryonal shield ectoderm when grafted under the fibrous capsule of the adult kidney;
(e) the relationship between growth rate and regionalisation in amphibian, avian and mammalian (mouse) embryos, with the question whether development is clonal;
(f) the control of somite formation in mouse embryos;
(g) the pattern of growth and morphogenesis of the cranial neural epithelium of rat embryos during neurulation (including ultrastructural aspects);
(h) 'catch-up growth' in rats; and

a review of 6 growth factors, with suggestions for further study of the role of such factors in pattern formation.

The resulting over-all picture, although emphasising the relationship between growth and morphogenesis, is as complex as a patchwork quilt with no simple straightforward pattern being discernible. This is no way detracts from the excellence of the papers, but leaves the average, non-specialist reader musing wistfully that development, like love, 'is a many-splendoured thing'.

It is a pity that the discussions – surely there must have been exciting and thought-provoking discussions – or at least précis thereof, have not been published; even more so that the editors have not presented a final summing-up. (Time and cost factors?) One fain would have had the advantage of their expertise to place the diverse aspects into perspective.

It is a saddening but sobering thought that, after a century of painstaking and often brilliant research scientists cannot come up with anything more concrete than the final citation of Waddington (1957) by one group of contributors. 'It seems impossible to hope that we shall ever discover any single basic mechanism of pattern formation or morphogenesis, as we may still hope to find, for instance in the mechanism of protein synthesis and its control by genes, the fundamental mechanism for substantive differentiation. In discussing pattern formation and morphogenesis, therefore, one can hardly hope to do more than provide a number of illustrations of the general nature of the processes which are at work.'

Be that as it may, in the final instance we shall have to attempt to bridge the gap not only between growth and pattern formation, but between developmental and molecular biology in respect of morphogenesis (as has been done to some extent by Smith and by Morriss-Kay in this work) no matter how complex the infrastructure required for that bridge. The editors, in their introduction, warn against the idea that everything can be explained in terms of physics and chemistry, as this 'has been the cause of an unhelpful tendency to look for simplicity where it may not exist'. But who, before 1943 (Avery), or 1953 (Watson & Crick), or even 1965 (Nirenberg & Khorana) for that matter, could have believed that the relatively simple and monotonous chemical configuration of DNA was capable of carrying such a highly complex and vast array of messages as required for the genetic code? (Shades of Spemann and his concepts of entelechy!) could it be that 'positional effect' will one day be translated as 'over-all effect of cell-to-cell interaction ("signalling") by virtue of their chemico-physical surface topography which is continually adapting to follow a specific gradient ("French flag phenomenon")?'

H.P.A. de Boom

THE TREATMENT OF *MORAEA POLYSTACHYA* (THUNB) KER-GAWL (CARDIAC GLYCOSIDE) POISONING IN SHEEP AND CATTLE WITH ACTIVATED CHARCOAL AND POTASSIUM CHLORIDE

J.P.J. JOUBERT* and R. ANITRA SCHULTZ*

ABSTRACT: Joubert, J.P.J.; Schultz, R. **The treatment of *Moraea polystachya* (Thunb) Ker-Gawl (cardiac glycoside) poisoning in sheep and cattle with activated charcoal and potassium chloride.** *Journal of the South African Veterinary Association* (1982) 53 No. 4 249-253, (En) Section of Toxicology, Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa.

Six out of 6 steers survived a lethal dose of *Moraea polystachya* (blue tulp) when treated with activated charcoal alone and 3 out of 4 wethers survived when treated with activated charcoal plus potassium chloride. Barbeque charcoal was not of therapeutic value. An increase in the level of serum potassium served as indicator of acute poisoning in only a third of the animals.

Key words: Cardiac glycoside poisoning, *Moraea polystachya,* activated charcoal, potassium chloride.

INTRODUCTION

Moraea polystachya (Thunb) Ker-Gawl (blue tulp), is one of many members of the Iridaceae known to contain bufadienolide cardiac glycosides as its toxic principle[6]. It is found throughout the Cape Province, Western Free State and Western Transvaal and is most poisonous in the flowering stage which usually extends from February to April[9].

The clinical signs of poisoning are of gastro-intestinal, cardiac and neuromuscular origin. Ruminal stasis, with consequent bloat and severe diarrhoea represent the gastro-intestinal symptoms. The cardiac signs include bradycardia, tachycardia, arrhythmia and atrio-ventricular dissociation; and the neuromuscular signs, hypersensitivity, paresis, ataxia, unsteady gait and even paralysis. The paresis is often accompanied by by severe dyspnoea and drooling of saliva[5 6].

The successful treatment of Transvaal slangkop (*Urginea sanguinea*) poisoning has been reported[4]. The object of the investigation on which this article is based was to answer certain questions e.g.:

1) Would potassium chloride and activated charcoal be effective in the treatment of other cardiac glycoside plant poisonings?
2) Is the treatment effective in other animal species?
3) Would the use of charcoal alone be as effective?

In this experiment, poisoned sheep were treated with both activated charcoal and potassium chloride and a group of steers only with activated charcoal. Another group of steers received ordinary barbeque charcoal. As this is inexpensive and readily available, the possibility of it being effective, had to be investigated.

Serum K, Na, Ca and Mg levels were monitored in the cattle whenever posible. The object of the electrolyte study was to determine whether serum K levels would be a useful guide in the diagnosis of acute poisoning[1].

MATERIALS AND METHODS

M. polystachya plants, in the flowering stage, were collected on the farm Rietfontein near Bloemhof in the Western Transvaal. Their indentity was confirmed by the Botanical Research Institute, Pretoria, and they were air-dried, milled and stored at about 4 °C until used.

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Twelve Merino wethers (milk toothed-4 toothed), with live masses varying between 28 kg and 37 kg were used as well as 20 Afrikaner cross steers, approximately 1 year old and with live masses varying between 147 and 243 kg.

The sheep and steers were deprived of water and food for 24 hours before mass measurements were recorded. They were then dosed with the dried *M. polystachya* material. Both tulp and therapeutica were suspended in water before dosing per stomach tube.

Four of the sheep were dosed with tulp at 0,5 g/kg, 0,75 g/kg, 1,0 g/kg and 1,5 g/kg respectively to determine the approximate lethal dose. The remaining 8 sheep were divided into 2 equal groups. All of them were dosed on Day 0 with 1,25 g/kg tulp. One group received no subsequent treatment, while the animals in the other group were given 5 g/kg activated charcoal plus 1 g/kg potassium chloride 4-6 h after being dossed with the tulp (Table 1).

Two of the steers were initially dosed with tulp at doses of 0,75 g/kg and 1 g/kg respectively to determine the approximate lethal dose. The remaining 18 animals were divided into 3 groups of 6 each (Table 2). All of them were dosed with tulp at 1,25 g/kg on Day 0 of the experiment. One group served as control and received no treatment, while the second group was dosed with 5 g/kg of activated charcoal 8-12 h after the administration of tulp. The animals in the third group received barbeque charcoal as follows: 2 steers were dosed once with 5 g/kg of finely milled barbeque charcoal mixed with water, and another pair received 10 g/kg of barbeque charcoal which had been milled and washed in distilled water and then dried at 190 °C (for 24 h). The remaining 2 animals each received 2 doses of of 10 g/kg of similarly treated barbeque charcoal.

Electrocardiographic (ECG) recordings were as previously described[7 8], while clinical examinations and routine blood chemical analyses were periodically carried out, each animal serving as its own control. The first blood specimens were collected before the tulp was dosed and were analysed comprehensively. Subsequent specimens were taken from the steers only if circumstances permitted and were analysed for serum K, Na, Ca and Mg.

RESULTS

The results of the experiment and other details are summarized in Table 1 and 2.

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Table 1: TREATMENT OF *MORAEA POLYSTACHYA* (1,25 g/kg) POISONING IN SHEEP WITH ACTIVATED CHARCOAL (5 g/kg) PLUS POTASSIUM CHLORIDE (1 g/kg) PER OS

Treated Group

Sheep		Inter-val* (h)	Before treatment Clinical signs and ECG changes	After treatment		Fate
No.	age			Clinical signs	ECG changes	
1	2t	6	6 h: Inappetence forced respiration	9 h: Inappetence forced respiration 24 h: Rumination slightly depressed, ate well	n/a	Survived
2	4t	6	6 h: Inappetence, forced respiration	6 h 15 min: Very apathetic, respiration forced, groaned	n/a	Died 9 h
3	mt	4	4 h: Inappetence, decreased rumination. ST segment depressed and increased amplitudes e.g. in Lead II the QRS wave (3,0mV) and T wave (1,7mV)	Day 1: diarrhoea Day 2: ruminated and ate	Depressed ST segment, increased amplitude of QRS wave and T wave until Day 5	Survived
4	4t	4	4 h: Inappetence, decreased rumination. Slight sinus arrhythmia	6 h: Vomition Day 1: faeces soft anorexia Day 2: remunited and ate	n/a	Survived

Control Group

Sheep		Clinical signs	ECG changes	Fate
No.	age			
5	4t	6 h: Inappetence 7 h: Severe diarrhoea 9 h: Forced respiration, foamed at mouth, remained recumbent	Within 6 h conspicously increased amplitudes e.g. in Lead II the QRS wave (4,0 mV) and T wave (1,7 mV)	Died within 24 h
6	2t	6 h: Inappetence 9 h: Severe diarrhoea, foamed at mouth, remained recumbent	Within 6 h increased amplitude e.g. in Lead II the QRS wave (2,9 mV) and T wave (1,5 mV)	Died within 24 h
7	4t	4 h: Inappetence, ruminal atony, increased respiratory rate.	4 h: n/a	Died within 24 h
8	4t	4 h: Decreased ruminal movements	4 h: n/a	Died within 24 h

*Interval = time interval between dosing of tulp and treatment
t = tooth
mt = milk toothed
n/a = nothing abnormal

The approximate lethal dose of the tulp for sheep

The sheep dosed with 0,5 g/kg of tulp survived without treatment while the one which had received 0,75 g/kg, died after 6 days. Tulp at doses of 1,0 g/kg and 1,5 g/kg killed the sheep within 24 h.

Treatment of sheep

Three out of 4 treated sheep survived a tulp dose of 1,25 g/kg while none of the controls did. Clinical signs seen were: inappetence, ruminal atony, forced respiration, severe diarrhoea and recumbency. Increased ECG wave amplitudes were observed within 4-6 h after dosing tulp in one of the treated sheep (in which it lasted for 5 days) and in 2 of the control sheep.

Approximate lethal dose for steers

The steers dosed with 0,75 g/kg and 1,0 g/kg of tulp died within 47 h and 27 h respectively.

Treatment of steers

All of the steers treated with activated charcoal survived. None of the controls nor those treated with the barbeque charcoal survived.

The clinical signs were inappetence, ruminal atony, watery diarrhoea and dehydration, forced respiration, salivation (probably due to paresis of the deglutition mechanism) and recumbency.

Increases in the amplitude of ECG waves were recorded in all animals of the control group, in some of those in the barbeque charcoal group (4 animals before and 4 after treatment) and in all the cattle in the activated charcoal group, and were maintained for as long as 24 h after being dosed with the tulp.

Serum Ca and Mg concentrations

No significant changes in concentrations of Ca and Mg were seen in serum of the steers.

Serum K Concentrations

Only 3 of the control steers were bled more than once. One of them showed a markedly increased concentration of serum K (17,5 mmol/ℓ) shortly before death, while that of the other 2 changed from concentrations of slightly above normal 6-7 mmol/ℓ) before tulp-dosing to normal (3,9-5,8 mmol/ℓ)[3]. All animals in the group treated with barbeque charcoal were bled more that once and 2 of them showed markedly increased serum K concentrations (12,5 and 14,3 mmol/ℓ) before death, while that of the other 4 changed from slightly above normal to normal. Two of the animals in the activated charcoal group showed markedly increased concentrations (10,3 and 11,3 mmol/ℓ) for up to 2 days after dosing with the tulp and them returned to normal. The remaining 4 animals in this group initially had slightly raised serum K concentration but these returned to normal shortly afterwards.

Serum Na concentrations

Eight of the steers, out of all groups, showed high serum Na concentration before tulp dosing: these returned to normal 8-24 h later. The remaining 10 steers had normal values throughout.

DISCUSSION

Typical clinical signs of cardiac glycoside poisoning developed after dosing the dried plant material. In general, increased wave amplitudes were seen in the ECG recordings of both sheep and cattle after dosing them with tulp. These high amplitudes persisted for some time after treatment before returning to normal limits. This is consistent with ECG changes to be expected in cardiac glycoside poisoning.

The initial increase and subsequent decrease of serum electrolytes in the steers may perhaps be explained by the development of a slight haemoconcentration due to the deprivation of water and food for 24 h before onset of the trial and rehydration by the volume of water (2-5ℓ) in which the tulp was suspended as well as the imbibition of water after dosing.

Bismuth et al.[1], reported that 24 human patients, who succumbed to digitalis poisoning, had initial (i.e. at admission to hospital) serum K levels of 5,5 mmol/ℓ or more, while 67 patients who recovered had initial levels of 5 mmol/ℓ or less. Normal concentration determined by them varied between 3,21 and 3,91 mmol/ℓ. Chung & Thomas according to the Bismuth et al.[1], however, found no correlation between serum K concentrations and the severity of digitalis poisoning in 17 patients.

The serum K concentrations of only a third of the steers in this experiment, increased markedly with acute poisoning, indicating that the serum K level of acutely poisoned animals is not a reliable diagnostic aid.

Barbeque charcoal was ineffective even at a dose of 20 g/kg.

This barbeque charcoal was heated in an unsuccesful attempt to activate it. According to the British Pharmaceutical Codex[2], charcoal can be activated by the addition of inorganic salts and heating it in a stream of activating gases such as stream of carbon dioxide. This process is beyond the scope of the average veterinarian or a farmer, who would thus be compelled to use the commercial product.

Table 2: TREATMENT OF *MORAEA POLYSTACHYA* (1,25 g/kg) POISONING IN CATTLE*

Activated charcoal (5 g/kg) treatment

Cattle No.	Interval** (h)	Before treatment Clinical signs and ECG changes	After treatment		Fate
			Clinical signs	ECG changes	
1	8	Decreased rumination, salivation Increased amplitudes e.g. in Lead II the QRS wave (2,1 mV) and T wave (1,8 mV)	24 h: Ruminal atony, faeces soft, apathy. 48 h: Appetite improved slightly	24 h: Increased amplitudes e.g. in Lead II the QRS wave (2,4 mV) and T wave (1,7 mV)	Survived
2	8	Decreased rumination, salivation. Increased amplitudes e.g. in Lead II the QRS wave (2,2 mV)	24 h: Decreased rumination, faeces soft, apathy. 48 h: Appetite improved	24 h: Increased amplitudes e.g. in Lead II the QRS wave (2,5 mV) Slight sinus arrhythmia	Survived
3	8	Ruminal atony, increased heart rate. Increased amplitude of the QRS wave in Lead II	24 h: Rumination improved slightly, faeces soft 48 h: Faeces firm, rumination as above	24 h: Amplitudes of QRS wave slightly increased	Survived
4	8	Ruminal atony Increased amplitudes e.g. in Lead II the QRS wave (2,0 mV)	24 h: Decreased rumination, soft faeces 48 h: Faeces firm	24 h: Increased amplitudes 48 h: Sinus arrhythmia	Survived
5	12	Ruminal atony Increased amplitudes e.g. in Lead II the QRS wave (3,1 mV)	20 h: Diarrhoea and ruminal atony 36 h: Slightly improved rumination	36 h: Increased amplitudes e.g. in Lead II the QRS wave (2,8 mV)	Survived
6	12	Decreased rumination Increased amplitudes e.g. in Lead II the QRS wave (2,1 mV)	20 h: Diarrhoea 36 h: Rumination restored, faeces soft	n/a	Survived

Table 2 continued Barbeque charcoal treatment

Cattle No.	Interval** (h)	Dose (g/kg)	Before treatment	After treatment		Fate
				Clinical signs	ECG changes	
7	8	5	Decreased rumination, salivation Increased amplitudes e.g. in Lead II the QRS wave (2,0 mV)	24 h: Ruminal atony, severe watery diarrhoea and dehydration, recumbent	24 h: Tachycardia (170/min) Increased amplitudes e.g. in Lead II the QRS wave (3,1 mV) and T wave (1,2 mV)	Died within 29 h
8	8	5	Decreased rumination, salivation Increased amplitudes e.g. in Lead II the QRS wave (2,3 mV)	24 h: Severe diarrhoea and dehydration, staggering gait	24 h: Extra-systoli, increased amplitudes e.g. in Lead II the QRS wave (3,1 mV) and T wave (1,9 mV)	Died within 30 h
9	8	10	Ruminal atony, faeces soft	24 h: Severe diarrhoea, dehydration, apathy, salivation	24 h: Increased amplitudes e.g. in Lead II the QRS (3,0 mV)	Died within 48 h
10	8	10	Ruminal atony	24 h: Severe diarrhoea and dehydration, salivation	24 h: Tachycardia (200/min) Increased amplitudes e.g. in Lead II the QRS wave (3,3 mV)	Died within 48 h
11	13 and 17	10 and 10	Ruminal atony and diarrhoea. Increased amplitudes e.g. in Lead II the QRS wave 4,0 mV) and T wave (2,3 mV)	As before	—	Died within 33 h
12	13 and 17	10 and 10	Ruminal atony and diarrhoea. Increased amplitudes e.g. in Lead II the QRS wave (2,2 mV)	As before	—	Died within 33 h

Control Group

Cattle No.	Clinical signs	ECG changes	Fate
13	16 h: Ruminal atony, severe diarrhoea and dehydration, respiration slightly forced	16 h: Tachycardia (180/min). Slightly increased amplitudes	Died within 48 h
14	17 h: Ruminal atony, severe diarrhoea and dehydration, forced respiration	16 h: Increased amplitudes e.g. in Lead II the QRS wave (2,8 mV). Severe ST segment depression, runs of ventricular tachycardia	Died within 25 h
15	Ruminal atony, apathy	8 h: Slightly increased amplitudes	Died within 24 h
16	24 h: Ruminal atony, recumbent, salivation	8 h: Increased amplitudes e.g. in Lead II the QRS wave (2,2 mV)	Died within 28 h
17	In *extremis*	—	Died within 23 h
18	In *extremis*	—	Died within 22 h

*Cattle = *c.* 1 year old steers
**Interval = time interval between dosing of tulp and treatment
n/a = nothing abnormal

Activated charcoal on its own, was as successful in the treatment of cardiac glycoside poisoning as the combination of activated charcoal and potassium chloride. Futhermore, it has been demonstrated that poisoning with either tulp or slangkop[4], can be treated effectively, in both cattle and sheep, with activated charcoal.

In natural cases of poisoning, the efficacy of activate charcoal may be influenced by the amount of tulp ingested, the time interval before treatment and the extent of dehydration (due to severe diarrhoea) of the animal. Where dehydration occurs, fluid therapy can be life saving. The dose of activated charcoal used to date, was 5 g/kg which is a large one to administer. It will therefore, be worthwhile to establish what the minimal effective dose of activated charcoal in cases of tulp poisoning is.

It often happens, in natural cases of poisoning, that animals die due to stress when they are caught and restrained in order to be treated. This is an aspect which requires attention in further research.

ACNOWLEDGEMENTS

The staff of the Toxicology Section is thanked for their assistance, especially Mr B.P. Maartens, who rendered invaluable assistance with the partical aspects of this experiment. We would also like to thank Mr C.J. Bezuidenhout for providing the plant material for this experiment.

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BOOK REVIEW

BOEKRESENSIE

ANIMAL ANATOMY AND PHYSIOLOGY

JESSE F. BONE

2nd Edn. Reston Publishing Company, Inc., Reston, Virginia 22090. 1982 pp. 540, numerous illustrations and tables, Price R29.95 (ISBN 0-8350-0216-1)

As clearly stated in the first edition, this book remains a basic work, designed to reveal to the reader the beginnings of the enormous fields of anatomy and physiology of the domestic animal. It was not intended to be adequate for the student studying in one of the professional medical fields. It caters very sufficiently for the reader who requires a deeper knowledge of these 2 subjects than is provided by survey courses of the structure and function of domestic animals. There are considerable quantities of more advanced texts available to take the student deeper into these subjects.

By dealing with the animal body systematically, there is a good intergration of structure and function. Certain areas are dealt with more superficially than others, for example the endocrinology, but the student should read elsewhere to cover these areas more fully. The horse is used as the basic organism of study, and other animals are studied comparatively.

Once again, the domestic animal is studied in the variety of fields, namely macroscopic anatomy, histology, embriology, physiology and biochemistry. Emphasis is placed on only those aspects which are of meaningful value to the student. As with the first edition, the comment can be made that the detail extends beyond the bounds of the interested layman or superficial student.

In both the anatomical and physiological spheres, the author has again used terminology which is out-dated. The continued use of anatomical terms derived from human anatomy causes this book to have a lower status in the eyes of the antomist. All anatomy books of merit should use the formal Nomina Anatomica Veterinaria terminology or good translations thereof. No attempt has been made by the author to expand this book by adding recently discovered knowledge concerning the control of metabolism and facts about DNA and RNA, which were sad emissions also in the first edition. Errors of a minor nature can be found in the sections of anatomy, physiology and intermediary metabolism. The ageing of domestic animals is extensive and useful. The final chapter dealing with the anatomy and physiology of the domestic fowl is of value to students at any level.

This book can certainly be recommended for the reading audience to which it is directed, namely the non-professionally orientated student of layman. It is regrettable that the changes made between the first and second editions of this book have been limited, at the author's own admission, to alterations in grammar, syntax and the use of italics.

G.J. Louw

CASE REPORT **GEVALVERSLAG**

GASTRIC IMPACTION IN CAPTIVE CROCODILES (*CROCODILUS NILOTICUS*)

E.P.S. ROGERS* and R.S. WINDSOR*

ABSTRACT: Rogers E.P.S.; Windsor R.S. **Gastric impaction in captive crocodiles (*Crocodilus niloticus*).** *Journal of the South African Veterinary Association* (1982) 53 No. 4, 254 (En) Veterinary Diagnostic Laboratory, Private Bag 0035, Gaborone, Republic of Botswana.

Five crocodiles kept in captivity for more than 4 years died within a period of 4 days. A post mortem examination of one showed that it had died from gastric impaction which had resulted from the feeding of guinea pigs. It was thought that the high ratio of hair to flesh in guinea pigs together with daily feeding cause the impaction.

Key Words: Crocodiles, gastric impaction, guinea pigs.

Within a period of 4 days, all 5 crocodiles kept in the National Museum in Gaborone, died. These crocodiles were captured in the Okavango Delta more than 4 years ago for the National Museum and at the time of death varied in age from 5-11 years.

In the museum grounds they were kept in 2 enclosures, the largest crocodile being in a pond measuring approximately 20 m by 5 m and the others in a circular pond measuring 6 m in diameter. Both ponds contained water to a depth of 400 mm and had reeds growing in them. There was a shaded area surrounding the ponds.

Until a week before deaths commenced, these crocodiles were fed meat and chickens carcases which had been condemned as unfit for human consumption and an occasional guinea pig. They were then given a diet consisting entirely of guinea pigs, about 50 being fed over a period of 3 or 4 days. The animals became listless and refused food and 4 days from the onset of clinical signs, all were dead. No examination was carried out on the first 4 dead crocodiles.

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The last crocodile to die which was also the oldest and largest was brought to the Veterinary Diagnostic Laboratory in Gaborone for post mortem examination. It weighed approximately 60 kg and was just over 2 m in length.

The cause of death was gastric impaction, the impacted mass consisted entirely of matted guinea pig hairs, weighed 2,1 kg and resembled a large hair-ball commonly seen in calves. The impacted mass was completely immovable. Since all had received the same food we consider it likely that impaction was the cause of death in the other 4 crocodiles.

It is a matter of conjecture as to the reason for this impaction, but we feel that either or both of the following factors could be involved:

1. Although crocodiles in the Okavango Delta are almost entirely fish eaters, (mainly catfish Barbel *Claris* spp.) these particular ones had lived quite happily on their mixed diet for 4 years, and it was not until they were fed solely on guinea pigs that trouble occurred. Possibly the high portion of hair to flesh was a factor in the impaction.
2. The crocodiles were being fed the guinea pigs daily whereas the normal feeding frequency is once every 2 weeks or so.

We would be interested to know if anyone else has had a similar experience, and would recommend that guinea pigs not be used in the diet of captive crocodiles.

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CASE REPORT GEVALVERSLAG

ANAESTHETIZATION OF A CAPE FUR SEAL (*ARCTOCEPHALUS PUSILLUS*) FOR THE TREATMENT OF A CHRONIC EYE INFECTION AND AMPUTATION OF A METATARSAL BONE

G.D. THURMAN*, S.J.T. DOWNES** and S. BARROW**

ABSTRACT: Thurman G.D.; Downes S.J.T.; Barrow, S. **Anaesthetization of a Cape fur seal (*Arctocephalus pusillus*) for the treatment of a chronic eye infection and amputation of a metatarsal bone.** *Journal of the South African Veterinary Association* (1982) **53** No. 4, 255-257 (En) South African Association for Marine Biological Research, Oceanographic Research Institute, P.O. Box 10712, 4056 Marine Parade, Durban, Republic of South Africa.

Tranquilization using 3 mg/kg of ketamine by intramuscular injection followed by inhalation anaesthesia using halothane was performed on an adult Cape fur seal in order to perform a metatarsal amputation and opthalmic examination. Ketamine was found to have little effect at the dosage used while halothane proved to be a rapid induction agent providing a safe, continued level of surgical anaesthesia. Variations in cardiac rate and body temperataure were recorded during anaesthasia and blood was sampled for haematology.

Key Words: Cape fur seal, ketamine, halothane, metatarsal amputation, opthalmic examination.

CASE HISTORY

A female Cape fur seal *(Arctocephalus pusillus),* weighing approximately 50 kg, suffered from a chronically recurring right eye infection for a few years. Although the infection healed after protracted therapy using chloramphenicol (Lennacol capsules, Boehringer Mannheim) and a proteolytic enzyme (Tromasin SA, Warner) systematically, chloramphenicol (Panicetine eye drops, Panvet), atropine (Minims, S & N) and cod liver oil – as a lubricant – topically, the condition returned 2 weeks after cessation of treatment. As this recurrence might have been due to the presence of an irritant under the conjunctiva, it was necessary to examine the eye more closely. To do this, heavy tranquilization or immobilization was necessary as the seal, although trained for use in shows, was inclined to be aggressive when handled.

As no facilities for immobilization were available at that time, tranquilization had to be the method of choice. Literature studies revealed ketamine to be the drug most favoured as a tranquilizer for seals, but authors differed in their opinions as to the therapeutic and toxic dosage rates [1 2 5]. It was apparent that the reaction to the drug was unpredictable and often life-threatening.

The dilemma of whether or not to use ketamine was finalized when the seal was involved in a fight with a large male seal. During the fight, which lasted only a few minutes, the male seal bit off the first digit on the right hind flipper of the female seal.

On examination, it was found that approximately 100 mm had been removed, and that a portion of the metatarsal bone protruded about 15 mm from the wound opening. In order to prevent an ascending infection it was considered advisable to surgically amputate the protruding section of bone and suture the overlapping skin edges together. At the same time, the infection eye could be examined and, if necessary, treated whilst the animal was anaesthetized.

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MATERIALS AND METHODS

Pre-operative preparation

In preparation for the operation, the seal was starved for 18 hours before the commencement of the operation.

Tranquilization

The seal was initially restrained by placing a large hoop net over her head and then injected with 150 mg ketamine (Ketalar, Parke-Davis) intramuscularly into the gluteal muscles. As dosage rates of 5-6 mg/kg body mass have resulted in the death of seals [1 2 5] the dosage was calculated at 3 mg/kg body mass.

Pre-anaesthetic medication

The seal was premedicated with 1 mg atropine sulphate intramuscularly.

Anaesthesia

Once sufficiently sedated, a cone shaped mask was placed over the muzzle and connected to a portable closed-circuit anaesthetic unit (comprising a 1 ℓ latex reservoir-bag, soda-lime canister and oxygen and fluotec apparatus). The inhalant anaesthetic used was halothane (fluothane, I.C.I.). Once surgical anaesthesia had almost been attained, the mask was removed and the seal intubated using a No. 7,0 endotracheal tube with an inflatable cuff. The endotrachael tube was then connected to the inhalant anaesthetic machine. Surgical anaesthesia was taken as that period characterized by the absence of a papabral reflex, lack of tone in the masticatory muscles, cessation of "trembling" in the hind flippers and regulation of rhythmic deep and slow respiration[4].

Body temperature and cardiac rate

While under anaesthesia, the seal's body temperature

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was monitored using a rectal thermoprobe connected to a battery-powered electronic temperature recorder, graduated in 0,5 °C. If hyperthermia occurred, iced water was on hand to sponge the pectoral flippers to promote heat loss. The cardiac rate was monitored throughout anaesthesia at 5-minute intervals by auscultation.

PROCEDURES

Metatarsal amputation

Once the animal was sufficiently anaesthetized, the wound was shaved free of hair and scrubbed using hexachloraphene liquid soap. It was then disinfected using the iodine spray (Vetedine, Vetlab). The area was draped using sterile cloths leaving only the severed stump exposed. A 20 mm longitudinal incision was made parallel to the metatarsal bone. The surrounding tissues were separated from the bone using blunt dissection. The metatarsal bone was amputated approximately 30 mm from the protruding fracture. The muscle layers were sutured over the end of the bone using No. 2 sterile chromic catgut (Ethicon, W.725). The skin layers were sutured together using the same. This was done to prevent rehandling the seal in order to remove the skin sutures. An antibiotic powder (Cicatrin V, Welcome) was placed in the wound prior to closure.

Opthalmic examination

The right eye was examined using a direct opthalmoscope. Apart from corneal scarring from previous ulceration, with a surrounding zone of nebulous keratitis, a haemorrhagic conjunctivitis was found. The cause of this is uncertain as no foreign or traumatic object could be found but may have resulted from the corneal scarring. No corneal ulceration was present was as there was no 2 % fluorescien dye uptake when administered topically onto the cornea. A subconjunctival injection of cortisone (Betsolan V Sol inject., Glaxo-Milvet) was administered into the affected eye. The cornea was flooded topically with an antibiotic eye ointment (Neosporin, Wellcome) and the superior and inferior conjunctive sutured together using No. 2 sterile chromic catgut (Ethicon, W. 725).

Post-operative procedures

In order to prevent post-operative infection of the wound, 2 ml long acting penicillin (Compropen, Glaxo) was administered intramuscularly into the gluteal muscles. Approximately 5 ml blood was collected from the iliac artery using a 20 gauge hypodermic needle and syringe. The blood was placed into a vacutainer containing EDTA for analysis (see monitoring data results).

On completion of surgery the seal was placed in a padded recovery pen and the endotracheal tube was removed. She remained in the recumbent position for a short while, whereupon she began to tremble lightly on recovery. The seal was fully awake and aware of her surroundings 45 minutes after termination of halothane administration. She gradully became more secure and steady on her flippers and attempted to escape the pen.

Approximately 1 h 40 min after surgery, the seal was released back into the water where she swam immediately without aid or difficulty.

MONITORING DATA

Cardiac rate

During anaesthesia and post-anaesthesia, the heart rate remained constant at 82-88 beats per minute

Respiratory rate

During induction the seal breathed between 10-15 times per minute. During surgical anaesthesia she breathed 6-8 times per minute. The rebreathing bag was adequate for her tidal volume which appeared to be just under 1 ℓ of air.

Body temperature

Her rectal temperature recorded at 5 minutes intervals is graphically represented in Fig. 1.

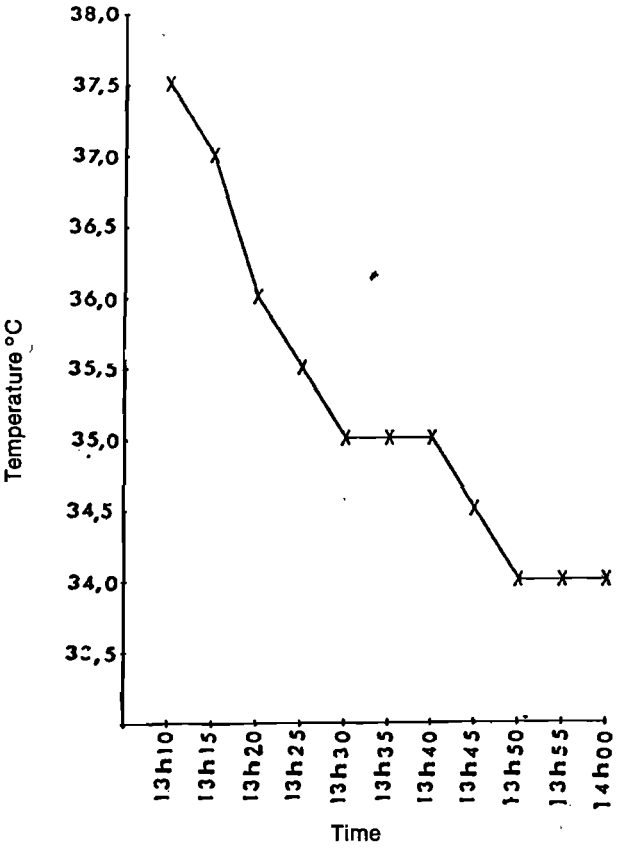

Fig. 1: Graphic representation to the body temperature decline observed during anaesthesia and surgery in a Cape Fur Seal.

The pectoral flippers were initially cooled with iced water during anesthesia, but when the temperature dropped below 36,0 °C (which is regarded as the lower normal limit[3]), this was stopped. The ambient temperature was 19,5 °C. The thermoprobe was removed at 14h00 as she showed active signs of recovery from anaesthesia.

Haematology

The results of the blood tests were as follows:

B-WBC x $10^9/\ell$	9,3
B-RBC x $10^{12}/\ell$	4,04
B-Hb g/ℓ	136

B-Ht	0,39
E-MCV fℓ	95,0
E-MCH pg	33,4
E MCHC g/dℓ	35,0
Lkc-Neutrophils %	70
Lkc-Lymhpcytes %	30
Lkc-Monocytes %	None
Lkc-Eosinophils %	None
B-Platelets x $10^9/\ell$	± 140

No normal values exist at present for this species. It does appear, however, that there is an elevation in the total white blood cell count and an increase in the circulating neutrophils indicating the possibility of infection as well as trauma in the wound.

DISCUSSION

No problems were encountered during anaesthesia or surgery. It would, however, be advisable to utilize a physical restraining apparatus in the form of a body clamp or crush during initial tranquilization and induction so as to avoid the handling risk. The dosage of ketamine used was insufficient to cause immobilization and the animal was still aggressive when handled. A higher dosage rate of 4-5 mg/kg of ketamine body mass following an accurate determination of the mass of the animal may produce better results.

The catgut sutures in the skin dissolved within 4-5 days leaving a few areas open and raw. Due to the chlorination of the water however, no infection resulted and the wound has since healed completely. The seal regained full use of the flipper after one week post-surgery and vision had significantly improved 2 weeks post-surgery to re-include her in shows.

ACKNOWLEDGEMENTS

We would like to express our gratitude to Dr Graham Ross for his invaluable advice on the use of ketamine in pinnipeds. Thanks are also extended to Dr Brian Wessels for the kind loan of his anaesthetic machine, and to Drs Carey and Vlok for the anaesthetic mask used during induction.

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ABSTRACT: Horak, I.G., 1982. **Parasites of domestic and wild animals in South Africa, XIV. The seasonal prevalence of *Rhipicephalus sanguineus* and *Ctenocephalides spp.* on kennelled dogs in Pretoria North.** *Onderstepoort Journal of Veterinary Research,* **49**, 63-68 (1982).

The seasonal prevalence of *Rhipicephalus sanguineus* and *Ctenocephalides* spp. on kennelled dogs in Pretoria North was determined by the regular examination of 3, initially, and later 2 dogs from March 1975 – January 1977.

Once the ticks had become established in the kennel peak burdens of immature ticks (larvae plus nymphae) were present on the dogs during early summer 1975 and from midsummer – late summer 1976 and early midsummer 1976/77. Peak numbers of adult ticks were present in midsummer 1975/6, from late summer-autumn and during early spring 1976 and during midsummer 1976/77. Few adults and even fewer immature ticks were present on the dogs during winter 1976 and the infestation overwintered in the pens as engorged nymphae.

The flea population took 10 months to become well established. Thereafter, the periods later summer-autum 1976 and early midsummer 1976/77 (when the survey stopped) were the most favourable and winterspring 1976 the least favourable for adult fleas.

The immature ticks preferred the sides and bellies, adult ticks the necks, and fleas the bellies of the dogs.

ABSTRACT: Reinecke, R.K., Bruckner, Christel, De Villiers, I.L., 1982. **Studies on *Haemonchus contortus.* VII. The effect of treatment of *Trichostrongylus axei* prior to challenge with *H. contortus.*** *Onderstepoort Journal of Veterinary Research,* **49**, 69 (1982).

Four-month-old worm-free Merino lambs were dosed with 20 000 infective larvae of *Trichostrongylus axei* on Day 0 and again on Day + 14. On Day + 83 they were treated with mebendazole at 15 mg/kg. All lambs in this group and a further group of 11 worm-free Merino control lambs were challenged with 50 000 infective larvae of *Haemonchus contortus* dosed from Day + 90-Day + 92. At necropsy 27 and 28 days later there was no significant difference between the worm burdens of the 2 groups. *T. axei* must be present in the abonasum to protect sheep from challenge with *H. contortus.*

CASE REPORT **GEVALVERSLAG**

OORGANGSEPTITEELKARSINOOM VAN DIE URIENBLAAS VAN 'N HOND

J.S.J. ODENDAAL* en STELLA S. BASTIANELLO**

ABSTRACT: Odendaal J.S.J.; Bastianello S. **Transitional cell carcinoma of the urinary bladder of a dog.** *Journal of the South African Veterinary Association* (1982) 53 No. 4 258-260 (Afr) 152 Benade Drive, Fichardt Park, 9322 Bloemfontein, Republic of South Africa.

The occurrence of a urinary bladder transitional cell carcinoma in a dog is reported. The spontaneous occurence of such tumours is rare in dogs and in this case no aetiological or predisposing factors for the development of this tumour could be determined. Histologically, the tumour was classified as a papillary, invasive transitional cell carcinoma.

Key words: Transitional cell carcinoma, urinary bladder, dog.

INLEIDING

Alhoewel oorgansepiteelkarsinome bekende gewasse aan die urienblaas is, kom hulle selde in honde voor. Volgens Theiler & Madewell[4] verteenwoordig blaasgewasse slegs 0,5 % van die totale aantal gewasse in honde. Ses-en-sewentig persent van die 114 blaasgewasse in hulle studie was egter oorgangsepiteelkarsinome[4]. Hierteenoor het Osborne[2] aangetoon dat net 36 % uit 130 blaasgewasse in honde wel oorgangsepiteelkarsinome was.

Daar blyk geen ras of geslagsprediléksie vir hierdie gewas in honde te wees nie. In party opnames was vroulike diere meer aangetas[1], terwyl in ander, die insidens òf hoër was in manlike diere òf geen geslagsvoorkeur bespeur is nie[4]. Oorgangsepiteelgewasse van die blaas kom meesal voor in honde met 'n gemiddelde ouderdom van 9,1 jaar[4].

Blaasgewasse kom meer algemeen voor in die mens as in diere[4]. In die mens word dié gewasse assosieer met persone wat met anilienkleurselle werk, strawwe sigaretrokers of mense wat schistosomiase onderlede het[4]. Die karsinogeniese stowwe in anilienkleursels blyk die afbreukprodukte α en β-naftielamien en -bensidien, en 4-aminofideniel te wees aangesien hierdie 3 stowwe aangetref word in die bloed en uriene van die werkers[4]. Dit is ook bekend dat die afbreukprodukte van hierdie anilienkleursels onder eksperimentele toestande [1 2] ook in honde oorgansepiteelkarsinome van die blaas kan verwek. Uit 34 honde waaraan 2-naftielamien per os toegedien is, het 24 oorgangsepiteelkarsinoom van die blaas ontwikkel[1]. Verder blyk dit dat blaaskanker meer algemeen voorkom in mense in stedelike gebiede as gevolg van lugbesoedeling[4].

GESKIEDENIS

'n Vyfjaar oue Bull Mastiffkruis-reun, afkomstif van 'n plaas, is ingebring met die klagte dat die hond oor 'n tydperk van omtrent 3 maande bloedgekleurde uriene passeer het. Later het stolsels bloed in die uriene voorgekom. Die frekwensie van urinering het vermeerder en die hond het later die mikturisiehouding van 'n teef ingeneem. Die hond het by tye ook pyn begin toon veral as daar aan die buik geraak is. Die hond was in goeie kondisie en het behalwe vir die bogenoemde klagtes, 'n aktiewe lewe gelei en normaal geëet en gedrink. Die diëet het hoofsaaklik bestaan uit 'n kommersiële droë hondekos (Dogmore, Glenmore Products Ltd). Die hond was nie voorheen vir enige blaasprobleme behandel nie en geen geskiedenis van moontlike kontak met karsinogeniese stowwe kon verkry word nie.

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KLINIESE ONDERSOEK

Tydens die kliniese ondersoek is al die klagtes van die eienaar bevestig. Alhoewel die buik met palpasie gevoelig was kon die vergrote blaas nogtans duidelike gevoel word. Hematurie is bevestig deur kateterisasie. Die pasiënt was nie koorsig nie. 'n Voorlopige diagnose van blaasstene is gemaak.

Laparotomie is uitgevoer om die blaas deegliker te ondersoek en vir die nodige chirurgie. Die blaaswand was so verdik dat palpasie vir blaasstene nie moontlik was nie. D.m.v. sistomie is bepaal dat die blaas feitlik toegegroei was weens uitgroeisels vanaf die mukosa.

Omrede die toestand klinies geoordeel, prakties onbehandelbaar was, is besluit om die pasiënt af te maak.

MAKROSKOPIESE PATOLOGIE

Patologiese letsels is slegs in die blaas waargeneem. Die bloedvate buite op die verdikte blaaswand was opvallend. Die slymvlies was ewe eens erg verdik deur die teenwoordigheid van onreëlmatige knopagtige uitgroeisels. Die mukosa het 'n bloederige voorkoms gehad as gevolg van klein ulkusse op die oppervlakte van die uitgroeisels. Die uitgroeisels was so oorvloedig en ekstensief dat die blaas se lumen feitlik uitgewis was (Fig. 1). Die aangetaste blaaswand was ca 40 mm dik. Die buite mate van die blaas was 100 by 80 mm. Daar was geen metastase na ander organe nie.

MIKROSKOPIESE PATALOGIE

Na fiksering in 10 % gebufferde formalien is toepaslike weefselblokkies in paraffienwas ingebed, 3-5 μm in dikte gesny en daarna gekleur met hematoksilien en eosien (HE) vir ligmikroskopiese ondersoek.

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Die hele mukosa en deel van die submukosa was vervang óf deur proliferatiewe papillêre massas van gewasselle wat om 'n sentrale bindweefselkoord groei, óf deur wye plate van selle sonder 'n sigbare sentrale bindweefselkoord. 'n Fyn netwerk van bindweefselvesels, wat die gewas ondersteun, het deur die hele gewas gestrek. As gevolg van ekstensiewe infiltrasie van gewasselle, was die muscularis mucosa nie sigbaar nie. Infiltrerende selle het voorgekom in die submukosa maar nie in die tunica muscularis nie.

Die gewas het meesal voorgekom as plate van selle wat dig teen mekaar lê (Fig. 2). Die vesikulêre, ronde tot ovale kerne van die selle het 1 of 2 onduidelike nukleoli getoon, en 'n eosinofiliese sitoplasma. Geen intersellulêre brûe kon waargeneem word nie. Verder was daar ook foki van selle met gevakuoleerde sitoplasma verspreid deur die gewas (Fig. 3). Hierdie vakuoles was meesal groot en het feitlik die hele sitoplasma opgeneem. Die kerne van hierdie selle was sentraal of na die kant van die selle verplaas en as 'n reël ook piknoties. Homogene helderpienk globules van variërende groote of fyn fibrillêre materiaal was soms teenwoordig in die vakuoles (Fig. 4).

Groepies limfosiete het verspreid deur die hele gewas (Fig. 3) voorgekom maar veral langs die basis en net onderkant die oppervlakte van die gewas. Laasgenoemde was hiperemies en baie van die selle het in die lumen afgeskilfer. Foki van koagulatiewe nekrose asook enkele nekrotiese selle het deur die gewas voorgekom.

Kleiner groepe van selle wat apart van die hoof gewas was, is in die submukosa teenwoordig.

BESPREKING

Hierdie is die eerste keer dat 'n oorgangsepiteelkarsinoom van die blaas in hierdie praktyk voorkom en is ook, sover die outeurs kon vasstel, die eerste aanmelding van so 'n gewas in die Republiek van Suid-Afrika.

Oorgangsepiteelkarsonome kan wissel in makroskopiese voorkoms van papillêr tot vratagtig, of nodulêr[1,2] en mag enkel of meervoudig wees[2]. Die gewas in die hond was ekstensief en nodulêr in voorkoms.

Histopatologies kon hierdie gewas beskryf word as 'n papillêre, infiltrerende oorgangsepiteelkarsinoom van die blaas. Volgens Moulton[2], gradeer Jewett die blaasgewas van die mens in 5 grade, d.w.s. zero – geen infiltrasie, A – infiltrasie van mukosa en submukosa, B – infiltrasie tot in die muskularis, C – infiltrasie van die peritoneum en vet om die blaas en D – metastase na

Fig. 1: Dwarssnit van die blaas met knopagtige, bloederige uitgroeisels op die mukosa. Die uitgroeisels wis die lumen omtrent heeltemal uit.

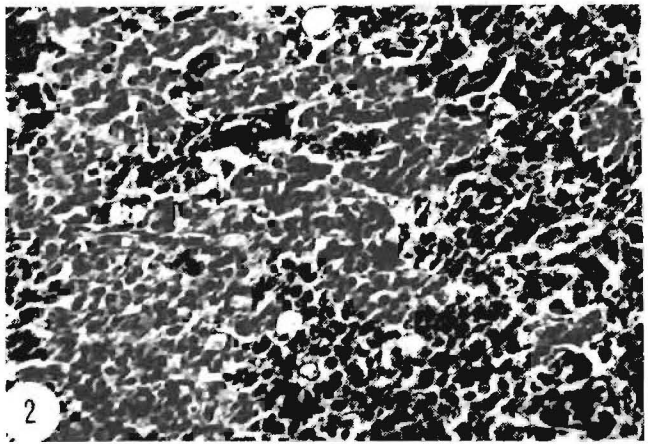

Fig. 2: Plate van gewasselle wat redelik dig teen mekaar lê. HE X 200

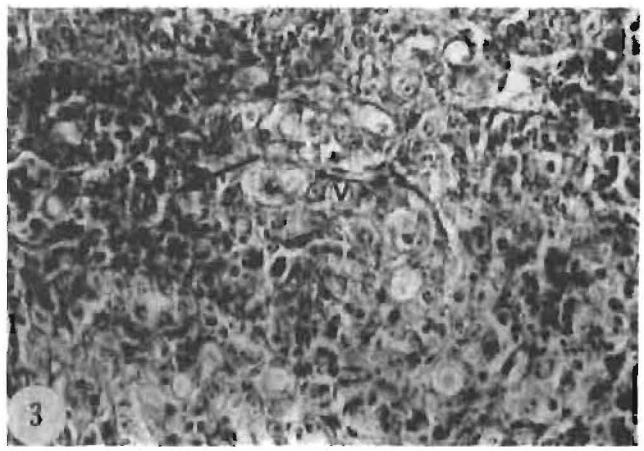

Fig. 3: Gewas met fokus van gevakuoleerde selle (V) asook 'n groep inflammatoriese limfosiete (pyl). HE X 200

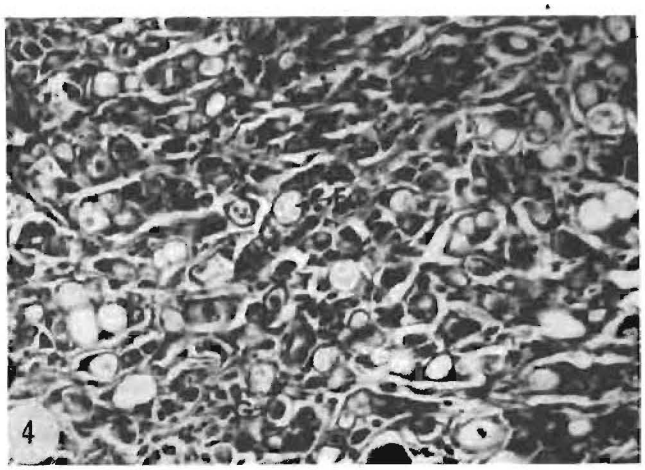

Fig. 4: Groot gevakuoleerde selle. Van die vakuoles bevat globules (G) of fibrillêre materiaal (F). HE X 500

ander organe. Die oorgangsepiteelkarsinoom in dié hond het slegs die mukosa en submukosa van die blaaswand geïnfiltreer maar het nie gemetastaseer nie. Volgens bogenoemde graderingssisteem sal dit dus gepas wees om hierdie gewas onder graad A te plaas. Indien 'n oorgangsepiteelkarsonoom metastaseer kan dit wydverspreid in verskeie organe voorkom maar volgens Schmidt[3] metastaseer oorgangsepiteelkarsinome gewoonlik eers laat gedurende die siekteverloop.

Vakuolêre veranderings soos dié teenwoordig in die gewas onder bespreking, is waarskynlik 'n aanduiding van preneoplastiese of neoplastiese plaveiselle in blaasgewasse[2]. Aangesien daar nêrens in hierdie oorgangsepiteelkarsinoom enige keratien of intersellulêre brûe gesien kon word nie, is dit onwaarskynlik dat die vakuoles in hierdie gewas binne-in plaveiselle was. 'n Elektronmikroskopiese ondersoek om die afwesigheid van desmosome te demonstreer sou egter nodig wees om te verseker dat die gevakuoleerde selle nie plaveiselle was nie. Baie van die gevakuoleerde gewasselle het óf geen kern bevat óf net 'n piknotiese kern wat daarop mag dui dat hierdie vakuolêre veranderings 'n litiese tipe van nekrose of vakuolêre degenerasie verteenwoordig. Die foki van nekrose en enkele nekrotiese selle dui op vinnige groei van die gewas en hierdie vakuolêre veranderinge kon dus as gevolg van onvoldoende bloedtoevoer aan die gewas ontstaan het. Wat presies die eosinofiliese globules en fyn fibrillêre materiaal binne-in die gevakuoleerde selle verteenwoordig, kon nie vasgestel word nie. Hulle mag oorblyfsels van dooie sitoplasmiese organelle of koaguleerde proteïne wees en dus ook 'n degeneratiewe proses verteenwoordig.

Die moontlike oorsake van die gewas in hierdie hond kon nie vasgestel word nie. Daar was geen aanduidings van die teenwoordigheid van anilien in die diët of omgewing van die hond nie. Die oorsake van spontane blaasgewasse in die hond is onbekend[1]. Die voorkoms van blaaskanker neem toe min mense wat in stede woon, waarskynlik te wyte aan industriële besoedeling van die omgewing[4]. Dit kan moontlik ook by honde 'n rol speel, maar aangesien dié hond van 'n plaas afkomstig was, is dit in hierdie geval onwaarskynlik dat dit 'n rol gespeel het.

BEDANKINGS

Die outeure wil graag die volgende bedank: Mnr J.J. Pulsen en die personeel van die Seksie Fotografie, Navorsingsinstituut vir Veeartsenykunde Onderstepoort vir die druk van die mikrofotos; die laboratoriumpersoneel van die Seksie Patologie vir die bereiding van die histologiese snitte en Mev A.M. Coetzer vir die tikwerk.

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CASE REPORT **GEVALVERSLAG**

DIAGNOSIS OF EQUINE ENDOMETRIAL CANDIDIASIS BY DIRECT SMEAR AND SUCCESSFUL TREATMENT WITH AMPHOTERICIN B AND OXYTETRACYCLINE

D. BROOK*

ABSTRACT: Brook D. **Diagnosis of equine endometrial candidiasis by direct smear and successful treatment with amphotericin B and oxytetracycline.** *Journal of the South African Veterinary Association* (1982) **53** No. 4, 261-263 (En), 20515 Covina Hills Road, Covina, Ca. 91724, U.S.A.

A case of endometritis, caused by *Candida albicans,* was diagnosed by uterine culture and direct smear. Treatment with amphotercin B and oxytetracycline was successful and the mare subsequently produced a live foal.

Key words: candidiasis, equine, diagnosis, treatment.

INTRODUCTION

Yeasts can often be recovered from mucous membranes, of which they may constitute part of a normal flora[3]. Kenney has shown that *Candida albicans* can be present in the reproductive tract of the mare without producing clinical or histopathological signs, the organism simply causing superficial contamination. He states that only on rare occasions does it invade the mucous membrane and generate a host reaction[7]. Work on endometrial cytology in the mare has shown that fungi can be isolated from the uterus without there being evidence on endometrial smear examination of inflammation, as manifested by the presence of neutrophils[8].

Other reports have shown that yeasts and fungi can be present in cases of clinically detectable endometritis[1 4 9]. These cases are said to occur usually after prolonged antibiotic therapy [4 5 7 10 12].

Various treatments have been proposed and used including euflavin[4], nystatin[1 12], iodine [1 12] and amphotericin B[9]. The case reported here describes the use of amphoteracin B in a case of endometritis from which *C. albicans* was isolated.

HISTORY

The mare was 12 years old and had been barren for one year. During the 1979 breeding season no pathogenic organisms were isolated from an initial swab taken from the uterus but *C. albicans* was cultured from a subsequent swab. At that stage she was treated with a variety of antibiotics administered by both the intra-uterine and parenteral routes as well as by irrigation of the uterus with iodine. The mare did not conceive and was presented to me for examination in August 1980. At that time she had a moderately heavy vaginal discharge and culture of her uterine contents produced a pure growth of *C. albicans.* An endometrial smear, stained by the Harleco "diff-quick" method, showed the presence of numerous neutrophils and fungal element (Fig. 1)

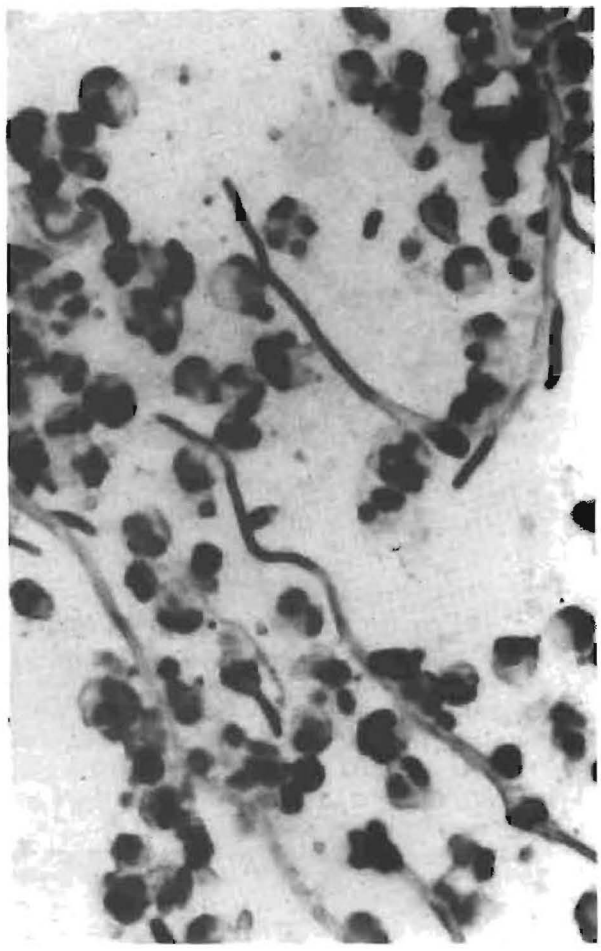

Fig. 1: Endometrial smear showing neutrophils and fungal elements. X400

TREATMENT

The mare was treated once a day for 10 days with a intra-uterine infusion of a solution containing 250 mg amphotericin B and 500 mg oxytetracycline-HCl in 250 ml normal saline solution. This solution was made by grinding up the required number of Vagmycin Vaginal Tablets (Squibb) which contained both these drugs and mixing the resultant powder with the saline. The solution was then infused into the uterus. An endometrial smear taken 4 days after the last treatment showed fewer neutrophils and no fungal elements (Fig. 2). Oestrus was subsequently induced by means of an intra-muscular injection of 10 mg dinoprost tromethamine (Lutalyse, Upjohn). Whilst in oestrus a

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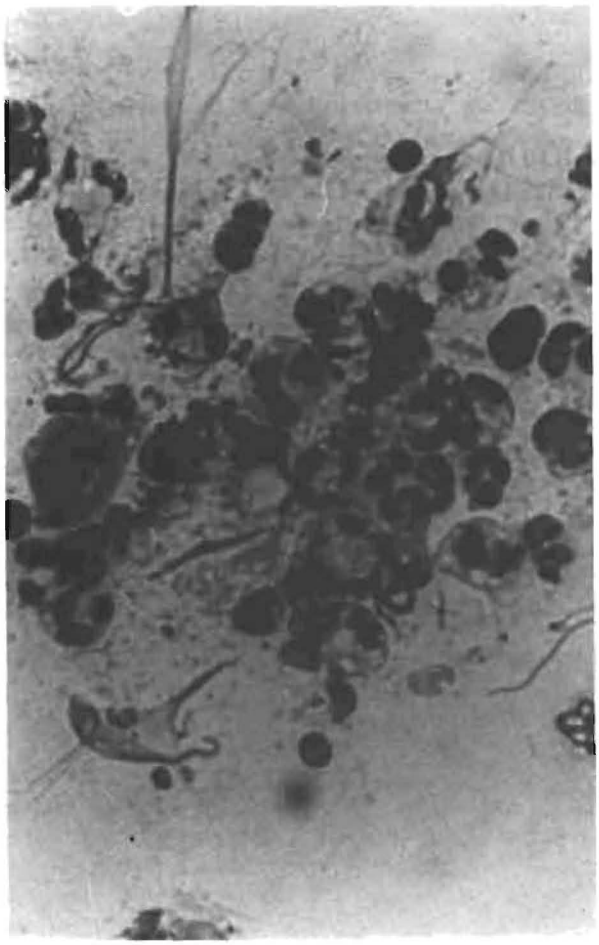

Fig. 2: Endometrial smear showing neutrophils. (x400)

culture of the uterine contents revealed no pathogens and an endometrial smear showed no neutrophils. The mare was not mated during this oestrus period nor in a subsequent induced one. Oestrus cycles thereafter occured normally, the first of which occured 14 days after the end of the last induced one. In this oestrus period endometrial cytology and bacterial culture were again both negative, and the mare was covered. She was subsequently proved to be in foal and later produced a live foal.

DISCUSSION

It is generally agreed that infections by *C. albicans* follow prolonged antibiotic therapy [4 5 10] and indeed the case described had been bombarded with a plethora of antibiotics and antiseptics during the previous breeding season. It should be emphasised that the isolation of a fungus or yeast from the reproductive tract of a mare is not sufficient evidence to incriminate the organism as the cause of disease. Spontaneous disappearance of *Candida*, ie without treatment, has been known to occur[7]. In another study 6 out of 111 swabs showed evidence of fungal presence on culture without evidence of endometritis[8]. Endometritis was deemed to be absent in these 6 cases because of absence of neutrophils on endometrial smear. This author did not state how many mares were involved in his study.

It has been shown that the presence of neutrophils in an endometrial smear is good supportive evidence for the existence of an endometritis[2 8 11]. The presence of fungal elements and neutrophils in the endometrial smear from the mare under review, coupled with the culture of a pure culture of *C. albicans,* was considered to be sufficient evidence to incriminate this organism as the cause of the problem.

Unfortunately, sensitivity testing was not carried out in this case but previously published work [1 4 9] has indicated that either nystatin or amphotericin B is the drug of choice. One report showed an apparent failure of nystatin to cure a case of pyometra caused by *C. rugosa*[1]. For this reason amphotericin B was used in this case and it was considered that the inclusion of tetracycline would be beneficial because of its broad spectrum of activity.

This case report demonstrates the usefulness of endometrial smears and their use together with bacterial and fungal culture of the uterine specimens is strongly advocated.

It should be pointed out that infection of the uterus by *C. albicans* has been known to occur without detectable evidence of the organism on endometrial smear. Neutrophils were, however, present[2].

Because of the beneficial effects of oestrogens on the process of phagocytosis[6] it was decided to bring the mare into heat twice before covering her, thereby allowing more time for the endometrium to return to normal.

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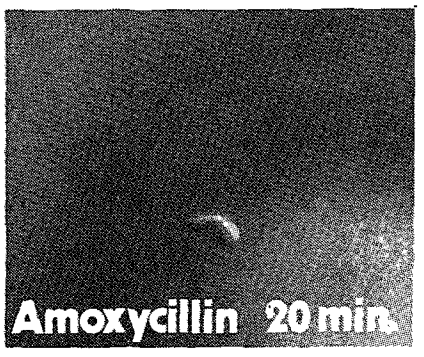

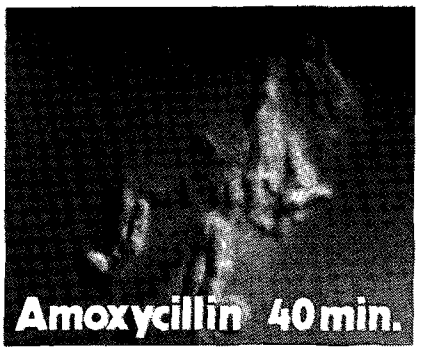

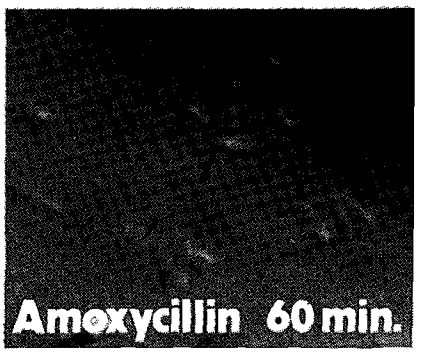

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SHORT COMMUNICATION **KORT BERIG**

THE MINIMAL EFFECTIVE DOSE OF ACTIVATED CHARCOAL IN THE TREATMENT OF SHEEP POISONED WITH THE CARDIAC GLYCOSIDE CONTAINING PLANT *MORAEA POLYSTACHYA* (THUNB) KER-GAWL

J.P.J. JOUBERT* and R. ANITRA SCHULTZ*

ABSTRACT: Joubert J.P.J.; Schultz R. Anitra **The minimal effective dose of activated charcoal in the treatment of sheep poisoned with the cardiac glycoside containing plant *Morea polystachya* (Thunb) Ker-Gawl.** *Journal of the South African Veterinary Association* (1982) 53 No. 4, 265-266 (En) Section of Toxicology, Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa.

The approximate minimal effective dose of activated charcoal, given to sheep 12 h after the administration of a lethal dose of *Moreae polystachya*, was found to be 2 g/kg. Serum cation levels remained within normal limits throughout the experiment.

Key words: Minimal effective dose, activated charcoal, cardiac glycoside poisoning, sheep, *Moraea polystachya*.

INTRODUCTION

Activated charcoal, as well as activated charcoal plus potassium chloride, was found to be effective in the treatment of cardiac glycoside plant poisoning of ruminants[2 3]. As the dose of activated charcoal previously used (5g/kg)[2 3] was cumbersome to administer because of its large volume, this pilot trial was conducted to detemine an approximate minimal effective dose. To accomplish this, sheep experimentally poisoned with *Moraea polystachya*, were treated with various doses of activated charcoal.

MATERIALS AND METHODS

Flowering *M. polystachya* (tulp) was air-dried, milled and stored at 4 °C. Both the tulp and charcoal were suspended in water for dosing per stomach tube.

The 18 sheep used in this pilot trial were milk-toothed Merino wethers with live masses varying between 18 and 36 kg.

Sixteen sheep were dosed with 1,23 g/kg of tulp of these 14 were treated (6 or 12 h after dosing) with 0,5-4 g/kg activated charcoal, 1 pair per dosing level. Another 2 sheep, which had each received 2g/kg of tulp, were treated after 6 h with 1 g/kg of activated charcoal (Table 1).

Electrocardiagraphic recordings, clinical examination and blood chemical analyses were done periodically, each animal serving as its own control. The first blood specimens were comprehensively analysed, while subsequent specimens were only analysed for serum levels of Na, K, Ca and Mg[2 3 4].

RESULTS

The results are summarized in Table 1. Both control sheep (Sheep 1 and 2) died, while all of those treated at 6 h recovered but the 2 sheep which received 0,5 g/kg activated charcoal were severely distressed for a week and only fully recovered 9 days after treatment. One of the 4 sheep treated after 12 h died (Sheep 15).

Na, K Ca and Mg values varied slightly but did not reach abnormally high levels at any stage of the experiment[1 3].

No electrocardiographic changes were observed before treatment and in those which did occur after treatment the changes were mild.

However, the increased wave amplitudes, tachycardia, sinus arrhythmia and AV block, are typical of cardiac glycoside poisoning.

DISCUSSION

In this attempt to find a minimal effective dose of activated charcoal against tulp poisoning, the lethal dose of 1,25 g tulp/kg body mass and the 6 h time lapse before treatment were similar to the protocols of a previous experiment[3]. As these sheep did not appear to be clinically affected after 6 h (i.e. no electrocardiographic and minor clinical changes being observed), the dose of tulp in some and the time interval in others were increased.

The only treated sheep that died (Sheep 15) developed a severe watery diarrhoea before treatment. Since it had received 1 g/kg activated charcoal and those which recovered had received 2 g/kg at 12 h, it was concluded that the approximate minimal effective dose was 2 g/kg.

It must be pointed out, however, that if the diarrhoea and subsequent dehydration from tulp poisoning are treated with fluids and electrolytes in conjunction with charcoal, more affected animals might be saved. Futhermore, the results of this experiment can only be used as a guide for treating natural cases of poisoning, as the number of animals used per dose level was inadequate for statistical analysis. As in previous experiments, the serum K levels did not serve as an indication of the severity of cardiac glycoside poisoning[1 3].

ACKNOWLEDGEMENTS

We wish to thank the staff of the Toxicology Section for their invaluable assistance.

*Section of Toxicology, Veterinary Research Institute, 0110 Onderstepoort.

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Table 1: TREATMENT OF *MORAEA POLYSTACHYA* POISONING IN SHEEP WITH ACTIVATED CHARCOAL

Sheep	*M. polystachya* dose (g/kg)	Before treatment Clinical signs and ECG changes	Interval before treatment (h)	Activated charcoal (g/kg)	After treatment		Fate	Recovery time (d)
					Clinical signs	ECG changes		
1	1,25	Inappetence, ruminal stasis, severe diarrhoea, forced respiration	—	—	—	—	Died within 27 h 30 m	—
2	1,25	Ruminal stasis, severe diarrhoea, forced respiration restless	—	—	—	—	Died within 70 h	—
3	1,25	Inappetence, polypnoea (70/min)	6	4	Decreased ruminal movements, soft faeces	QRS wave amplitude abnormally high (3,1 mV at 8h)	Survived	3
4	1,25	Forced respiration, slightly bloated	6	4	Decreased ruminal movements, severe diarrhoea	Sinus arrhythmia (8 h)	Survived	3
5	1,25	Inappetence	6	3	Ruminal stasis, soft faeces	AV block did occur once	Survived	3
6	1,25	Inappetence	6	3	Ruminal stasis, severe diarrhoea	Sinus arrhythmia and QT segment depression (8 h)	Survived	3
7	1,25	No changes observed	6	2	Ruminal stasis, diarrhoea, polypnoea (110/min)	QRS wave: amplitude increased (2,9 mV), sinus arrhythmia and transient QRS alternation	Survived	2
8	1,25	No changes observed	6	2	Ruminal stasis, faeces soft, slightly forced respiration	n/a	Survived	2
9	1,25	No changes observed	6	1	Ruminal stasis, faeces soft, forced respiration	n/a	Survived	2
10	1,25	Severe sinus arrhythmia	6	1	Ruminal stasis, severe diarrhoea, polypnoea (120/min)	Severe sinus arrhythmia	Survived	2
11	1,25	Mild forced respiration, slightly bloated, apathy	6	0,5	Ruminal stasis, severe diarrhoea	n/a	Survived	9
12	1,25	Forced respiration, slightly bloated	6	0,5	Ruminal stasis, severe diarrhoea	n/a	Survived	9
13	2,0	Forced respiration, polypnoea, bloated	6	1	Severe diarrhoea, inappetence, apathy	n/a	Survived	5
14	2,0	Forced respiration, polypnoea, slightly bloated	6	1	Severe diarrhoea, inappetence, apathy	T wave amplitude decreased to 0,1 mV on Day 3	Survived	5
15	1,25	Ruminal stasis, severe diarrhoea, forced respiration	12	1	Severe diarrhoea, dehydrated	Tachycardia (250/min) T and P waves superimposed	Died within 36 h	—
16	1,25	Ruminal stasis, soft faeces, bloated	12	1	Soft faeces, arrhythmic heart beats	n/a	Survived	4
17	1,25	Decreased rumination, slightly forced respiration	12	2	Inappetence, soft faeces	n/a	Survived	4
18	1,25	Decreased rumination, polypnoea (68/min), forced respiration	12	2	Inappetence, soft faeces	n/a	Survived	5

n/a = nothing abnormal

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SHORT COMMUNICATION KORT BERIG

A MODIFIED SHORR'S STAIN: A PRACTICAL RAPID STAIN FOR CANINE VAGINAL CYTOLOGY

E.E. OETTLÉ* and ANNA A. WELDHAGEN**

ABSTRACT: Oettlé E.E.; Weldhagen A.A. **A modified Shorr's stain: a practical rapid stain for canine vaginal cytology.** *Journal of the South African Veterinary Association* (1982) 53 No. 44, 267-268 (En) Department of Genesiology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

A simple modification of Shorr's stain for vaginal cytology is described. The technique involved 8 steps and takes less than 10 minutes from time of making the smears until time of examination. The method is considered to be quick, easy, accurate and practical for use by the small animal practitioner.

Key words: Canine vaginal cytology, modified Shorr's stain.

INTRODUCTION

Canine vaginal cytology is a useful diagnostic aid for the clinician because it broadens the range of gynaecological services which may be offered to the client. The ability to monitor a bitch's oestrus cycle more accurately has several clinical advantages. Optimal time for insemination or service can be pinpointed, enhancing the chances of conception. Where natural service is used, more judicious use can be made of the male, thereby preventing overuse. Oestrus cycle abnormalities can be readily detected by sequential monitoring of cytological changes. The staining method[3] currently in use in our clinic, while accurate, is involved and time consuming. Some of the short methods described [1 4] have yielded poor results. Because of these factors, few practitioners tend to make use of this diagnostic tool and thus the development of a quick and accurate method for the staining of vaginal smears would provide a valuable aid for the clinician.

MATERIALS AND METHODS

Preparation of Stain

Haematoxylin (Lillie-Mayer)[2]:

Distilled water	350ml
Haematoxylin (Merck)	2,5 g
Ammonium alum	25 g
Glycerine	150 ml
Glacial acetic acid	10 ml
Sodium iodate	0,25 g

Heat water to 40° C, then add and dissolve consituents in sequence. The stain is ready for use after 24 hours.

Shorr's (Modified):

Biebrich Scarlet	0,5 g
Orange G	0,25 g
Fast Green FCF	0,75 g
Phosphotungstic acid	0,5 g
Phosphomolybdenic acid	0,5 g
Glacial acetic acid	1 ml
50% Alcohol to 100 ml	

Agitate till all constituents are dissolved. Store in a dark, tightly stoppered bottle.

Preparation of the smear

Impression smears of the vaginal mucous membrane (take care to avoid the skin) are transferred by means of a squash-smear technique to another slide in order to draw out the cells. The second slide is immediately fixed in 70 % ethyl alcohol for 3 minutes. It is important that the smear is not allowed to dry prior to fixing. If the smear do dry out, they should be placed for 5 minutes in distilled water and then fixed in the alcohol. Similarly, fixed smears may be dried if required for transport and need only be rehydrated in distilled water for 5 minutes before proceeding with staining. It is essential to take duplicates for control and comparison. Smears should be taken every second day for sufficient assessment of the stages of the oestrus cycle.

Staining procedure:

Wash in distilled water	10 seconds
Haematoxylin (Lillie-Mayer)	30 seconds
Wash in running tap water	2 minutes
Shorr's (Modified)	1 minute
Tap water rinse	5 seconds
Absolute alcohol rinse	5 seconds
Xylol rinse	5 seconds

Mount the smear, while still wet with xylol, in Canada Balsam or Eukitt and a cover slip. Stains should be replaced when they no longer give adequate staining of the cells. When not in use, the stains should be stored in sealed bottles to prevent evaporation and thus concentration of the stain. The alcohol and xylol should be replaced when they become cloudy.

RESULTS AND DISCUSSION

The reader is referred to Schutte[3] for a detailed interpretation of the significance of cells found in vaginal smears. With the method described, cornified cells stain orange and non-cornified cells stain turquoise. Nuclear detail is excellent and metoestral cells may be easily identified under low power. Some cells stain both orange and turquoise but there is usually no doubt as to the approximate Eosinophilic Index and thus the stage of the cycle may be easily assessed.

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If the smears are allowed to dry before fixing and not rehydrated as described, the non-cornified cells do not take up the turquoise stain, with the result that all cells stain orange and thus an 'Eosinophilic Index' of 100 % is obtained.

Despite precautions, there is often a degree of drying at the sides of the smear and hence it is always advisable to examine the middle of the smear microscopically, since the truest Eosinophilic Indices are obtained there. Time required for fixing, staining and mounting is short and the smears may be ready to be examined less than 10 minutes after being taken.

Shorr[4] described a one-step stain for vaginal cytology but this has given repeatedly unsatisfactory results in our laboratory. This technique has been modified by including a naematoxylin stain to enhance nuclear detail and by increasing the Fast Green FCF concentration 10 fold, since with the lower concentration[4] incorrect Eosinophilic Indices[3] were obtained when compared with the standard Shorr's trichrome stain[3]. The Lillie-Mayer haematoxylin used in the described modification is 4 times more rapid in action than Ehrlich's acid haematoxylin suggested by Schutte[3]. In addition to this, the Lillie-Mayer haematoxylin does not need the 6 month maturation period needed by Ehrlich's acid haematoxylin.

This technique places the use of canine vaginal smears within the limits of those specialised procedures able to be performed by the average busy city pratitioner who wishes to offer a more comprehensive small animal gynaecological service to the public.

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BOOK REVIEW

BOEKRESENSIE

TIERGEBURTSHILFE

J. RICHTER en R. GÖTZE

3e hersiene uitgawe deur R. Rosenberger en H. Tillman

Verslag Paul Parey, 2000 Hamburg 1, Spitalerstrasse 12, Wes-Duitsland. 178. pp 921. ISBN 3-489-73316-9. Prys nie vermeld nie.

Hierdie is 'n hersiene uitgawe van 'n omvattende Duitse handboek oor diereverloskunde wat oorspronklik in 1947 gepubliseer is en in 1960 herdruk is.

As inleiding word normale bevrugting, embrionale ontwikkeling, dragtigheid, partus en die puerperiale periode bespreek. Dit sluit selfs bestuur en voeding van dragtige diere in. Met tipiese Duitse deeglikheid beslaan die inleiding 174 bladsye!

Die res van die boek bestaan uit die volgende hoofstukke: patologie van dragtigheid; patologie van partus; patologie van die puerperium; patologie van die konseptus, pasgeborene en suigeling; infeksies van sogende kleintjies; en regsprobleme en aanspreeklikheid i.v.m. verloskunde. Elke onderwerp word grondig bespreek en toegelig met illustrasies, waarvan daar 638 is, sommige selfs in kleur. Veral die obstetriese manpulasies word baie duidelik beskryf en pragtig geïllustreer.

Hierdie handboek is dus baie meer as bloot 'n verloskundige handleiding en kan sterk aanbeveel word by elkeen wat 'n leeskennis van Duits het.

B.L. Penzhorn

SHORT COMMUNICATION **KORT BERIG**

PRELIMINARY REPORT: A PREGNANCY FROM FROZEN CENTRIFUGED DOG SEMEN

E.E. OETTLÉ*

ABSTRACT: Oettlé E.E. **A pregnancy from frozen centrifuged dog semen.** *Journal of the South African Veterinary Association* (1982) **53** No. 4, 269-270 (En) Department of Genesiology, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

Semen obtained from a pure-bred Beagle was diluted 1:3 with a Tris (hydroxymethyl) – aminomethane buffered egg yolk diluent, centrifuged at 450 *g* for 10 minutes and the spermatozoa thus concentrated stored in 8 Cassou mini straws (0,25 ml) in liquid nitrogen. One month after collection, a single semen straw was thawed, rediluted with 4 ml of diluent containing no glycerol and inseminated into a Beagle in oestrus. This was repeated daily for 4 days. The vagina of the bitch was flushed with 4 ml of the diluent immediately prior to insemination on each occasion. The bitch whelped 7 pups 60 days after the first insemination.

Key words: Centrifuged dog semen, frozen semen, pregnancy, vaginal washing.

INTRODUCTION

The first documented report of conception resulting from the use of frozen dog semen was from Saeger[7] who reported a pregnancy obtained from semen which had been stored in pellet form in liquid nitrogen for 6 months. The diluent he used consisted of 11 % lactose, 4 % glycerol and 20 % egg yolk. The pellets were thawed in 3 % sodium citrate solution a 30 °C and were deposited intravaginally.

Andersen[2] reported pregnancies using semen diluted with a Tris-fructose-citric acid extender containing 8 % (v/v) glycerol and 20 % egg yolk, frozen in medium sized straws (0,5 ml). They were thawed for 6,5 seconds in water at 75 °C and were inseminated into the uterus via the cervical canal. Each insemination dose contained 200-300 million spermatozoa and the bitches were inseminated 2 or 3 times.

Martin[5] froze dog semen in diluents containing 12 % skim milk and 8 % glycerol and found that the motility of spermatozoa after thawing was very poor unless they were first washed by centrifugation at about 300 *g* for 5 min and then resuspended in a diluent free of glycerol. However, the semen was not used for insemination.

This preliminary report outlines a method for storing semen in a concentrated frozen form and the insemination technique used to utilize the semen effectively.

MATERIALS AND METHODS

The basic diluent used consisted of the 0,16 mol Tris (hydroxymethyl) – aminomethane buffered egg yolk described by Vaske[11]. However, the kanamycin was replaced by 1 000 IU penicillin and 1 mg streptomycin per ml of diluent. Furthermore, the diluent was divided into 2 parts: Diluent A containing no glycerol and Diluent B containing 20 % glycerol. Both diluents were stored in PVC straws at – 15 °C until required.

Semen was collected by digital manipulation[7]. The ejaculate was fractionated visually by using glass collecting funnels and only the sperm-rich (second) fraction was retained for freezing. The equipment was warmed to 35 °C to prevent cold shock to the sperm. The semen was diluted 1:2 with Diluent A at 32 °C. The sample was then centrifuged at 450 *g* for 10 min and the supernatant discarded, leaving a sediment of 1,8 ml irrespective of the original volume. The temperature of the sediment was lowered at a rate of 1 °C per min to 5 °C, and then further diluted with 0,6 ml of Diluent B. The final glycerol concentration was 5 % (v/v). The sample was then equilibrated for 3 h at 5 °C, and then frozen in 8 mini straws (0,25ml) 25 mm above the liquid nitrogen level for 10 min[4] and finally submerged in the liquid nitrogen.

One month after freezing the semen was inseminated into a pure-bred multiparous Beagle bitch. Oestrus was detected by means of vaginal cytology[6]. Inseminations began when the eosinophilic index rose above 60 %. They were performed daily for 4 days. One each occasion the vagina was first flushed by instilling 4 ml of Diluent A with an insemination pipette inserted as far as possible into the anterior vagina, massaging the abdomen and then aspirating and discarding as much of the fluid as possible. The forequarters of the bitch were also elevated to assist drainage of the fluid. One straw (0,25 ml) of semen was used for each insemination. After thawing for 30 seconds in a water-bath at 30 °C the straw was emptied into a test-tube containing 4 ml of Diluent A (containing no glycerol), thus rediluting the semen 1:16. In this way the glycerol concentration was reduced to a negligible amount. The sample was evaluated microscopically and 60 % of the spermatozoa showed progressive motility. The rediluted semen was inseminated into the anterior vagina by means of a sterile syringe, rubber connector and a 450 mm insemination pipette. Each insemination contained a total of 125 million spermatozoa. The bitch's hindquarters were raised to an angle of 60 ° and held thus for 5 min. Thereafter, she was walked for 15 min to help prevent loss of semen from the vagina.

RESULTS

The bitch whelped 7 pups 60 days after the first insemination.

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DISCUSSION

Centrifugation of semen has the following advantages: 1) less storage space is needed and 2) the semen may be reconstituted on thawing in a glycerol-free and hence more favourable diluent. Preliminary investigations indicate that, when semen is treated in this way, the spermatozoa after thawing survive in vitro at 32 °C for significantly longer periods than do control samples which are not centrifuged and thus are also not rediluted. It seems therefore that the benefits of centrifugation outweigh the disadvantages of possible damage to sperm as a result of both the gravitational forces and the extra handling required.

Freezing the semen in mini straws was found to be convenient because each ejaculate yielded 8 straws, sufficient to inseminate 2 bitches. Labelling and storage is simple and thus records are easy to maintain. To date, all pregnancies resulting from frozen semen in dogs have been from semen which had been stored either in the pellet form[10] or in medium-sized (0,5 ml) straws[2 3]. Andersen[3] used 4-5 medium sized straws per insemination, which requires considerably more liquid nitrogen storage space than the single mini straw used in this trial. Diluent for redilution is stored in a refrigerator at -15 °C.

Andersen[1] reported that intravaginal deposition of semen frozen in medium-sized straws resulted in no pregnancies in the 8 bitches used in one trial. Pregnancies only resulted when the semen was deposited through the cervix and into the uterus[2 3] but this requires some degree of manual dexterity and practice. It would be advantageous to be able to obtain pregnancies from intravaginal deposition of semen. Saeger [7 8 9 10] obtained pregnancies from intravaginally deposited pelleted semen but the volumes he used were large (3-9 ml, i.e. 30-90 pellets).

In this trial, the problem of unfavourable vaginal environment was overcome by using diluent to flush out any debris present in the vagina prior to insemination. This also served to buffer any pH differences and line the vagina with a medium favourable for the semen.

Four ml of diluent were found to be a convenient amount both to store and to inseminate.

A larger trial is being conducted to test the methods described.

ACKNOWLEDGEMENTS

I should like to express my appreciation to Professor R.I. Coubrough and Dr R.O. Gilbert for their valuable advice.

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ABSTRACT: Gray, J.S. & Potgieter, F.T., 1982. **Studies on the infectivity of *Boophilus decoloratus* males and larvae infected with *Babesia bigemina*.** *Onderstepoort Journal of Veterinary Research,* **49**, 1-2 (1982).

Babesia bigemina was transmitted by male *Boophilus decoloratus* and also by intravenous inoculation of a homogenate prepared from infected incubated larval ticks.

ABSTRACT: Coetzer, J.A.W., 1982. **The pathology of Rift Valley Fever. II. Lesions occurring in field cases in adult cattle, calves and aborted foetuses.** *Onderstepoort Journal of Veterinary Research,* **49**, 11-17 (1982).

Since the original description of Rift Valley fever in sheep, cattle and man in the Rift Valley in Kenya in 1931, very little has been published on the disease in cattle. This report deals with the macroscopic and microscopic pathology of field cases of Rift Valley fever in 22 adult cattle, 8 calves and 8 aborted foetuses.

The microscopic liver lesions in 13 adult cattle were characterized by marked centri- and midzonal eosinophilic necrosis, involving almost ⅔ of the lobules, and accompanied by sparsely distributed primary foci of necrosis. In 3 animals, however, the hepatic lesions were more focal in nature, while a massive hepatic necrosis was evident in 6 others.

In calves, the lesions in the liver ranged from cases showing numerous haphazardly scattered primary foci of necrosis to cases where the latter were accompanied by eosinophilic necrosis of the remaining hepatocytes in the lobules. Vascular lesions, thrombosis and sinusoidal fibrin deposits were sometimes seen in the livers of both calves and adult cattle.

Although the aborted foetuses were in a fairly advanced state of autolysis, it was still possible to make a diagnosis of Rift Valley fever from the characteristic lesions which were similar to those reported for new-born lambs.

Other noteworthy lesions in adult cattle and calves included pyknosis and karyorrhexis of lymphocytes in the spleen and lymph nodes, widespread serosal and visceral haemorrhages which were sometimes accompanies by copious free blood in the gastriontestinal tract, and a nephrosis.

SHORT COMMUNICATION **KORT BERIG**

A SIMPLE REPAIR OF THE RUPTURED ANTERIOR CRUCIATE LIGAMENT IN THE DOG

C.G.N. TRACE*

ABSTRACT: Trace C.G.N. **A simple repair of the ruptured anterior cruciate ligament in the dog.** *Journal of the South African Veterinary Association* (1982) **53** No. 4, 271-273 (En) Highlands, 2 Kenilworth Rd, Harare, Zimbabwe.

A relatively simple surgical procedure for the repair of a ruptured anterior cruciate ligament in dogs is described.

Key words: Canine anterior cruciate ligament.

METHOD

The stifle joint is prepared for aseptic surgery in the usual manner. The joint cavity is approached through a cranio-lateral incision as shown in Fig. 1 (A. B. and C).

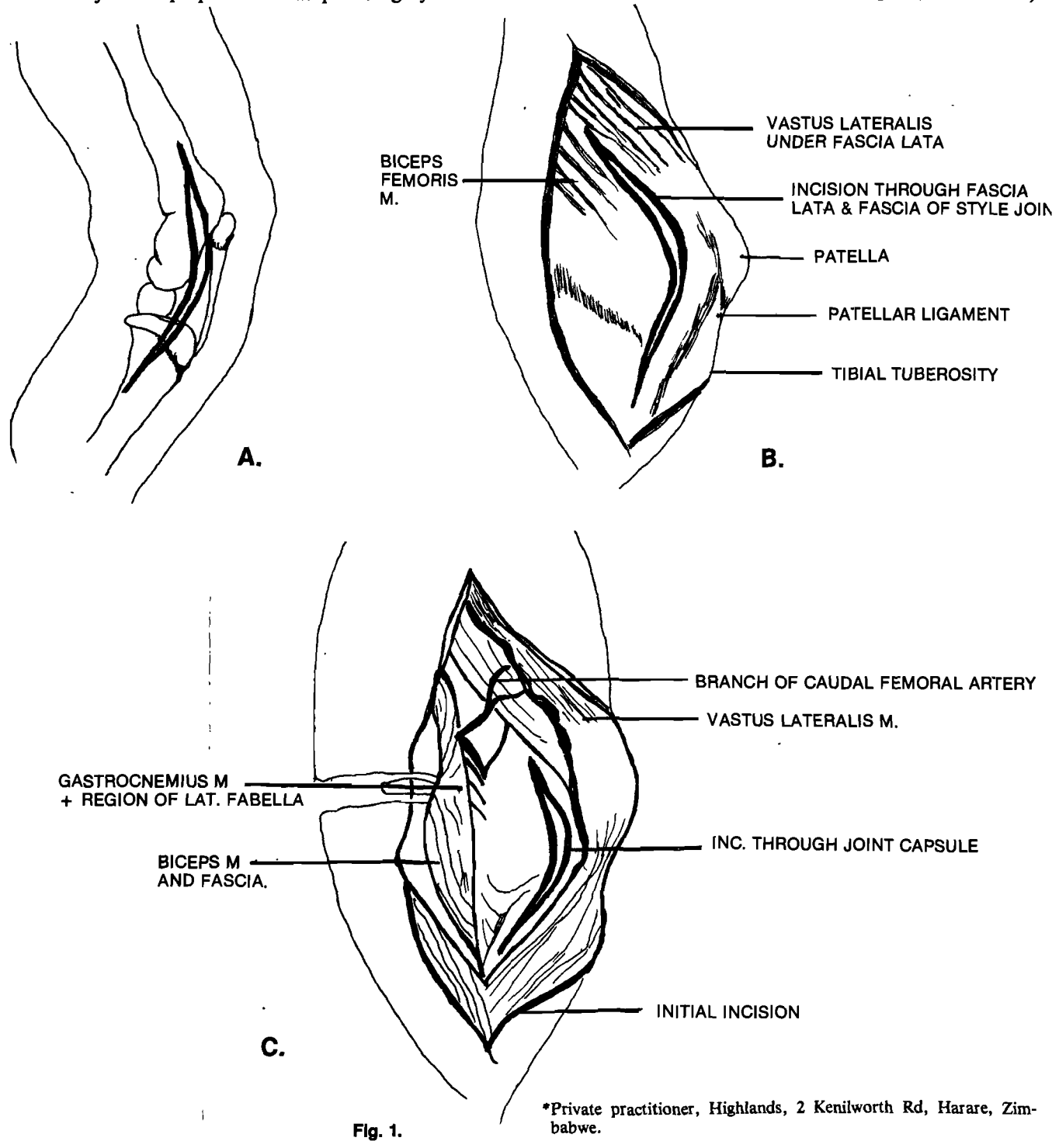

Fig. 1.

*Private practitioner, Highlands, 2 Kenilworth Rd, Harare, Zimbabwe.

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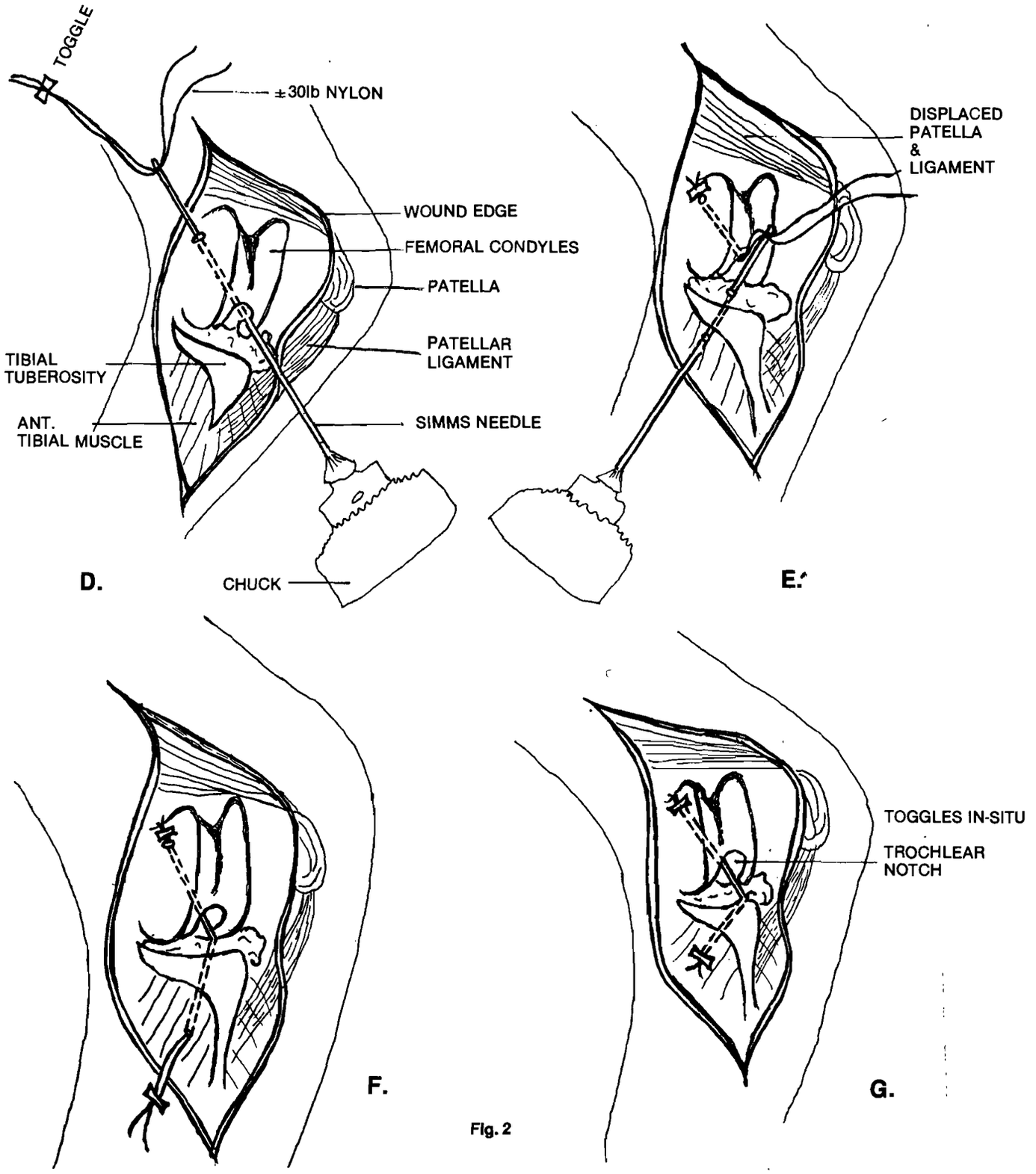


Fig. 2

The patella and its ligaments are displaced medially, and the joint fully flexed, giving excellent access to the joint and ruptured cruciate ligament (Fig. 2 D).

A Simms needle, the eyelet having been sharpened to act as a bone drill, is used to drill a passage from the trochlear notch, dorso-laterally through the lateral condyle to the lateral margin of the joint capsule (Fig. 2 D)

Two pieces of 20 lb or 30 lb nylon (monofilament) are threaded through the eye. The needle is then withdrawn taking the nylon with it. The dorsal ends are firmly tied to a small toggle made from a piece of suitable circlage stainless steel wire as in Fig. 3 (Hi and Hii)

The Simms needle is now freed from the nylon and redirected through the lateral ridge of the tibial tuberosity, as in Fig. 2 E, starting in the anterior tibial muscle and passing dorso-medially to appear as close as possible to the central tibial process. The loose ends of the nylon are again threaded into the eye of the needle and withdrawn as shown in Fig. 2 F.

A second wire toggle is attached between the nylon threads and pulled up tight once the joint has been extended; this toggle is embedded in the anterior tibial muscle origin and the nylon tightened to prevent all 'Drawer Movement' in the exposed joint.

The patella is replaced, a drop of chloramphenicol in-

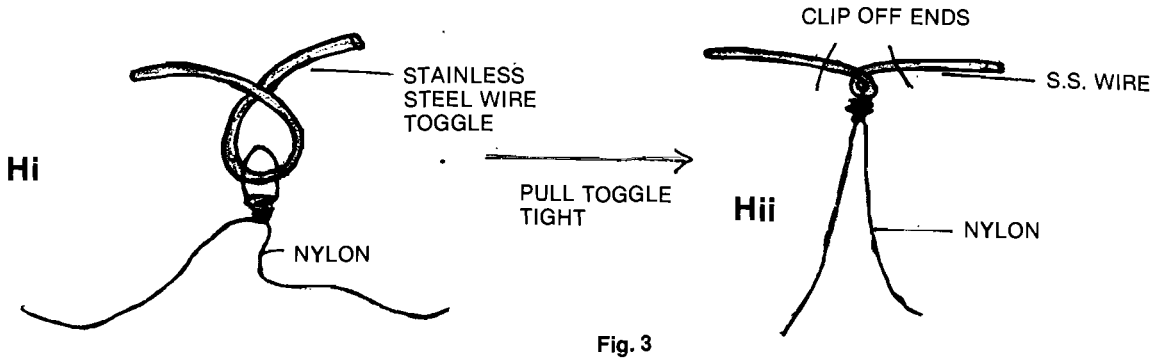


Fig. 3

jectible is placed into the joint and the capsule and the facia closed with 3/0 Dexon sutures, suitably strengthening the lateral patella ligament to prevent all medial luxation taking place.

The skin is closed in the normal manner. Prophylactic antibiotics are given for 5 days post operatively, and full functional movement should return in 10 days.

DISCUSSION

The operation is simple, quick and easy and can be repeated if necessary. 20 lb nylon is used for small dogs, and 30 lb for large dogs.

This article was written a year ago when insufficient cases had been operated on to be sure of the results. To date there have been no relapses, and no complications and this is now the standard method of cruciate repair in this practice.

The operation is much quicker and far less traumatic than all previous methods.

ABSTRACT: Herr, S., Te Brugge, Lesley A. & Guiney, M.C.M., 1982. **The value of the microtitre serum agglutination test as a second screening test in bovine bucellosis.** *Onderstepoort Journal of Veterinary Research,* **49,** 23-28 (1982).

The use of the serum agglutination test (SAT) as a 2nd screening test led to a reduction in false negative screening reactions especially in sera delayed in transit and provided an easy check on human error in the rose bengal test (RBT) and on each individual complement fixation test titre. The SAT in microtitration plates was more sensitive than when done in tubes and effected a saving of time, labour and materials.

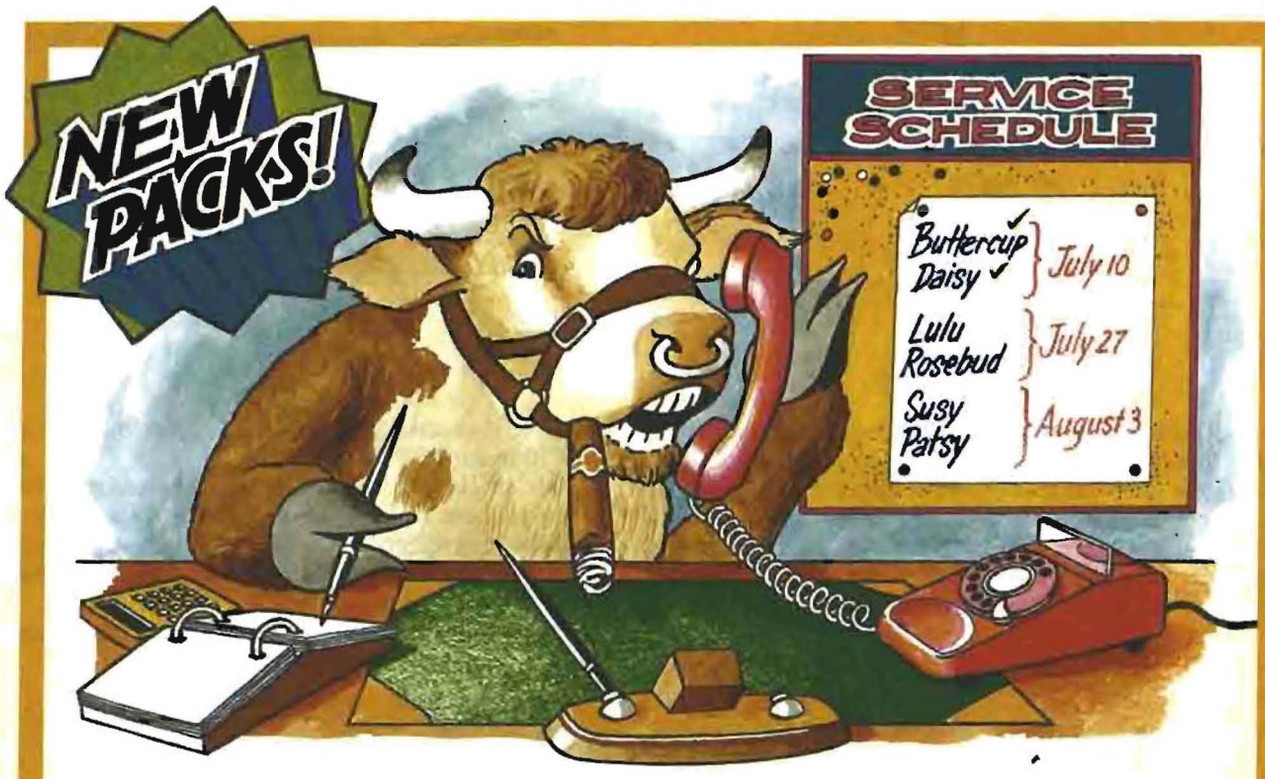
ABSTRACT: Viljoen, J.H. & Coetzer, J.A.W., 1982. **Studies on *Parafilaria bovicola* Tubangui, 1934, III. Pathological changes in infested calves.** *Onderstepoort Journal of Veterinary Research,* **49,** 29-40 (1982).

More lesions were found in the carcass of an animal that has been naturally infested with *Parafilaria bovicola* than in one artifically infested with a single subcutaneous injection of infective larvae of this species. This may be because natural infestations are either more frequent or more successful. Similarities in the distibution of lesions in naturally and experimentally infested animals suggest that certain predilection sites may be used by the intermediate fly hosts. Subcutaneous areas infiltrated with eosinophils are more conspicuous during the first 20 days after infestation and during the patent phase of the life cycle of *P. bovicola.*

Yellowish discolorations caused by oedema are usually present in all lesions. When these are combined with eosinophil infiltrations, the lesions become yellowish-green. After the appearance of bleeding spots the green colour of lesions is dominated by the appearance of a brown pigment (haemosiderin) in numerous macrophages. The histopathological changes in the dermis, subcutis and superficial muscles bordering the affected areas are described.

ABSTRACT: Boomker, 1982. **Parasites of South African freshwater fish. I. Some nematodes of the catfish [*Clarias gariepinus* (Burchell, 1822)] from the Hartbeespoort Dam.** *Onderstepoort Journal of Veterinary Research,* **49,** 41-51 (1982).

A seasonal study of the parasites of fish in the Hartbeespoort Dam was undertaken in 1979. This paper deals with 4 nematode species recovered from catfish, namely, *Paracamallanus cyathopharynx* (Baylis, 1923), *Procamallanus laeviconchus* (Wedl, 1862), *Contracaecum* sp. and *Skrjabinocara* sp. Total numbers of parasites recovered are tabulated and their seasonal variation illustrated diagrammatically. *Paracamallanus cyathopharynx* was recovered from 23 out of 43 catfish examined and *Procamallanus laeviconchus* from 13, while *Contracaecum sp.* larvae were present in all the catfish. *Skrjabinocara* sp. was recovered from 1 catfish only, but it is not regarded as being parasitic in fish, as it was also recovered from 1 out of 4 cormorant examined. *Paracamallanus cyathopharynx* and *Procamallanus laeviconchus* are illustrated and the measurements of the Hartbeespoort Dam material compared with those given by various authors who recovered the same parasites from other fish species elsewhere in Africa.



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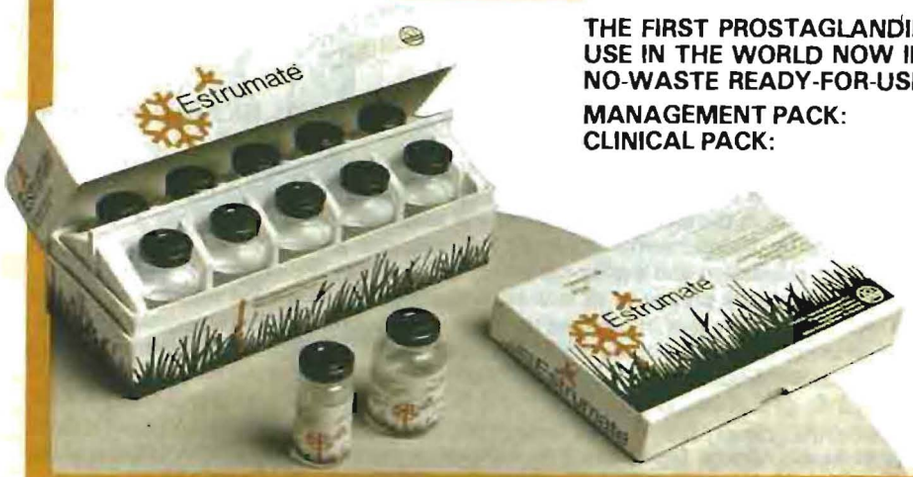

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THE URETHRAL DIVERTICULUM OF THE BULL

A.J. BEZUIDENHOUT* and D.J. COETZER*

ABSTRACT: Bezuidenhout A.J.; Coetzer D.J. **The urthral diveticulum of the bull.** *Journal of the South African Veterinary Association* (1982) **53** No. 4, 275-276 (En) Department of Anatomy, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Africa.

The exact location of the urethral diverticulum of the bull was determined. It was found to lie 80-100 mm caudal to the colliculus seminalis, at the transition from the pelvic to the penile urethra. The presence of the diverticulum makes catheterization of the urinary bladder via the penile urethra very difficult.

Key words: Urethral diverticulum, bull, location, urethral catheterization.

INTRODUCTION

In their description of the bulbo-urethral glands of the bull, Leydig and Oudemans, as quoted by Disselhorst[1], state that the ducts of the glands open into the urethra on a fold of mucous membrane, forming a small blind sac. Attention to the presence of the fold was again drawn by Le Roux & Smuts[2]. The latter authors for the first time refer to the hollow as the *Diverticulum urethrale,* a term at present not recognized by Nomina Anatomica Veterinaria[3].

The present study was undertaken to determine the exact location of the diverticulum and to remind clinicians of this feature of the bovine male urethra.

MATERIALS AND METHODS

The pelvic urethrae of 50 freshly slaughtered male bovines were examined. To expose the diverticulum, the uerthra was opened along its ventral wall, starting at the uranary bladder. The position of the diverticulum in relation to the bony pelvis was determined on 3 formalin fixed pelvices.

RESULTS

The ducts of the bulbo-urethral glands open into the urethra on a fold of mucous membrane. The fold spans the width of the lumen of the urethra, forming a blind sac dorsally to it. The blind sac or diverticulum so formed varied in depth from 10-15 mm in those animals which were studied. The free end of the fold, i.e. the opening into the diverticulum lies 80-100 mm caudally to the colliculus seminalis, with the blind end at the level of the bulbo-urethral glands where the penile bulb starts (Fig. 2). This implies that the opening of the diverticulum and also the urethral openings of the bulbo-urethral glands lie in the pars spongiosa or penile urethra, while the blind end lies at the junction of the pelvic and penile urethra. The diverticulum lies just caudally to the point where the urethra turns ventrally around the ischial arch, so that the entrance into the diverticulum points ventrally (Fig. 2). A line connecting the left and right ischial tuberosities passes through the diverticulum.

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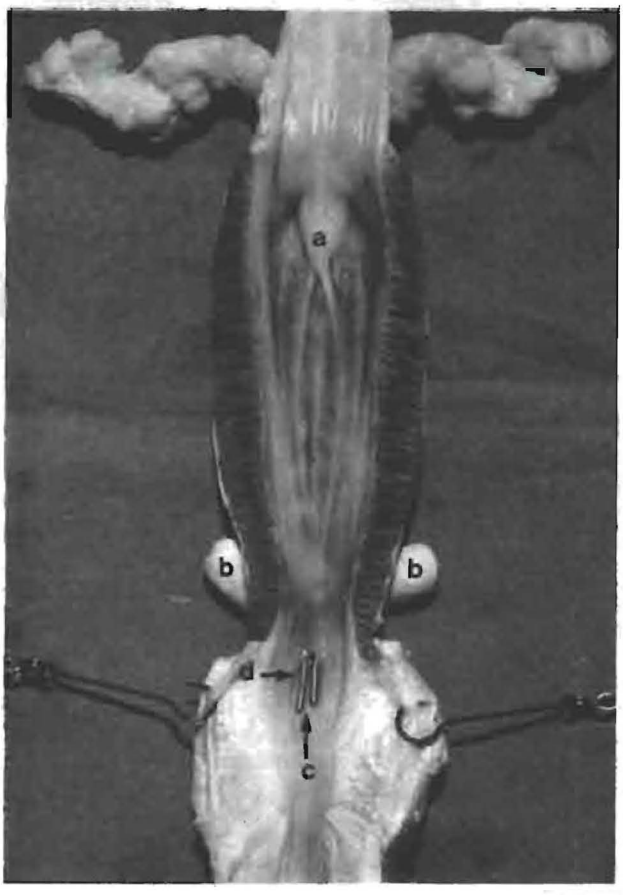

Fig. 1: Pelvic and penile urethra opened along its ventral surface with the colliculus seminalis a, bulbo-urethral glands b with probes c in their urethral openings and urethral diverticulum d.

The diameter of the pelvic urethra narrows abruptly at the diverticulum, from where it is continued as the penile urethra.

DISCUSSION

The presence of a diverticulum in the urethra of the male bovine animal can present the veterinary clinician with 2 problems:

Firstly, a catheter which is passed up the penile urethra generally enters the diverticulum. It is very dif-

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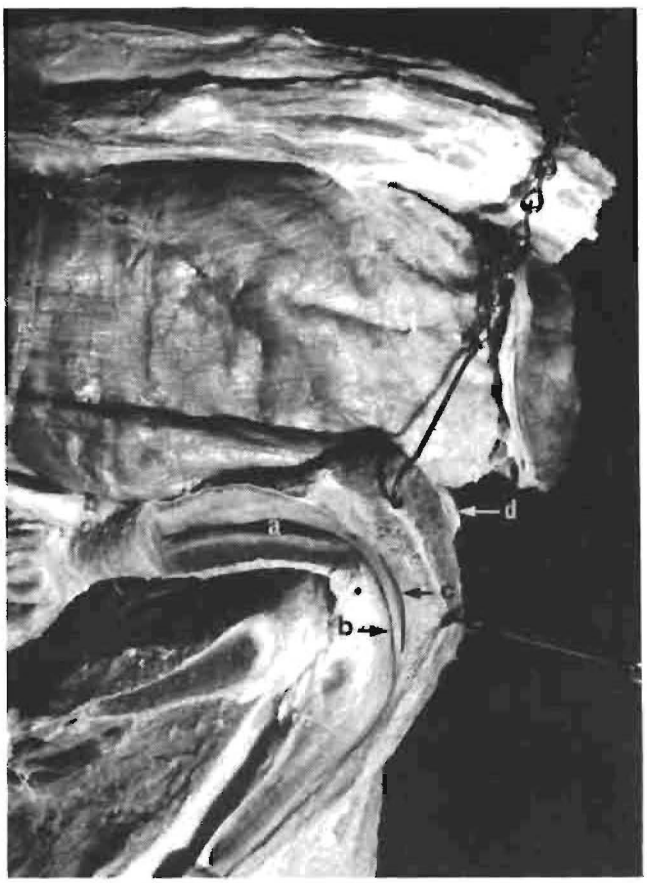

Fig. 2: Urethra in situ. Pelvic urethra a, penile urethra b, urethral diverticulum c, ischial tuberosity d and pelvic symphysis e.

ficult to get the catheter past the diverticulum, making catheterization of the urinary bladder via the penile urethra often impossible;

Secondly, because of the narrowing of the lumen of the urethra, urinary calculi can lodge in the constricted part just distally to the diverticulum. If an attempt is made to push the calculus back into the bladder, it may enter the diverticulum. In this case the clinician must then resort to surgery.

It is therefore essential that the clinician should be aware of the presence of the urethral diverticulum when catheterizing the urethra or when dealing with urinary calculi in the male bovine animal.

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ABSTRACT: Reinecke, R.K., De Villiers, I.L. & Brückner, Christel, 1982, **Studies on *Haemonchus contortus*. VI. Attempts to stimulate immunity to abomasal trichostrongylids in Merino sheep.** *Onderstepoort Journal of Veterinary Research,* **49,** 3-6 (1982).

Two doses of infective laevae of 20 000 *Triehostrongylus axei*, dosed to Merino lambs at an interval of 14 days and subsequently challenged with *Ostertagia circumcincta*, caused a significant reduction ($P < 0,01$) in the establishment of 5th and adult stages of the latter. *T. axei* was unable to protect Merino sheep against homologous challenge nor was *Haemonchus contortus* a successful vaccine against challenge with the same species. The vaccinated group showed a reduction ($P < 0,025$) only in 5th and adult *H. contortus*, but not in the total worm burdens.

ABSTRACT: Horak, I.G., Meltzer, D.G.A & De Vos, V., 1982 **Helminth and arthropod parasites of springbok, *Antidorcas marsupialis*, in the Transvaal and western Cape Province.** *Onderstepoort Journal of Veterinary Research,* **49,** 7-10 (1982).

The helminth burdens of 17 springbok from 2 localities in the Transvaal and of 4 springbok from the western Cape Province were determined. Eight of the animals from the Transvaal and the 4 from the Cape Province were also examined for arthropod parasites.

In all, 26 helminth species, 5 species of ixodid ticks and 4 species of lice were recovered from the springbok. Of the helminths *Dictyocaulus magnus, Trichostrongylus axei, T. falculatus and Agriostomum equidentatum* were recovered from animals in each of the surveys. The lice *Damalinia antidorcus, Linognathus antidorcitis and L. bedfordi* were present on animals in the Transvaal and the western Cape Province.

ABSTRACT: Coetzer, J.A.W. & Theodoridis, A., 1982. **Clinical and pathological studies in adult sheep and goats experimentally infected with Wesselsbron disease virus.** *Onderstepoort Journal of Veterinary Research,* **49,** 19-22 (1982).

The clinical symptoms and pathology in 33 adult sheep and 31 adult goats experimentally infected with Wesselsbron disease virus are described. There was moderate to severe hyperthermia in most animals, but no other clinical signs of disease or deaths were recorded.

Eleven sheep and 6 goats were sacrificed for pathological studies at various stages during the febrile response. The macroscopic and microscopic lesions in these cases are described. Microscopic studies revealed that the liver was consistently affected and showed small foci of necrosis. These were sparesely distributed and associated with a marked localized Kupffer cell response ("retothelial nodules"). In addition, acidophilic bodies and small groups of necrotic hepatocytes were evident in some lobules. Apart from the hepatic lesions, mild to moderate pyknosis and karyorrhexis of lymphocytes were seen in the spleen and lymph nodes.

This report also compares the microscopic lesions in the livers of adult sheep and goats with those of new-born lambs for Wesselsbron disease as well as with those reported for Rift Valley fever in adult sheep.

SHORT COMMUNICATION **KORT BERIG**

MECHANICAL MYDRIASIS DURING INTRA-OCULAR SURGERY

S.W. PETRICK

ABSTRACT: Petric S.W. **Mechanical mydriasis during intra-ocular surgery** *Journal of the South African Veterinary Association* (1982) 53 No. 4, 277 (En) Department of Surgery, Faculty of Veterinary Science, University of Pretoria, P.O. Box 12580, 0110 Onderstepoort, Republic of South Arica.

The use of an improvised iris speculum during intra-ocular surgery is described.

Ample mydriasis is achieved by using a mydriatic prior to intra-ocular surgery. A single application the evening prior to the day of surgery and two or three applications hourly before the operation is usually sufficient.

However, in a high percentage of cases, miosis occurs shortly after the anterior chamber is opened and the aqueous humour escapes. This can only be attributed to the loss of pressure in the anterior chamber. It stands to reason that further intra-ocular surgery is difficult without trauma to, and haemorrhage of the iris.

To prevent miosis one or more iridotomy incisions are indicated.

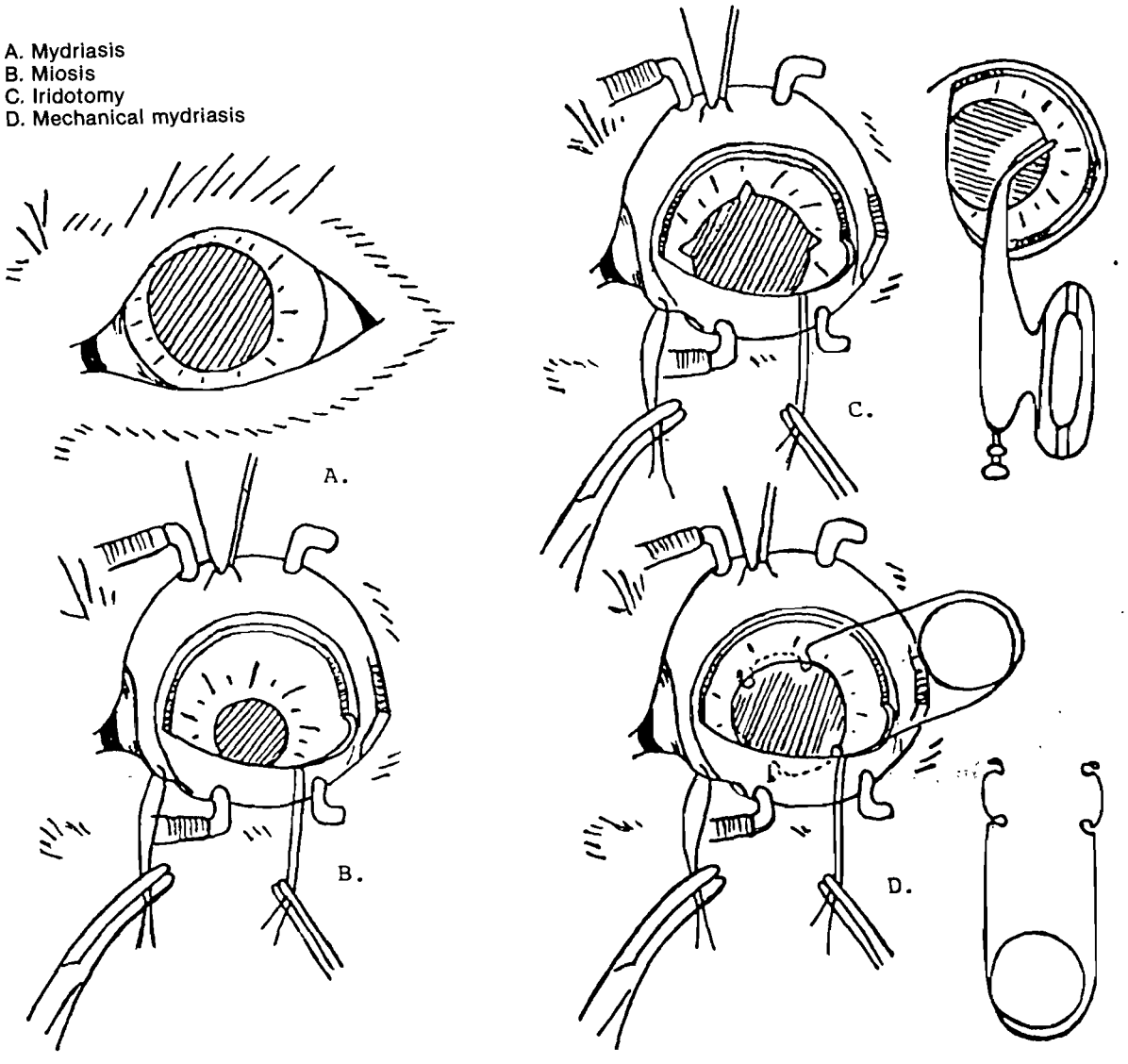
Apparently a more vascular iris in animals, compared to that of *Homo sapiens*, lends itself to unnecessary haemorrhage during this procedure.

Mechanical mydriasis is achieved by using an iris speculum prepared from thin surgical wire. Various iris retractors are available but as yet no iris speculum.

The need for a properly designed and constructed iris speculum is well justified and its use advocated.

Fig. 1 schematically illustrates mydriasis before intra-ocular surgery, miosis after the loss of aqueous humour, iridotomy with iris scissors and mechanical mydriasis by means of an iris speculum.

Fig. 1: A. Mydriasis
B. Miosis
C. Iridotomy
D. Mechanical mydriasis



0038-2809/82/04/0277-0277

TO THE EDITOR **AAN DIE REDAKSIE**

OXYTETRACYCLINE PLASMA LEVELS IN DOGS

Further to my previous communication on the paper 'Oxytetracycline Plasma Levels in Dogs after Intramuscular Administration of Two Formulations' by A. Immelman and Gillian Dreyer, I would like to add the following:

It seems that a certain amount of confusion has arisen since publication of this paper, in that practitioners are using propylene glycol (P.G.) formulations of oxytetracycline injectables at "double strength" (i.e. 20 mg/kg) and expecting 'long acting' results.

Two very important points need to be made:

- As is shown by Prof. Immelman's results the Long Acting formulation (Terramycin LA*, in 2-pyrrolidone base) gives plasma levels above 0,6 ug/ml (generally regarded as the Mean Inhibitory Concentration (M.I.C.) for oxytetracycline) for longer than 72 hours post injection, whereas the polyvinylpyrrolidone (P.V.P.) formulation (Terramycin 100*) drops below this level after 48 hours post injection.
- What is of even greater importance to the practitioner is that the P.V.P. formulation gives longer lasting blood levels than the more commonly available P.G. formulation and thus when using this drug type an even shorter acting effect can be expected.

I trust that this serves to clear up any existing misconceptions.

C.W. Moore
Pfizer Laboratories (Pty) Ltd
P.O. Box 783720,
Sandton
2146

TO THE EDITOR **AAN DIE REDAKSIE**

ACID QUARTER MILK AND THE CALIFORNIA MASTITIS TEST

In performing Schalm's California Mastitis Test the characteristic dark purplish blue viscous mass is well known as a positive reaction indicating alkaline milk with a high somatic cell content. Normal milk has a pH of about 6,6 and as the pH rises, so does the brom-cresol-purple indicator in the test reagent become progressively more dark & purple. We have recently come across a few instances where direct CMT examination of fresh quarter milk has revealed a bright yellow (& therefore acid) gel and in all instances *Streptococcus agalactiae* could be isolated.

When Broadhurst-Paley stained smears of such milk was examined microscopically the somatic cells were found to have a distinctly reddish colour instead of a dark purple colour usually observed.

All three the common mastitis streptococci (*S. agalactiae, S. uberis* & *S. dysgalactiae*) have the ability to ferment lactose although in the former it is a variable characteristic.

The acidic reaction could not be reproduced by artificial induction of mastitis, using the original isolates from acid milk and so we conclude that other factors must play a role in neutralising the carbonates and chlorides which abound in the secretion of an inflamed quarter. Perhaps there is a dual infection of such glands and the acidifying cooperation of a strongly lactose fermenting organism such as *S. lactis* is a distinct possibility.

We would be most pleased to hear from colleagues who encounter such acidic gels when doing the CMT on quarter milk and would in particular like to receive aseptically drawn milk samples for laboratory investigation.

L.W. v.d. HEEVER and
J.H. du PREEZ
Division of Veterinary Public Health
Department of Pathology
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P.O. Box 12580
0110 Onderstepoort

AWARD **TOEKENNING**

GOUE MEDALJE VAN DIE SAVV VIR 1982
HENRI PIETER ALBERT DE BOOM

Die Goue Medalje van die Suid-Afrikaanse Veterinêre Vereniging is vanjaar aan prof. H. P. A. de Boom toegeken.

Henri Pieter Albert de Boom die seun van 'n Nederlandse immigrantevader en boerenooimoeder is op 16 Oktober 1914 te Pretoria gebore. Hy voltooi sy skoolloopbaan aan die Hogere Oost Eind School Pretoria en kwalifiseer as veearts aan die Fakulteit Veeartsenykunde, Universiteit van Pretoria in 1936.

Hy begin sy wetenskaplike loopbaan in 1937 as staatsveearts verbonde aan die Patologie Seksie van Navorsingsinstituut vir Veeartsenykunde, Onderstepoort. Hy konsentreer aanvanklik op diagnostiek en bekwaam homself mettertyd as 'n gesiene histopatoloog met spesiale belangstelling in onkologie.

Sy belangstelling in anatomie en embriologie word geprikkel en in 1940 aanvaar hy 'n deeltydse aanstelling as lektor in anatomie (spesifiek embriologie en vergelykende anatomie) aan die Fakulteit Veeartsenykunde, Onderstepoort. 'n Statebondsbeurs stel hom in 1953/54 in staat om nagraadse studie aan Cornell Universiteit, New York te onderneem in embriologie, histochemie, neuroanatomie en endokrinologie en in 1955 volg hy prof. Cecil Jackson op as Hoof van die Departement Anatomie.

Alhoewel hy nog steeds as patoloog funksioneer is sy hoofopdrag nou egter anatomie en vanaf 1957 is hy voltydse professor in Anatomie 'n pos wat hy met besondere onderskeiding vul tot met sy aftrede as Professor Emeritus in 1974.

Tydens sy ruim 38 jaar as leermeester van aspirant veeartse het H.P.A. de Boom – of Boompie – sinoniem geword met entoesiasme, warm menslikheid, wetenskaplike nuuskierigheid en verstommende insig, ook in die geesteswetenskappe. Sy bydrae tot die vooruitgang van die wetenskap lê by uitnemendheid in die hoë standaard wat hy gestel het en die meesterlike wyse waarop hy as dosent van Anatomie en verwante vakrigtings sy studente geïnspireer het tot selfstandige denke en kritiese waarneming.

Hy is 'n wetenskaplike van wêreldformaat op sy vakgebied en sy insig in die ontwikkeling van die soogdierembrio kan kwalik oortref word op internasionale vlak. Hy het gevolglik 2 keer (1962/63 en na sy aftrede 1975/76) die eer tebeurt geval om genooi te word as besoekende Professor in Veterinêre Anatomie aan die New York State Veterinary College, Cornell Universiteit, Ithaca. Die waarde van sy 2 besoeke aan Cornell spreek uit die hoë agting en liefde waarmee kollegas en studente hom onthou. Prof. Evans, Hoof van daardie Departement Anatomie en huidig President van die Wêreldvereniging van Veterinêre Anatome, was gedurende 1981 gasprofessor alhier. Hy het weer eens sy landgenote se hoogste agting vir prof. de Boom se kennis en bekwaamhede bevestig.

In 1968/69 en 1969/70 is hy op versoek van die Portugese regering aangestel as Professor en Hoof van die Departement Anatomie aan die Fakulteit Veeartsenykunde te Lourenco Marques en doen hy baanbrekerswerk aan hierdie Universiteit. Een van sy uitstaande eienskappe en deugde word hier illustreer: Die vermoë om ander mense te inspireer en te laat voel dat hul die potensiaal en toerusting het om self te skep en te bou. Hy het homself nooit beskou as die sleutelfiguur nie maar het voortdurend met geesdrif en insig andere se idees en oorspronklikheid ondersteun en laat blom. Hierdie selflose leierskap is beantwoord met al sy medewerkers se lewenslange respek en geneëntheid.

Sy besondere belangstelling in teratologie het daartoe gelei dat 'n unieke versameling van monsters oor baie jare opgebou is en uitgestal word en in die beplande Fakulteitskompleks 'n ereplek sal kry as "Die de Boom-versameling". Hy is dan ook by die veterinêre farmaseutiese nywerheid 'n gesogte deskundige wanneer

dit by die teratogene newe-effekte van sekere middels kom. Sy baanbrekerswerk en uiters oorspronklike navorsing op die mikroskopiese identifikasie van wildshare op biltong het die polisie in staat gestel om wilddiewe aan die kaak te stel en het grootliks bygedra tot die bekamping van hierdie euwel. Die tegniek is nou oorgeneem deur die S.A. Polisie se Forensiese Laboratorium en word nog steeds gebruik.

Prof. de Boom is vanaf 1937 'n hoogs aangeskrewe lid van die SAVV. Hy stel intens belang in die wetenskap skryfkuns en sy lang en aktiewe betrokkeneid by die redaksie van die joernaal van die vereniging oor 'n periode van 29 jaar (11 daarvan as Redakteur) het sy breë belangstelling in al die fasette van veeartsenykunde ingeskerp. Hy het meestal self die wetenskaplike bydraes deurgelees en kon talle kere feitelike foute wat ver buite sy vakgebied lê raaksien en aan outeurs uitwys. Uit sy eie pen het 20 publikasies oor wyd uiteenlopende onderwerpe verskyn. Behalwe dat hy 8 jaar lank Fakulteitverteenwoordiger op die Veeartsraad was, was hy vir 2 jaar ook die SAVV-verteenwoordiger. Die talle jare van onbaatsugtige diens wat hy aan die Vereniging, en besonder aan die Raad gelewer het, het daartoe gelei dat hy in 1977 tot Lewenslange Ere-Visepresident van die Vereniging verkies is.

Hy geniet die hoogste aansien as veterinêre opvoedkundige en wetenskaplike, en is betrokke by talle verenigings: onder andere is hy sedert 1942 assesorlid van die Suid-Afrikaanse Akademie vir Wetenskap en Kuns en is volle lid sedert 1962 en was hy ook voorsitter van die Eugène Nielen Marais-tak van die Akademie. Hy was vir talle jare aktief betrokke by die besture van die S.A. Biologiese Vereniging sowel as die Suid-Afrikaanse Vereniging ter Bevordering van die Wetenskap en die Anatomiese Vereniging van Suider-Afrika en was President van al drie.

Prof. de Boom het na sy aftrede nog eens so aktief voortgegaan en was o.a. vir 2 jaar besoekende Professor in Anatomie te Cornell en het proff. Smuts van die Fakulteit Veeartsenykunde en Stampa van Fort Hare vir kort periodes afgelos. Toe hy deur die Mediese Fakulteit van die Mediese Universiteit van Suider-Afrika genader is om vir 2 jaar Menslike Anatomie en Embriologie te doseer het hy dit met die groot gemak en sukses gedoen – weer eens 'n bewys van sy aanpasbaarheid en akademiese breedte dat hy onmiddellik kon inskakel by die menslike anatomie en aan sy studente die vergelykende aspekte kon oordra wat sin gee aan anatomiese begrip. Dit is dan ook nie verbasend dat hier die veelsydige leermeester vanaf Julie 1982 (weliswaar weens ouderdom op 'n tydelike basis) as Professor en Hoof van die Departement Veterinêre Anatomie aan Medunsa aangestel is nie.

Hy is dwarsdeur sy loopbaan getrou en baie bekwaam deur sy vrou Sarie bygestaan en haar aandeel in hierdie besondere toekenning is aansienlik.

Hierdie begaafde veelsydige wetenskaplike en mens wat sonder eiewaan sy talent van hart en verstand in diens van Veeartsenykunde gestel het, verdien die hoogste eerbewys van ons Vereniging.

ABSTRACT: Herr, S., Riley, A.E., Neser, J.A., Roux, D. & De Lange, J.F., 1982. ***Leptospira interrogans* serovar *pomona* associated with abortion in cattle: isolation methods and laboratory animal histopathology.** *Onderstepoort Journal of Veterinary Research,* **49**, 57-62 (1982).

Leptospira interrogans serovar *pomona* was successfully isolated from cattle urine in the western Transvaal after an abortion storm had occurred. Direct inoculation of EMJH medium proved the most successful method. The selective agent, 5-fluorouracil, was most effective in controlling contamination when used at the 0,4 mg/ml level. The strain isolated was pathogenic in hamsters, but specific lesions and the leptospirae were seen only where overwhelming infection occurred.

ABSTRACT: Herr, S., 1982 **Prozones and delayed reactions in the rose bengal test for bovine brucellosis.** *Onderstepoort Journal of Veterinary Research,* **49**, 53-55 (1982).

A prozone and a somewhat delayed reaction were found to be present in the rose bengal test (RBT) when sera, delayed in transport, gave rise to false negative results. A postulate as to the mechanisms possibly involved in this slow prozone reaction is offered.

AWARD **TOEKENNING**

JACK BOSWELL AWARD FOR 1982
VERA JEAN EVELYN AMOS

The Jack Boswell award for 1982 has been awarded to Dr Vera Jean Evelyn Amos, private practitioner of Durban for dedicated services to the veterinary profession. Vera Amos can be regarded as the mother of the veterinary profession in South Africa: she is the daughter of a veterinary surgeon, wife of a veterinary surgeon and two of her sons and a daughter-in-law are practising as veterinarians in South Africa.

She was born in Durban and completed her schooling at Wykeham School in Pietermaritzburg.

She wanted to study veterinary science at London but the principal at Camden Town would not permit women students and she consequently went to the Liverpool School. While there she was elected Woman President of the Students' Union. Due to a change in principals at London, to one who would accept women, she moved from Liverpool and qualified in London in 1934 as an M.R.C.V.S.

She went straight into a mixed practice in Yorkshire which she enjoyed very much but due to her father's health, she returned to Durban in 1934 and joined him starting a small animal practice on the stoep of the house under very trying circumstances.

The South African Veterinary Association always played an important part in her life: She joined it in 1935 at a conference at Onderstepoort. She has been a highly esteemed member of the Natal Branch since 1936 and was given the status of Honoured Member of this branch in 1976. She is an extremely active member who almost without fail attends all branch and group meetings in which she actively participates.

In 1936 Leonard Morford came from England and joined the practice. They were married in 1937 and continued working for her father. In 1940 her husband was called up to serve in the Veterinary Corps and she was left with 2 small children and a lot of work on her hands in a busy practice. In 1946 they bought the practice from her father and their ambition of an own surgery was fulfilled in 1951. By this time, having had 5 children and a very hard working life during the war, her consulting hours were cut and she was given all the operative work. She always had a particular interest in small animal surgery and still actively operates today.

Her diversity as a veterinarian is further demonstrated by the fact that when her husband died in 1964, she was appointed in his place as veterinary surgeon to the Oceanographic Research Institute – a post which she held for 10 years.

Professionally she was intimately associated with the SPCA. In 1935 she was made Honorary Veterinary Surgeon to this society and served them for 12 years. In 1939 she established an Animal Emergency Depot and trained her own black and white helpers. For this she was awarded the South African Medal for War Services.

Her wide interest in leadership is further amplified by the fact that she has been active member of Soroptomists International of Durban since 1965 and that she has just held office as President of the South African National Council of Soroptomist Clubs for 2 years.

She has had an intense interest in veterinary science all her life and (in her own words) her main objects in professional life are:

Firstly: To maintain strictly ethical relationship with all colleagues which lead to friendship throughout the profession

Secondly: To instill in all people that caring for the patient is an essential without which medical treatment brings little reward.

She has always welcomed young and prospective colleagues who wanted to see what working with small animals means and finds them stimulating and rewarding. Numerous veterinarians have passed through her capable bands and have been positively influenced by her enthusiasm and exemplary professional standards. She has also succeeded in keeping contact with many of these people all over the world.

There is no doubt that Vera Amos has elevated the image of the veterinary profession in an outstanding manner and it is most fitting that the first Boswell Award to be bestowed on a lady, be awarded to her.

CLINICAL AWARD OF THE SAVA FOR 1982
DAVID JOHN MOORE

The second recipient of this award of the SAVA is Dr David John Moore of Johannesburg and the award is made for outstanding work published in 1979.

Dr Moore graduated from the Faculty of Veterinary Science at Onderstepoort in 1968.

He directly joined a private practice in Johannesburg and his professional life has been characterised by the exceptional high scientific standards which he maintains throughout. Despite a very busy practice he enrolled at the Faculty of Veterinary Science of the University of Pretoria and through part-time study over several years was awarded the M.Med.Vet.(Med.) degree in 1978. The title of his dissertation was: Disseminated intravascular coagulopathy: A complication of *Babesia canis* infection in dogs. At present he is registered for a D.V.Sc.

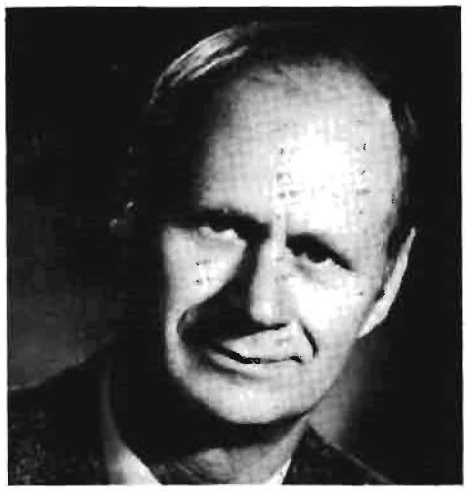
The Clinical Award for 1982 is awarded to him for the following excellent series of 3 articles which have appeared in the Journal of the SAVA on the subject of his dissertation:

Moore J.D. 1979. Disseminated intravascular coagulation: A review of its pathogenesis manifestations and treatment. Journal of the South African Veterinary Association 50: 259-264.

Moore J.D., Williams M.D. 1979. Disseminated intravascular coagulation: A complication of *Babesia canis* infection in the dog. Journal of the South African Veterinary Association 50: 265-275.

Moore J.D 1979. Therapeutic implications of *Babesia canis* infection in dogs. Journal of the South African Veterinary Association 50: 346-352

NAVORSINGSTOEKENNING VAN DIE SAVV 1982
DANIEL JACOBUS SCHNEIDER



Die tweede ontvanger van hierdie toekenning is dr Daniel Jacobus (Ters) Schneider.

Dr Schneider het in 1950 met lof as veearts aan die Fakulteit Veeartsenykunde, te Onderstepoort gekwalifiseer. Hy begin direk met 'n privaat praktyk in Wellington waar hy vir die volgende 25 jaar (waarvan 20 jaar alleen) tot 1976 met onderskeiding as 'n hoog aangeskrewe, suksesvolle en beminde veearts gewerk het.

In 1976 besluit hy dat hy graag dieper ondersoek wil instel na die talle probleme wat hy as praktisyn mee te doen gekry het in die Boland en sluit hy by die Afdeling Veeartsenydiens aan en begin hy werk by die Streekslaboratorium op Stellenbosch. Hy stel veral belang in toksikologie en gedurende die volgende paar jaar ontdek en beskryf hy 3 nuwe plantvergiftiging in sy omgewing en is ook intiem betrokke by die eerste diagnose en beskrywing van stachybothriotoksikose in die Kaap – uitstaande prestasie vir 'n privaat praktisyn wat vir 25 jaar slegs in praktyk gewees het.

Hierop volg sy uitstaande diagnose van eenjarige roggrasvergiftiging wat in die volgende artikel beskryf word:

Schneider D.J. 1981. First report of annual ryegrass toxicity in the Republic of South Africa. Onderstepoort Journal of Veterinary Research 48: 251-256.

Die prestasie lê veral daarin dat dr Schneider vir hom en die RSA, 'n onbekende probleem teëgekom het wat hy as staatsveaarts met relatief min fasiliteite tot sy beskikking deur noukeurig waarneming en eenvoudige maar goed beplande eksperimente, korrek gediagnoseer het. Hierop is dit opgeskryf en in 'n joernaal met hoë standaarde gepubliseer. Vir hom as oudpraktisyn was hierdie 'n onbekende sfeer wat selde betree word vanweë al die obstruksies langs die pad. Nogtans het hy die hele paadjie geloop.

Vir hierdie besonder oorspronklike en korrek afgeronde werk word die Navorsingstoekenning vir 1982 aan dr Schneider toegeken.

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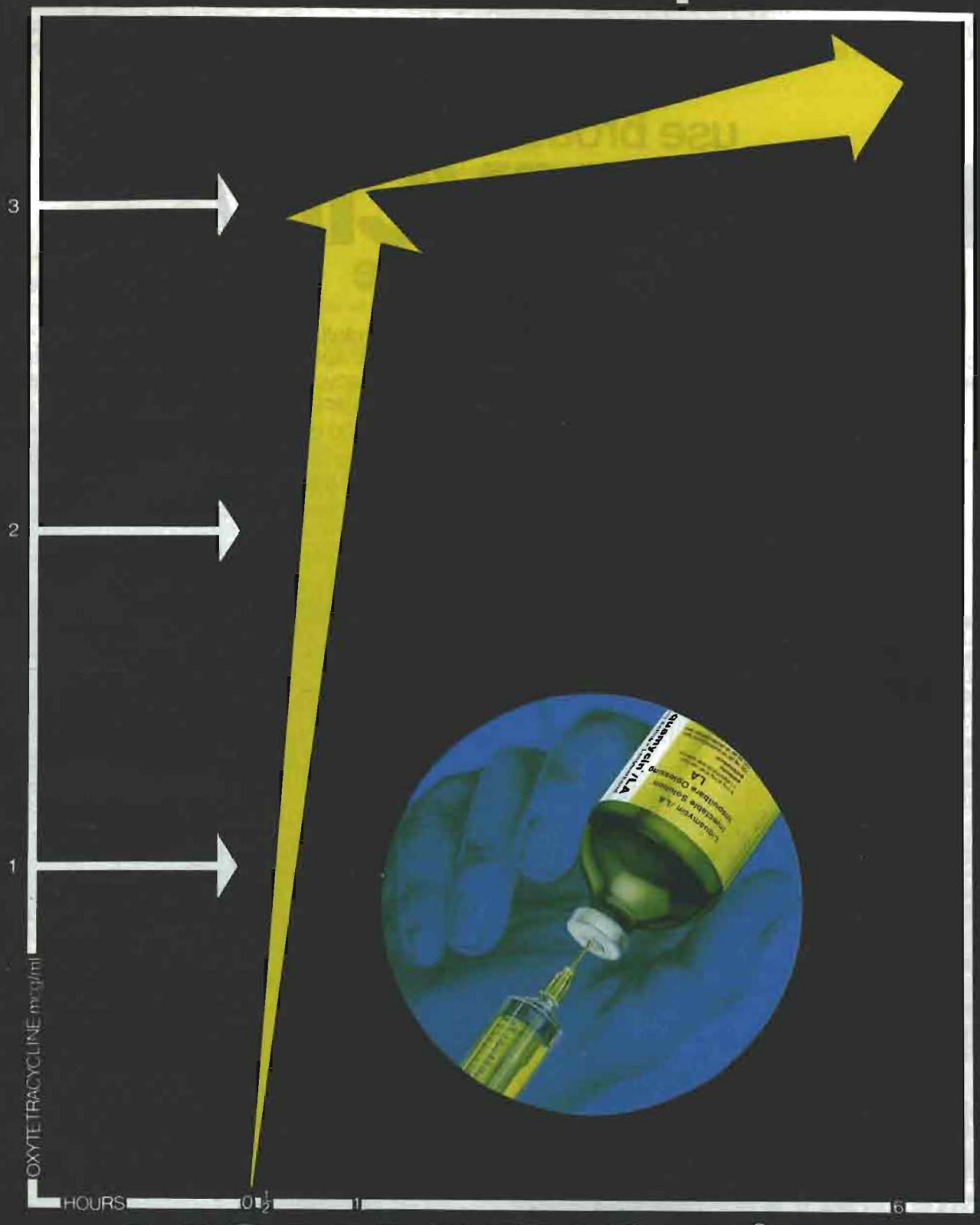

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(2) swine — in under 30 minutes, maintaining this high level for 8 hours;
(3) sheep — in under 30 minutes maintaining this high level for 12 hours;
(4) calves — in 15 minutes, maintaining this high level for 8 hours.

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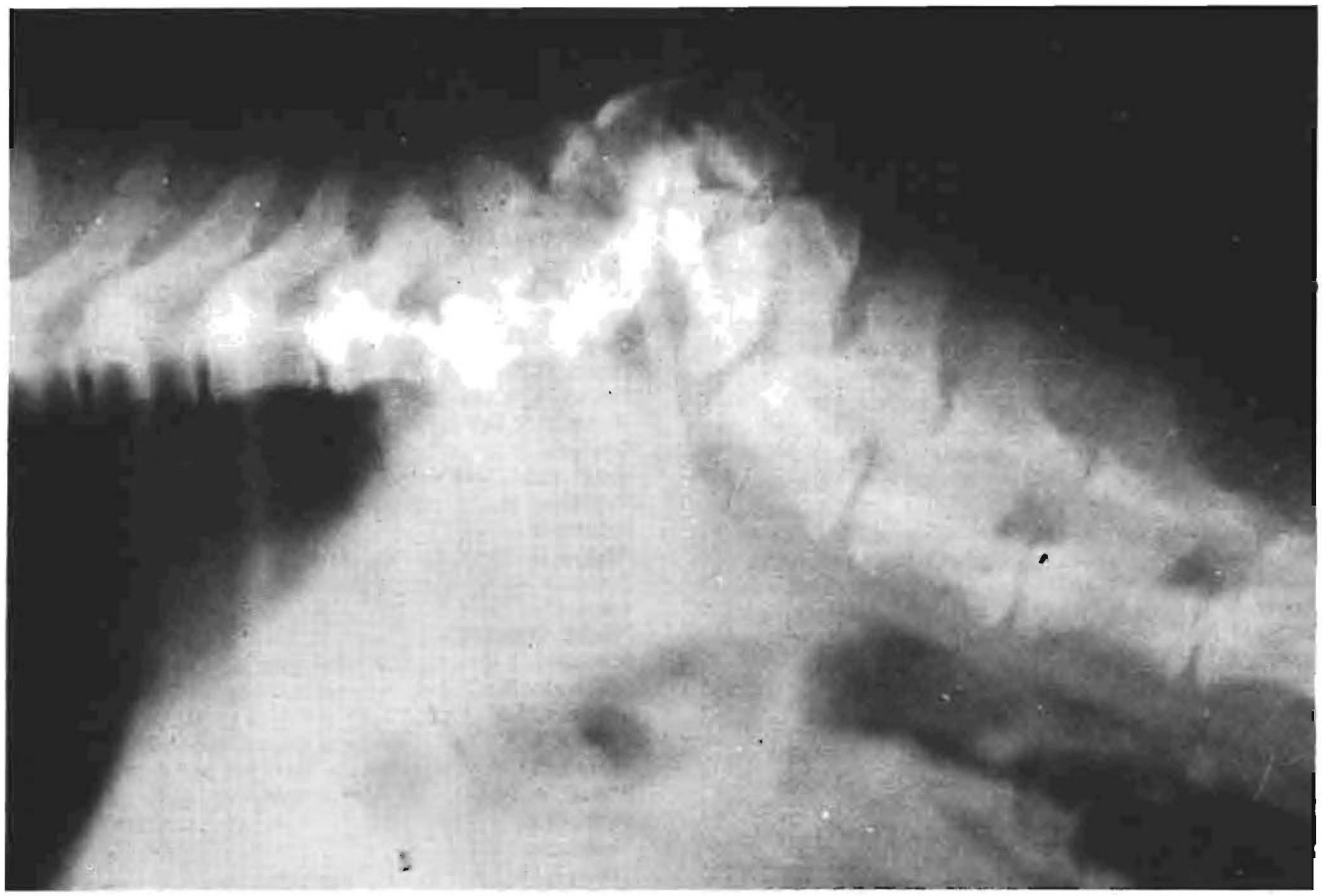
FEATURE PAGE **TREFFERBLAD**



CONGENITAL MALFORMATION OF THE VERTEBRAL COLUMN IN A CROSSBRED DOG

When a 4-month-old crossbred bitch was presented for routine vaccination, a distinct lump was observed on the vertebral column. According to owner it had been present since birth. The above radiograph was then taken, and shows an acute angle as well as malformation of the vertebrae in the region of the thoracolumbar junction. It is remarkable that the dog shows no neurological abnormalities and seems to be normal in all other respects.

Thanks are extended to Mr. P.L. Meyer of the Faculty of Veterinary Science, University of Pretoria for the printing of the photoghraph.

Submitted by: Dr Selma van Schouwenburg, 223 Bronkhorst St., New Muckleneuk, 0181 Pretoria.

KONGENITALE WERWELKOLOM-MALFORMASIE BY 'N KRUISRAS-HONDJIE

Toe 'n kruisras teefhondjie op 4 maande ouderdom vir roetine inenting gebring is, is 'n duidelike knop óp die werwelkolom waargeneem. Volgens die eienaar was dit reeds van geboorte af teenwoordig. Bygaande X-straal foto is toe geneem, en toon 'n skerp knak sowel as misvorming van die werwels in die gebied van die torako-lumbaal aansluiting. Merkwaardig is dat die hondjie geen neurologiese afwykings toon nie, en in alle ander opsigte heeltemal normaal is.

Mnr. P.L. Meyer van die Fakulteit Veeartsenykunde, Universiteit van Pretoria word bedank vir die afdruk van die foto.

Ingestuur deur: Dr Selma van Schouwenburg, Bronkhorststr. 223, New Muckleneuk, 0181 Pretoria.

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● Billing entry automatically calculates amount due by use of procedure codes ● Allows for patient hospital stay, retaining invoice until patient is released ● Allows for payment entry at time of billing and payment type (cash, check, visa, etc.) ● Prints customer invoice if required ● Prints daily invoices register, broken down by payment type ● Automatically updates patient history, inventory and accounts receivable file ● Hospital management

Options

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● Ages open invoices, showing vendor name, invoice date and discount date and amount ● Checks may be released automatically by date or exception ● Vendor report show year to date activity by vendor

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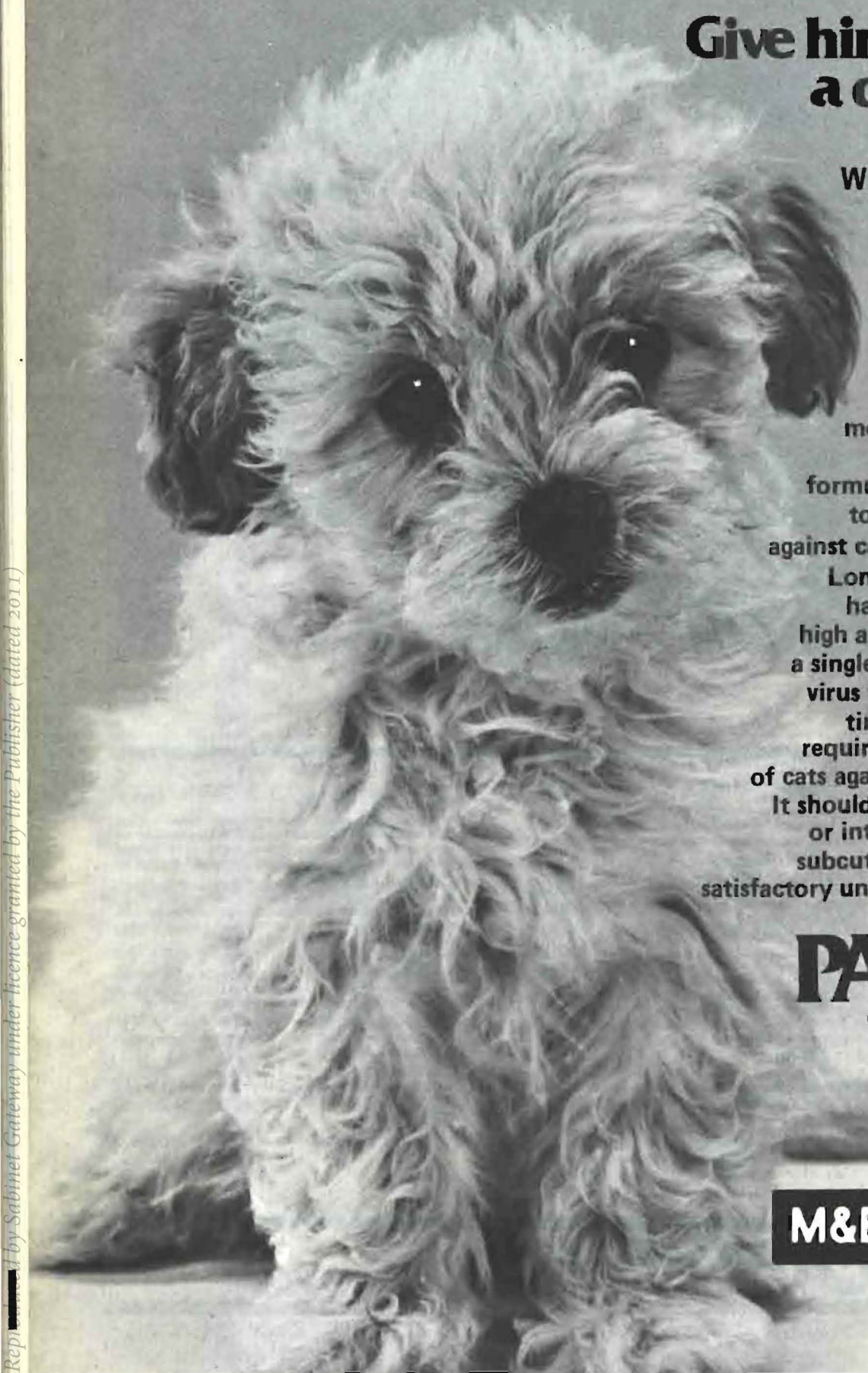

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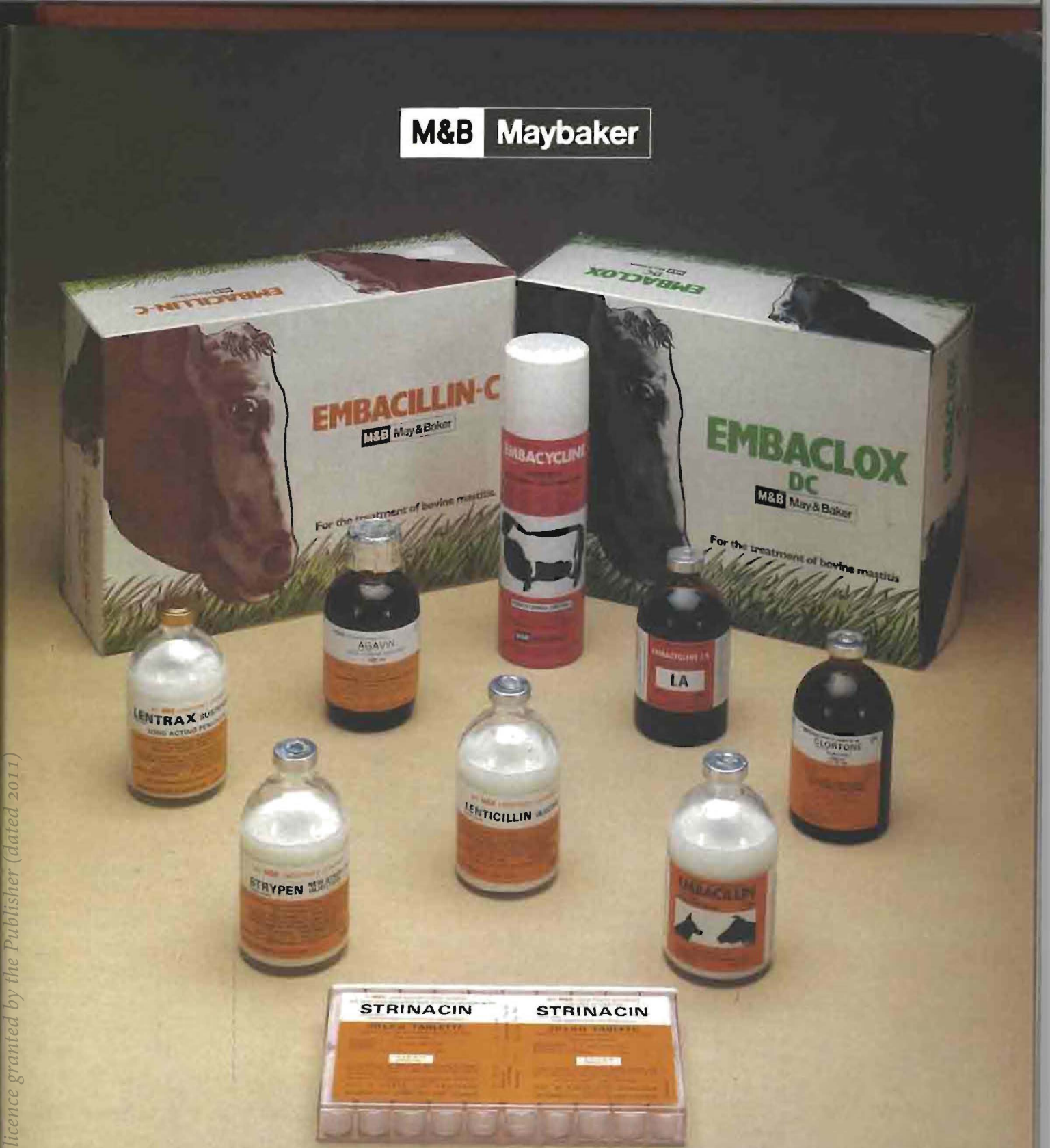

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